COMPARATIVE STUDY BETWEEN THE EFFECT OF ABDOMINAL AND VAGINAL HYSTERECTOMY ON COAGULATION PATTERN

THESIS

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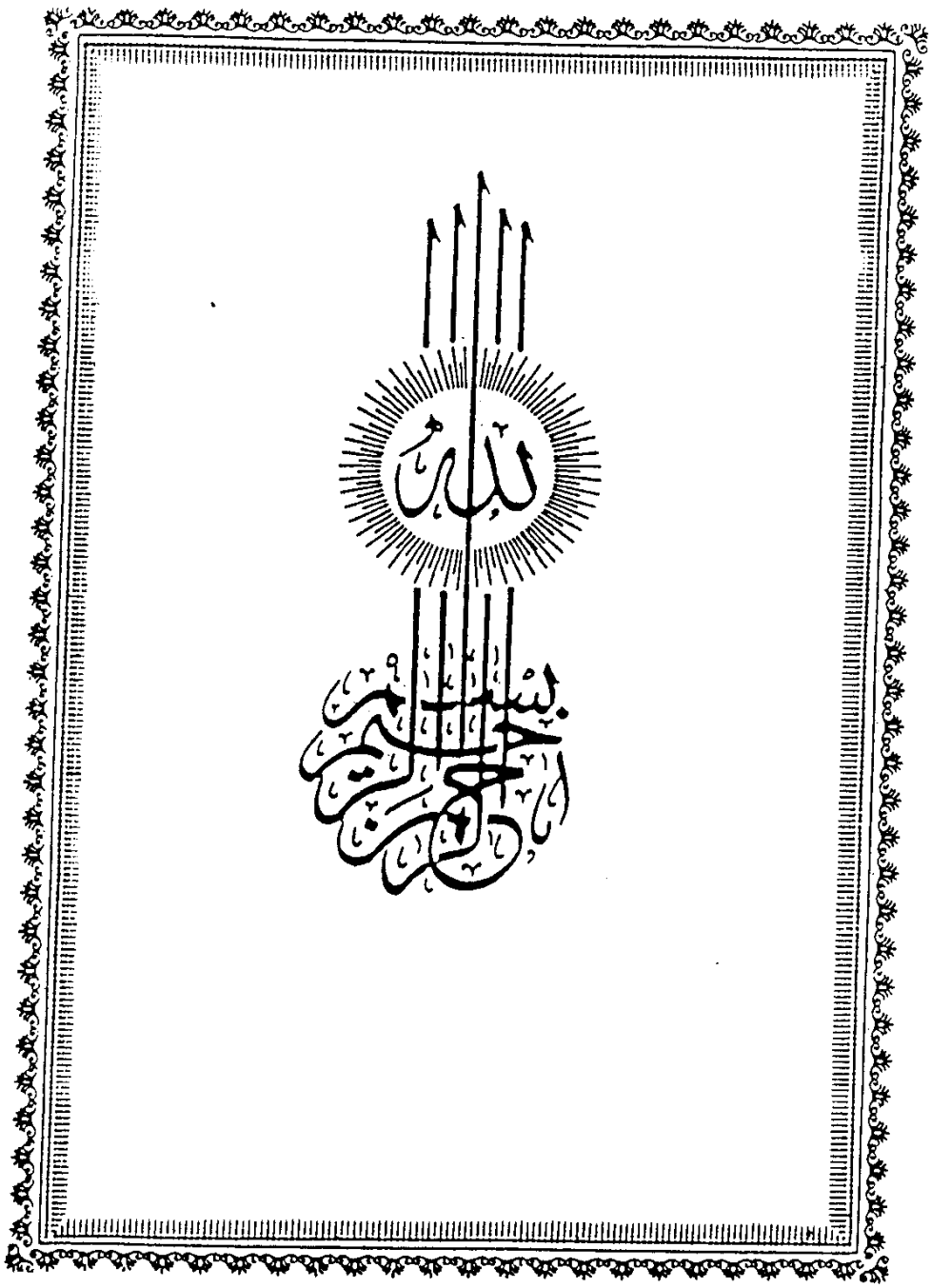

