HAEMOSTASIS AND SURGERY

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BY

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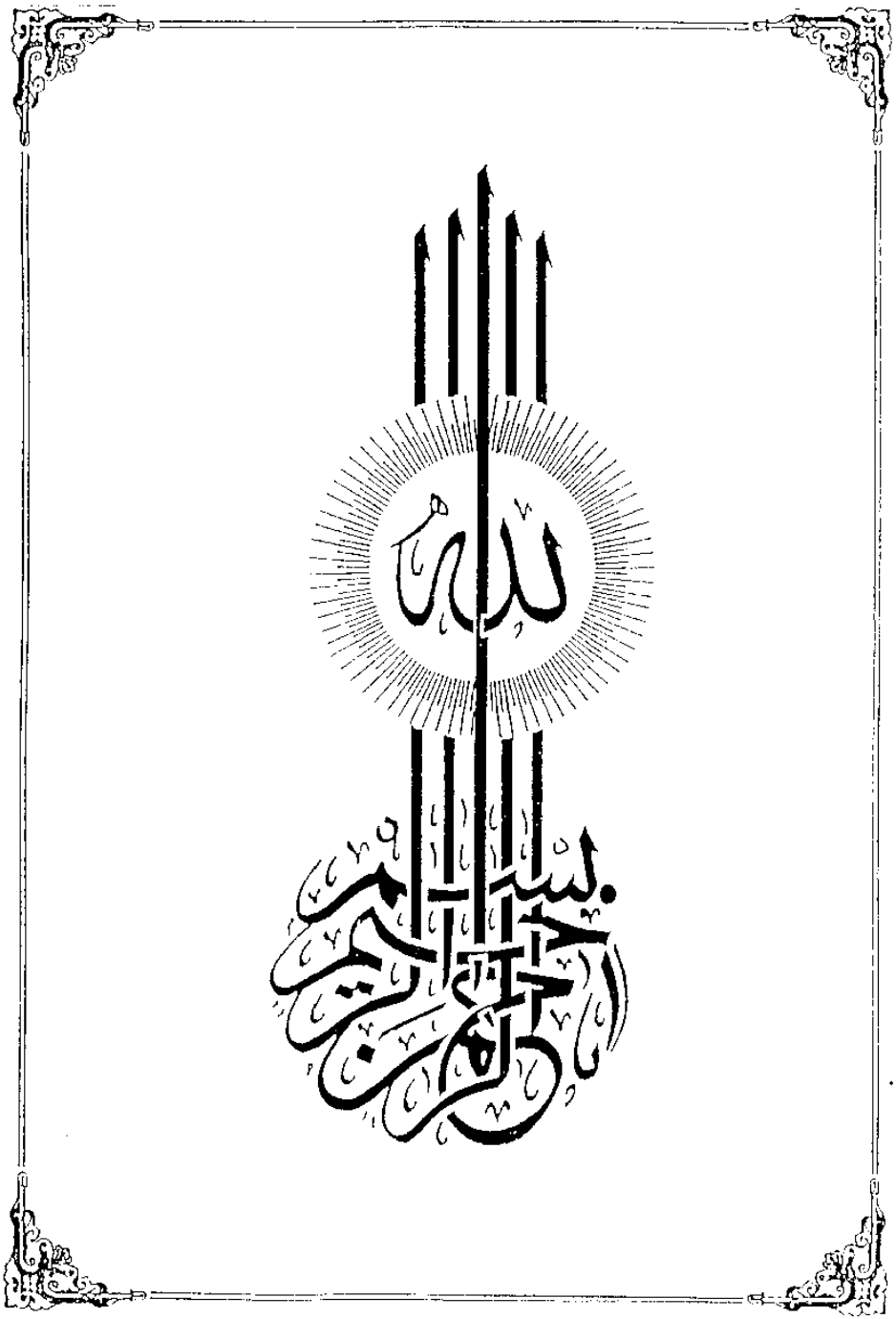
C O N T E N E S



INTRODUCTION

Obviously every surgeon would like to believe that in his own practice complications will be, at the least, occasional events. This aim can be elicited by covering details of etiology, predisposing factors and methods of prevention .

