POSTOPERATIVE CHANGES IN PROTEIN C ACTIVITY

T H E S I S

Submitted For Partial Fulfilment Of

Master Degree In Clinical Pathology

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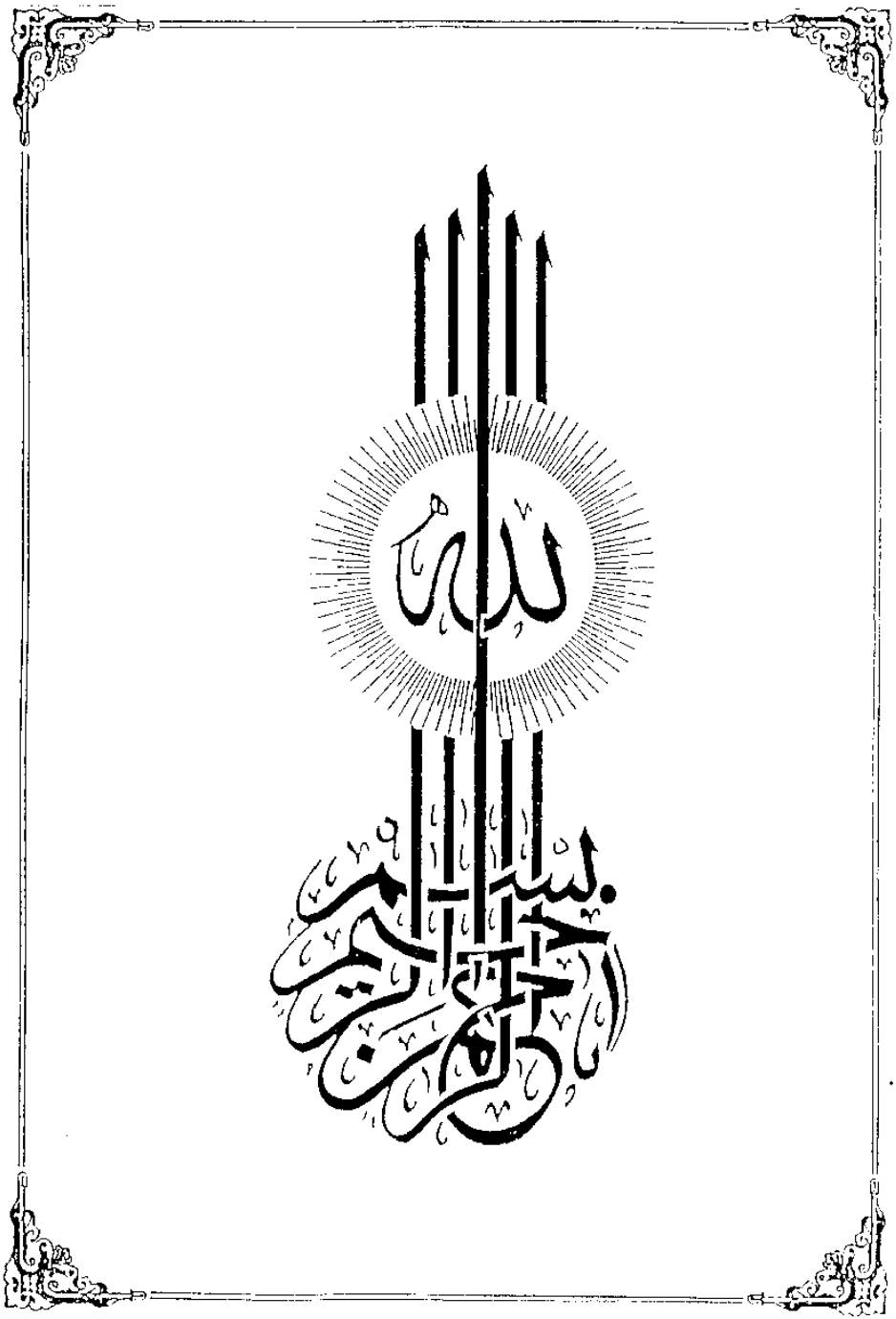