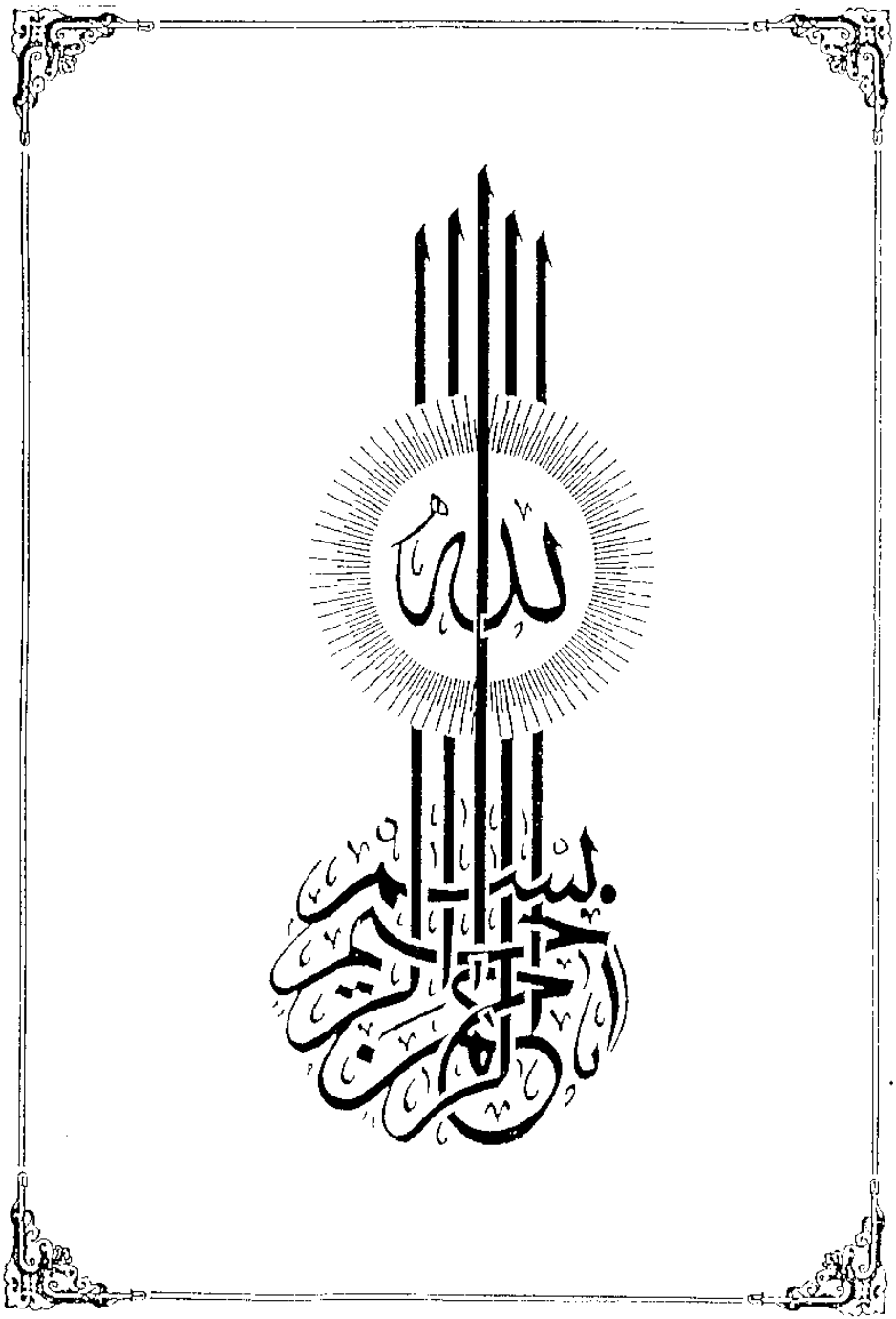

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INTRODUCTION

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Major surgery is followed by changes in the coagulation and fibrinolytic systems which may favour the development of postoperative thrombo embolic complications (Rem et al., 1981).

The importance of clotting abnormalities in the post operative period is well known, as post operative thrombo embolism continues to be a major theat, although numerous authors have studied a variety of coagulation parameters in the postoperative period there is decided lack of homogeneity in cases studied and most authors reported changes in a limited number of variables. There is tendency towards hypercoagulability in the post operative period, this is manifested by changes in a number of coagulation parameters and if it is not offset by some protective mechanism, thrombosis may occur (Collin et al., 1977).

Patients undergoing major gynaecological surgery are known to develop laboratory and or clinically overt signs of deep vein thrombosis with a frequency of 15-30% (BredBacka, et al., 1986).

