Blood Loss During Vaginal Delivery and Caesarean Section

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رسالة

By

Wael Hamed Abd El Khalek Helmy
M.B., B.Ch. (Cairo University)

618.277
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44568

Under Supervision of

Prof.
Alaa El Din Kamal El Atriby
Professor of
Obstetrics and Gynaecology
Ain Shams University

Prof.
Samia Abd El Razzak Hemeida
Professor of Public Health
National Research Center

Dr. Magdy Mohamed Kamal
Lecturer of Obstetrics and Gynaecology
Ain Shams University

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INTRODUCTION

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INTRODUCTION

Haemorrhage (hge) is one of the most serious and critical events that may face the obstetrician and can be a cause of maternal mortality. As a direct factor in maternal mortality postpartum haemorrhage is a cause of about one quarter of deaths from obstetric hge in group that includes: postpartum hge, placenta praevia, placental abruption, ectopic pregnancy, hge from abortion and rupture of the uterus **(Williams, 1989)**.

Definition

PPH is usually defined as blood loss greater than 500ML. However, this definition is problematic because visual estimates of blood loss are notoriously inaccurate. In several studies using reliable methods to determine the amount of bleeding, blood loss in vaginal deliveries averaged about 500ML suggesting that this value is actually normal **(Brant, 1967)**.

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In a recent study, cases of hge were defined by a haematocrite (Hct) reduction of 10 points or more between admission and postdelivery or by the need for red cell transfusion **(Combs et al., 1991)**.

A definition of PPH based on Hct change has several advantages. First, it is objective and relatively precise. Second, admission and postpartum Hct are routinely and simply obtained, allowing study of a large population of patients. Third, Hct is a clinically relevant variable often used in decision making regarding the need for transfusion or iron therapy. Fourth, Hct change is affected not only by Hge in the delivery room but also by delayed hge. Finally, the definition of hge used here is similar to the citerion proposed by the American College of Obstetricians and Gynaecologists as an indication for quality assurance review.

The definition based on Hct value, however, has several disadvantages. First, Hct change is sensitive to both antenatal and postpartum blood loss. Second, Hct change will not necessarily reflect hge that has been treated with immediate blood transfusion. Third, Hct change is sensitive to antenatal haemoconcentration and postpartum haemodilution.

Incidence

Blood loss during vaginal delivery and caesarean section is an inevitable event in obstetric practice and when it mounts to postpartum haemorrhage, it becomes a major cause of maternal morbidity in

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developing countries where maternal mortality rates are 5-10 per 1000 livebirths. PPH remains a leading cause of maternal death **(Harrison, 1989 and El Kady et al., 1989)**.

Brant (1967), stated that the incidence of postpartum hge on the basis of 600 cubic centimeters blood loss or more is usually in the neighbourhood of 5-6 percent.

Aim of the Work

1. To estimate the amount of blood loss during vaginal and Caesarean section deliveries.
2. To determine the predisposing factors so that patients with risk factors can be offered practices that might reduce the incidence or morbidity of hge including delivery in hospital rather than at home, establishment of intravenous access during labour, prophylactic administration of oxytocics before delivery of the placenta and blood banking procedures to ensure ready availability of blood for transfusion.
3. To indentify the changes in the management of labour that might have contributed to the increase in the amount of blood loss.

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REVIEW OF LITERATURE

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REVIEW OF LITERATURE

Physiology of the Third Stage

Strictly speaking, the third stage of labour represents the period from birth of the infant to the delivery of the placenta. From a practical point of view, however, the two hours following delivery of the placenta are just as important as the actual third stage. Indeed these two periods may be said to be more dangerous to the mother than the other stages of labour, as is reflected in the immediate danger due to hge and the more remote risk of puerperal sepsis **(Brant, 1967)**. The normal physiological mechanisms which operate to prevent excessive blood loss during labour include:

1- Contraction and retraction of the uterine muscle to control the large blood vessels and sinuses at the placental site. The greater part of the uterine musculature is composed of intermediate criss-cross layer of muscle fibres which are arranged trellis fashion and through the interstices of which the maternal blood vessels supplying the placental site run a tortuous course. Closing the gaps in this trellis

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by retraction shuts off the supply of blood to the placental site and for this reason these fibres are known as the "living ligatures of the uterus" **(Greenhill, 1974)**.

2- Thrombosis occurs in some of the uterine vessels at the placental site and this will reduce the number of the opened uterine vessels after delivery.

3- During pregnancy also the pituitary gland increases in size slightly and the blood flow through it will be modified as the greatest part will go to the postrior lobe which releases oxytocin, so playing an important role in controlling the uterine contractions.

4- There is thickening of the vaginal mucosa and loosening of the connective tissue together with hypertrophy of smooth muscles. These changes help the distension of the vagina during labour and protect it from tearing.