C O N T E N T S

L I S T OF F I G U R E S

L I S T OF T A B L E S

A B B R E V I A T I O N S

a	Activated.
Ab	Antibody.
Ag	Antigen.
APC	Activated protein C.
APL	Acute promyelocytic leukaemia.
Arg-	Arginine.
Asp-	Aspartic acid.
AT III	Antithrombin III.
Ca^{2}	Calcium ions.
C_1	Complement one.
C_4 b	Complement four fragment (b).
DFP	Diisoprophylflourophosphate.
DIC	Disseminated intravascular coagulopathy.
E. Coli	Escherichia coli.
EDTA	Ethylene diamine tetraacetic acid.
ELIZA	Enzyme linked immunosorbent assay.
et al.	Etalli = and other coworkers.
F	Factor.
Factor I	Fibrinogen.
Factor II	Prothrombin.
Factor III	Tissue factor.

Factor IV	Calcium.
Factor V	Pro-accelerin.
Factor VII	Pro-convertin.
Factor VIII	Antihaemophilic.
Factor IX	Christmas factor.
Factor X	Stuart-Prower factor.
Factor XI	Plasma thromboplastin antecedent.
Factor XII	Hageman factor.
Factor XIII	Fibrin stabilizing factor.
FDPs	Fibrin degradation products.
Gla-	Gamma carboxyglutamic acid.
Gly-	Glycine.
HMW kininogen	High molecular weight kininogen.
Ile	Isoleucine.
M.Wt.	Molecular weight.
N-terminal	Amino terminal.
PC	Protein C.
PCCs	Protein C concentrates.
Pro-	Proline.
PS	Protein S.
PT	Prothrombin time.
PTT	Partial thromboplastin time.
RVV-X	The protease from Russell's vipor venous that activates factor X because it contains VIIa.
Val-	Valine.

S Y M B O L S

∝	=	Alpha.
B	=	Beta.
C	=	Degree centigrade.
ɣ	=	Gamma.
g	=	Gram.
Kg	=	Kilogram.
L	=	Liter.
u	=	Micro.
ug	=	Microgram.
uL	=	Microliter.
mg	=	Milligram.
nm	=	Nanometer.
%	=	Percent.
P	=	Propability.
rpm	=	Revolution per minute.
U	=	Unit.
>	=	More than.
<	=	Less than.

A C K N O W L E D G E M E N T

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INTRODUCTION AND AIM OF THE WORK

INTRODUCTION

Thromboembolic accidents are known troublsome postoperative complications. It is related to many factors particularly, the type of the operation, age of the patient and many haemostatic factors. The haemostatic dynamic equilibrium is mainly centered upon the delicate balance between the fibrinogenic versus the fibrinolytic processes. Factors affecting the coagulability of blood or affecting the inhibitory systems of blood coagulation are accused as implicated in the pathogensis of postoperative thromboembolic complications.

Recently, a previously known autoprothrombin II-A, plasma protein was found to have a regulatory action on the dynamic equilibrium of haemostasis. This protein was first characterized by Stenflo (1976) and named it " Protein C" because it was purified from a protein fraction (Pool C) obtained after gradient elution of a prothrombin complex concentrate on the third peak of DEAE-Sephadex column chromatography.

Stenflo (1976) found that it is a vitamin K-dependent and counmarin-sensitive protein which circulates in the

blood as a zymogen that is converted into its active form by thrombin.

The activated form in vitro is a powerful inhibitor of activated factor V mainly and activated VIII:C and a stimulator of fibrinolysis (Comp and Esmon, 1978). Activated protein is not inhibited by heparin and antithrombin III (Kisiel, 1979) but by a specific inhibitor.

Many recent works are trying to correlate between protein C deficiency and conditions associated with hypercoagulable states.