Venous thromboembolism remains a frequent preventable cause of mortality in Obstetric and Gynaecology (Clarke Person et al., 1981).

Pulmonary embolism has been noted to be the major postoperative complication contributing to death after radical hysterectomy and pelvic lymphadenectomy for carcinoma of the cervix and after total abdominal hysterectomy and bilateral salpingo oophorectomy for endometrial carcinoma (Clarke-Person et al., 1983).

In 1984 Clarke-person et al., reported that the incidence of venous thromboembolism after gynecologic surgery was 17% other reports from gynecologic services was found a range of incidence between 7% and 45% depending on the risk factors related to age of the patient, surgical procedure, coexistant malignant disease and multiple other associated risk factors.

AIM OF THE WORK

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The aim of this work is to evaluate how far the activity of some coagulation factors are affected in women subjected for either abdominal or vaginal hysterectomy, together with comparison between this affection in both techniques.

REVIEW OF LITERATURE

NORMAL COAGULATION

Blood normally circulates through endothelium lined vessels without coagulation or platelet activation taking place and without appreciable haemorrhage. Injury to the vessel triggers the haemostatic process, platelets adhere to the damaged endothelium or exposed subendothelium and change shape preparatory to aggregation and secretion of intracellular contents at the same time plasma proteins react with the subendothelium resulting in activation of the contact phase of coagulation (Robert, 1982).

Over 40 different substances that affect blood coagulation have been found in blood and tissues, some promoting coagulation called procoagulants and others inhibiting coagulation called anti coagulants, whether or not blood will coagulate depends on the degree of balance between these two groups of substances, normally the anticoagulants predominate and the blood does not coagulate but when a vessel is injured the activity of procoagulant in the area of damage becomes much greater than that of the anticoagulant and then a clot does develop (Gyton, 1986).

Table (1): The coagulation system.

Factor	Name(s)
I	Fibrinogen
II	Prothrombin
III	Thromboplastin
IV	Calcium
V	Proaccelerin, labile factor, accelerator globulin
VI	Accelerator globulin
VII	Proconvertin, SPCA, stable factor
VIII	Antihemophilic factor (AHF), antihemophilic factor A, antihemophilic globulin (AHG)
IX	Plasma thromboplastic component (PTC), Christmas factor, antihemophilic factor B
X	Stuart-Prower factor
XI	Plasma thromboplastin antecedent (PTA), antihemophilic factor C
XII	Hageman factor, glass factor
XIII	Fibrin-stabilizing factor, Laki-Lorand factor
HMW-K	High molecular-weight kininogen, fitzgerald factor
Pre-K	Prekallikrein, Fletcher factor
Ka	Kallikrein
PL	Platelet phospholipid

(Quoted from Ganong 1987)

Activation of The Coagulation Mechanism:

The coagulation system (table 1) consists of a series of zymogen that are activated to proteases with the eventual formation of thrombin and fibrin clot.

There appears a connection between the fibrinolytic mechanism, the complement system, kallikrein system and coagulation mechanism mediated by Hageman factor.

The coagulation mechanism can be divided into three parts (Esnouf, 1977).

1- An intrinsic system.

2- An extrinsic system.

3- A final common pathway which is initiated by the activated factor X and results in the formation of fibrin clot.

Either intrinsic or extrinsic mechanisms can initiate clotting. Factors within the vescular space activate the intrinsic system and tissue thromboplastin from outside the vascular space activate the extrinsic system. These two systems converge in a final common pathway, the activation of factor X, which carries on the clotting sequence (Robert, 1982).

The Intrinsic System:

Activation of intrinsic clotting pathway occurs when the Hageman factor (Factor XII) comes in relation to a negatively charged surfaces as subendothelial tissues and platelet membrane.

When factor XII binds to a negatively charged surface, it undergoes a conformational change. This change renders it more susceptible to limited proteolytic cleavage by prekallikrein in the presence of high molecular weight kininogen. Kallikrein is derived from prekallikrein and it circulates as complex with high molecular weight kininogen. Also activated factor XII cleaves prekallikrein into kallikrein the latter in turn activates factor XII.

Factor XIIa activates factor XI. Factor XI can also be activated by platelets (Walsh, 1974).

Activated factor XI will result in activation of factor IX that in turn activates factor VIII. These in turn will activate factor X, that is bound to the phospholipid in platelet membrane by calcium bridge with α carboxyglutamic acid.