شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

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شبكة المعلومات الجامعية

بعض الوثائق الأصلية تالفة

THE HAEMOSTATIC CHANGES INDUCED BY CHRONIC NON VALVULAR ATRIAL FIBRILLATION AND THEIR ROLES IN ISCHEMIC STROKE

Thesis

Submitted to the faculty of medicine

University of Alexandria

For partial Fulfillment of the requirement for

Master degree in

Critical Care Medicine

By

Ehab Hassan EL GHaysha

(M.B.B.CH.)

Resident in Emergency Hospital Faculty of medicine

Mansoura University

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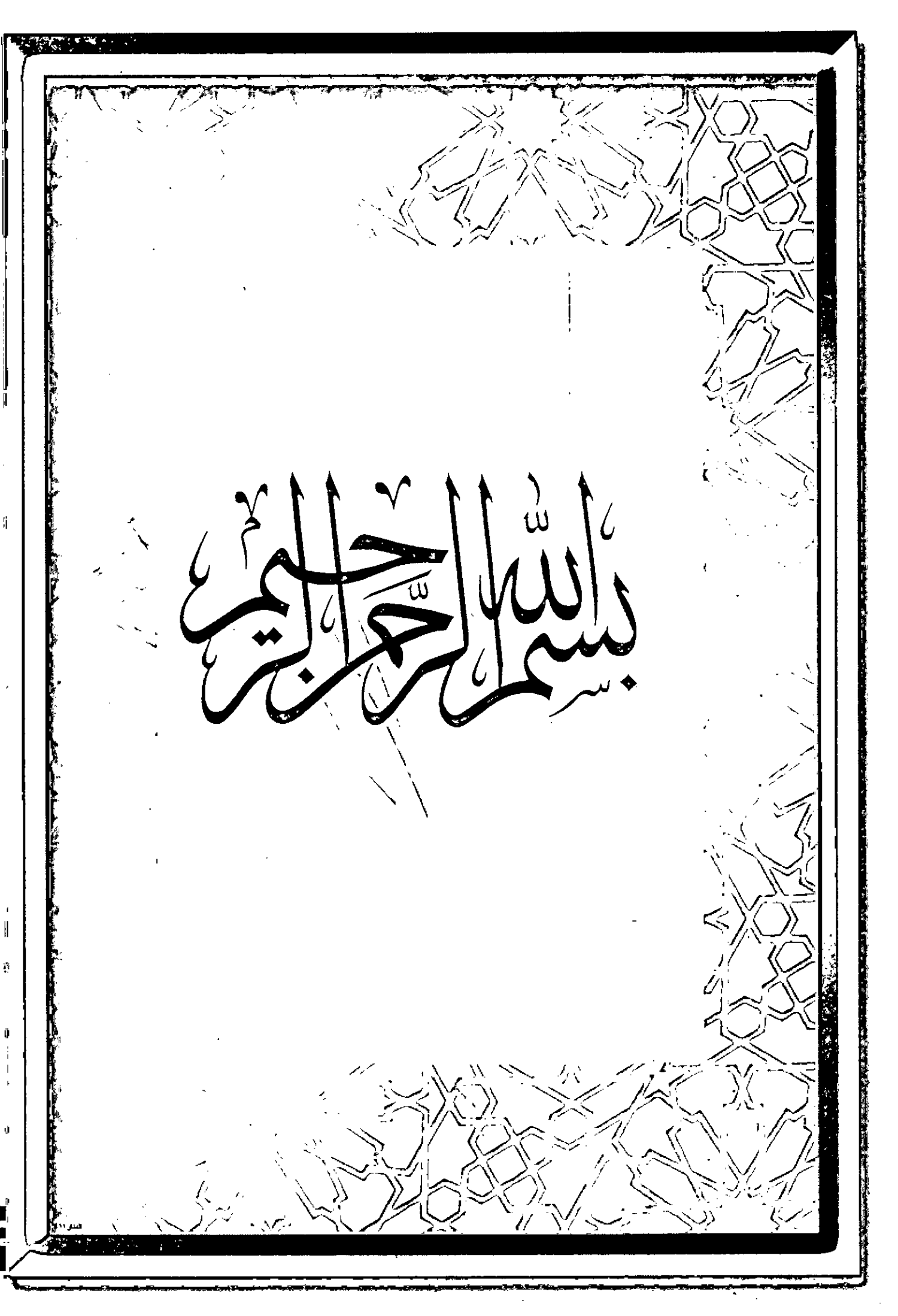
بسم الله الرحمن الرحيم

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Introduction And Aim of work

INTRODUCTION
AND AIM OF THE WORK

Atrial fibrillation is the most common sustained cardiac dysarrhythmia which is commonly associated with increased mortality and morbidity especially due to its thromboembolic complications, including ischemic stroke (1,2).

Chronic non valvular atrial fibrillation increases five fold the risk of ischemic stroke and is present in about 15 % of patients presenting with acute ischemic stroke (3).

The pathophysiologic mechanism by which atrial fibrillation produces stroke is uncertain and it may be due to absence of atrial systole ("atrial kick") with consequent intra cardiac thrombus formation which may result in thromboembolism. The recently discovered haemostatic changes in the form of hypercoagulable state and increased intravascular thrombogenesis which occurs in association with atrial fibrillation is another explanation for the increased incidence of ischemic stroke in chronic non valvular atrial fibrillation patients (4).

Proper understanding of the pathophysiologic mechanisms by which atrial fibrillation induces ischemic stroke may be helpful in reducing ischemic stroke incidence among these patients category.

Review of Literature

HAEMOSTASIS

Haemostasis is to arrest haemorrhage at the site of blood vessel injury (5). Normal haemostasis depends on a delicate balance and complex interaction between the following: blood vessel, platelets, plasma coagulation proteins and fibrinolytic system (6).

Blood vessels and haemostasis:

Blood vessels form a non leaking closed circuit which maintains blood in a fluid state. When a leak does occur platelets and coagulation system temporarily close the defect until the cells in the vessel wall permenantly repair the leak. If blocked by a thrombus, blood vessels can usually re-establish blood flow by lysing the thrombus. These capabilities are for the most part manifestations of the functional properties of the endothelial cells that line the blood vessels (7).

Endothelial basement membranes contain collagen, von willebrand factor, vitronectin, microfibrils and thrombospondin. In the absence of endothelial cells, the subendothelium acts as a secondary barrier against the passage of blood (8). Fibrinogen is not normally present in the subendothelium. While endothelial cells do not synthesize fibrinogen, fibrinogen/fibrin is present in the subendothelium of healing blood vessels (8).

Normal endothelium can be regarded as a highly active metabolic and endocranial organ that plays a major role in the maintainance of a proper balance between the formation of haemostatic plugs and the avoidance of intraluminal thrombi (9).