But when they do occur, the surgeon must be alert to how they can be early recognized, investigated and managed .

This essay deals with haemorrhage as a complication during or after surgical operation which mainly may be due to mechanical fault. But sometimes it may be due to coagulation disorders which also may be preoperatively pre-exist or may complicate surgery .

CHAPTER I

HAEMOSTASIS AND BLOOD COAGULATION

HAEMOSTASIS AND BLOOD COAGULATION

Haemostasis is one of the major protective mechanisms in the body, it controles bleeding in injury, helps in the preservation of vascular integrity, assists in localizing infections and participates in wound healing (Davies, I972) .

Haemostasis can be defined as spontaneous cessation of blood loss from the intravascular space, provides a fibrin network for tissue repair and ultimately removes the fibrin when it is no longer needed .

The normal haemostasis occurs as consequence of four major physiologic reactions :

I - response of the injured vessels (vascular constriction)

2 - platelet activities (platelet plug formation)

3 - biochemical changes in the coagulation mechanism(fibrin formation)

4 - fibrinolysis (Schwartz, I989)

Vascular response

The initial vascular response to injury even at the capillary level is vasoconstriction . The narrowing of the end

Platelet activities

Blood platelets are 2 um in diameter, number 200,000 to 400,000 per cubic millimeter of blood in which normally circulate for 7 to 9 days . They carry a simillar electrostatic charge to that on intact vascular endothelium and are repelled by it . They are not adherent to each other or to the blood vessel wall . But they are strongly attached to damaged , depolarised vascular endothelium, to basement membrane, and to collagen fibres .

In the slow physiological turnover of vascular endothelial cells, platelets temporarily plug any intercellular gaps by adhering to the exposed basement membrane, without releasing any of their active compenents, and thus they help to maintain vascular integrity (Davis, I972) .

In pathological vascular damage platelets play a fundamental role in haemostasis through three basic reactions; adherance, secretion (or release), and aggregation .

A - Adherance

In less than I5 seconds following vascular injury, platelets escaping from an injured blood vessel come into contact with,

and adhere to a number of tissues, in particular, to collagen, this involves the interaction between a specific receptor on the platelet's surface and a glycopeptide group of collagen and requires Von Willebrand factor (VWF) as a co-factor (Hutton, I98I) .

B - Release reaction

Within seconds of platelets adhesion to collagen they swell and release a variety of chemicals including :-

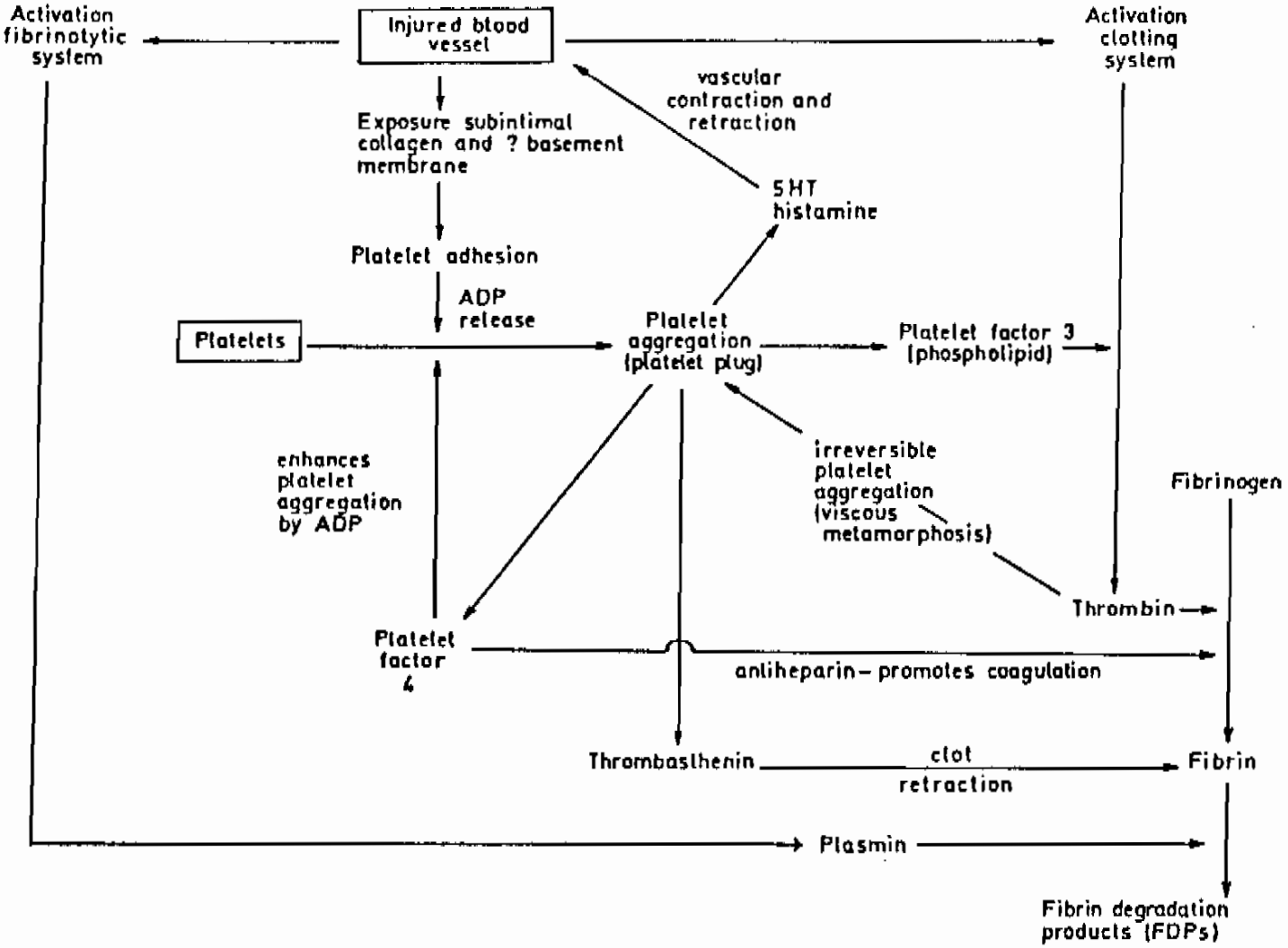
- Adenosine diphosphate (ADP) from their own stored adenosine triphosphate which induces platelet aggregation .
- Phospholipid (platelet factor 3) which activates the clotting system, through activation of factor XII (which involved in the intrinsic mechanism of coagulation) .
- Platelet factor 4, it is a heparin neutralizing substance .
- Serotonin (5-hydroxy tryptamine) and other vasoconstrictors which enhance local vascular constriction .
- Thrombosthenin which is a contractile protein complex identical with the actinomysin system of the muscle and lead to mechanical clot retraction, which acts as natural or physiological ligature of the wound (Walter and Israel, I984) .

- Also platelet releases arachidonic acid from its membrane which is converted by cyclooxygenase enzyme to prostaglandin endoperoxides which in turn are converted to thromboxan A_2 which is a potent platelet aggregator and vasoconstrictor (Schwartz, I989) .

C - Aggregation

Following release reaction, it is believed that platelets expose another specific glycoprotein complex receptor which mediates aggregation, the process by which platelets interact with each other,and they lose their individual identity and begin to fuse with one another leading to formation of platelet plug . It is seem that ADP is a powerful inducer of platelet aggregation and also fibrinogen is necessary for this reaction presumably as a bridge between glycoprotein receptor on the adjacent platelets (Ansell, I986) .

The platelet plug formation is called primary haemostasis and it is seem to correspond whate we measure with the bleeding time. In small vessels such as capillaries, the combination of vaseconstriction and platelet plug may be adequate to achieve haemostasis (Morrison, I98I) .



The role of platelets in haemostasis.

(Davies, 1972)

Blood coagulation

The fundamental reaction in the clotting of blood is conversion of the soluble plasma protein fibrinogen to insoluble network of fibres which is called fibrin .

Normal blood coagulation proceeds as complex cascade of reactions involves 20 or so substances, most of which are plasma proteins . Most of these have been assigned roman numerals, however some are occasionally referred to by the surnames of the patients in whom they were first discovered . e.g, factor IX (Christmas factor), factor X (Stuart-power factor); factor XII (Hageman factor), and other some are referred to its function .

All of the blood clotting factors circulate in blood in precursive forms, or zymogens, or as cofactor proteins that have somewhat different structures until activated or altered during the clotting process (Mckee, I985) . These factors can be divided into three groups on the basis of their general properties .

Group (I) : Vitamin K dependent clotting factors

Group (2) : Thrombin sensitive clotting factors

Group (3) : The contact factors

BLOOD COAGULATION FACTORS

Roman numeral	Substance
I	Fibrinogen
II	Prothrombin
III	Tissue thromboplastin; tissue factor
IV	Calcium ion
V	Proaccelerin : liable factor
VI	Not assigned
VII	Proconvertin stable factor
VIII	Antihaemophilic factor
IX	Christmas factor: plasma thromboplastin component(PTC)
X	Stuart factor or Stuart-power factor
XI	Plasma thromboplastin antecedent (PTA)
XII	Hageman factor
XIII	Fibrinogen stabilizing factor
-	Prekallikrein (Fletcher factor)
-	High molecular weight kininogen (HMWK)
-	Von Willebrand factor
-	Protein C
-	Protein S

(Mckee, I985)

Group (I) : Vitamin K dependent clotting factors

-Prothrombin

-Factor VII

-Factor IX

-Factor X

-Protein C

-Protein S

These factors have in their molecular structure an amino acid residue, which is ɣ-carboxy glutamic acid, which is necessary for:

-Lipid and calcium binding .

-Full expression of the proteolytic activity of the active form of these factors (except thrombin) .

In the absence of vitamin K; these factors are still synthesized but with a glutamic acid rather than carboxy-glutamic acid,(the carboxylation is a vitamin K-dependent), in this form they are inactive .

Group (2) : Thrombin sensitive clotting factors

-Fibrinogen

-Factor XIII

-Factor V

-Factor VIII

All these factors are attacked by thrombin, which activates factor V, VIII, XIII , and it also splits off two peptide fragments (fibrino-peptide A and B) from a molecule of fibrinogen to form fibrin monomer which then polymerises spontaneously with its fellow to form strands of fibrin .

Group (3) : The contact factors

-Factor XII (Hageman factor)

-High molecular weight kininogen(HMWK)

-Prekallikrein

-Factor XI

These factors are slowly synthesized by the liver and they are involved in the earliest phases of clotting; the contact factors not only have a role in coagulation but they are also involved in kinin formation and fibrinolysis(Hougie and Baugn, I980) .

We must note that:

I - Factor III (thromboplastin) described a function and it is not referred to a single substance, this function is activation or catalyzing the conversion of prothrombin to thrombin .

Substances with thromboplastic activity are contributed by the plasma, the platelets and the tissue .

From the plasma :-Factor VIII (AHG)

-Factor IX (PTC)

-Factor XI (PTA)

-Factor XII

-Factor X

-Factor V

From platelets :-Platelet thromboplastic factor

From the tissue :-Tissue factor (Harper, I977)

2 - Factors II, VII, IX, X, XI, XII, XIII are all enzymes and they of a class called serine proteases(Walter and Israel,I984).

3 - Factor VIII circulates in a complex with Von Willebrand factor and requires cleavage by a thrombin like enzyme to develop the procoagulant activity that interacts with factor IX, ca^{++} and phospholipid to form an activator of factor X. Also factor VIII-Von Willebrand- complex has an activity not effected by thrombin which is necessary for platelets to adhere to the severed edges of blood vessel or to foreign surfaces (Von Willebrand activity) (Mckee, I985) .

4 - Although a factor VI has been described, it is currently believed that no such separated factor exists, and consequently it has been delected (Harper, I977) .