5- Haemostatic mechanisms that operate during pregnancy to secure haemostasis: **Gibson (1991)** stated that human pregnancy is associated with unique physiological changes in many components of haemostasis and fibrinolysis. Despite or because of these adaptations, both hge and thrombosis remain major causes of maternal morbidity and mortality even in modern obstetrical practice. A brief review of the normal coagulation process includes the following:

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I. Normal Haemostasis

Both the control of blood loss and the maintenance of vascular patency are dependent upon normal haemostasis which includes:

1- Platelet vessel interaction

2- The coagulation system

3- The inhibitors of coagulation

4- Fibrinolysis

1- The Platelet-vessel interaction:

The vascular endothelium is the site of synthesis of a number of important components of haemostasis, in particular, collagen, von Willebrand factor (VWF) and prostacyclin (PGI_2). Collagen is exposed when the vascular endothelial surface is damaged. Circulating VWF is adsorbed to the collagen and adheres to the platelet glycoprotein membrane receptor (GPLb) causing platelet adhesion. Platelets adhering to the collagen are activated and change in shape to become spherical and release adenosine diphosphate (ADP) and serotonin from their dense bodies resulting in platelet aggregation. The prostaglandin pathway provides an additional route for platelet aggregation and is initiated when platelet arachidonic acid is synthesized from phospholipids under the influence of membrane phospholipase. Cycloxygenase converts arachidonic acid to the prostaglandin endoperoxides, these in turn are converted to thromboxane A_2 (TXA_2) a potent aggregating agent and vasoconstrictor by thromboxane synthetase.

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Thromboxane B_2 (TXB_2) is the stable metabolite of (TXA_2). Prostacyclin (PGI_2) is an unsatble prostaglandin synthesized by blood vessels and is a powerful vasodilator and potent inhibitor of platelet aggregation. In health, the production of PGI_2 by the vessel wall and TXA_2 by platelets is balanced.

Fibrinogen is the most important of the many substances released from platelet alpha granules and binds to the specific platelet glycoprotein membrane receptors (GP IIb and GP IIIa) facilitating further aggregation. Platelet phosphlipid (platelet factor 3) on the platelet surface, catalyses the intrinsic and extrinsic pathways, resulting in the local formation of thrombin. Thrombin causes irreversible platelet aggregation and fibrin formation. Finally, platelets adhere to fibrin via the receptors GP IIb and GP IIIa forming a stable platelet fibrin plug.

2- The Coagulation System:

It's useful to consider the clotting mechanism as following two pathways: The intrinsic and the extrinsic pathway.

Intrinsic System:

Factor XII (Hageman Factor) circulates in the blood as an inactive zymogen. The contact system is initiated when factor XII binds to a negatively charged surface (damaged endothelium) where it's converted to factor XIIa by autoactivation. Surface bound factor XIIa activates prekallikrein (PK) to kallikrein, which reciprocates by accelerating the activation of factor XII to factor XIIa. The former reaction is enhanced by high molecular weight kininogen (HMWK), which also acts as a cofactor for the activation of factor XI by factor

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XIIa. Factor XIa activates factor IX in a calcium dependant reaction. Factor IX (like II, VII and X) is rich in glutamic acid residues, which are transcarboxylated by vitamin K to form alpha-carboxyglutamic acid. The modified vitamin K-dependant protein is then able to bind calcium ions, which in turn act as bridges binding the factor IX molecule to phospholipid. Factor X is then activated to factor Xa by a reaction which takes place on the platelet cell surface and involves a complex of factor IXa, thrombin-activated factor VIIIc, calcium ions and phospholipid.

Extrinsic System:

The conversion of factor X to factor Xa via the extrinsic system involves tissue factor (TF), factor VII and calcium ions. The active component of TF released from cells is thought to be a lipoprotein complex, which activates factor VII to factor VIIa in the presence of calcium ions. The lipid of the TF plays an additional role by adsorbing factor X onto its surface and thereby enhancing the reaction between factors VIIa, X and calcium ions. Factor VII can be also activated by factor IXa and XII fragments (XIIF).

Common Pathway:

This involves the conversion of prothrombin (II) to thrombin (IIa), clotting fibrinogen (I) to fibrin and the stabilization of the fibrin clot. Factor Xa formed by either the intrinsic or extrinsic system forms a cell surface complex with phospholipid, calcium ions and thrombin activated Va. This complex cleaves prothrombin into thrombin and prothrombin fragments 1 and 2. Thrombin converts fibrinogen to fibrin and also activates factors VIII, V, and (Positive Feedback) XIII. The

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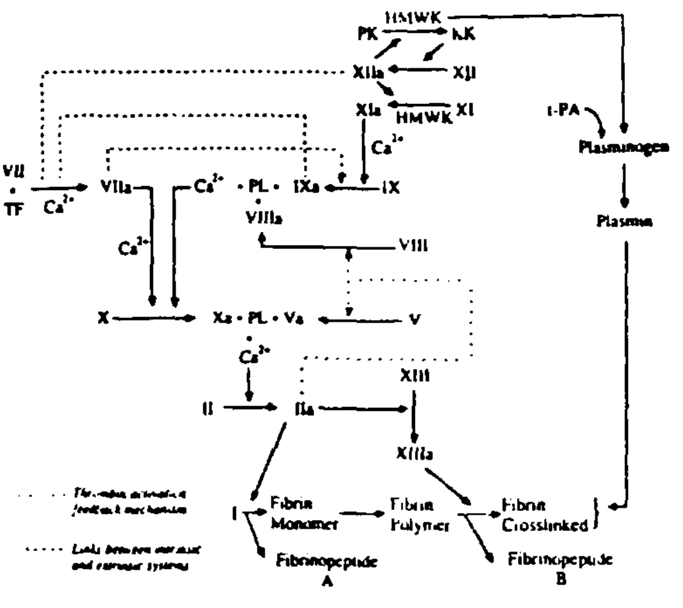
conversion of soluble fibrinogen to insoluble fibrin involves the thrombin-mediated cleavage of peptides A and B from the amino-terminal ends of the B-Beta and A-alpha chains of fibrinogen. The resulting fibrin monomers polymerize into a meshwork of fibrin. Factor XIII (Previously activated to factor XIIIa by thrombin) stabilizes the fibrin clot by cross-linking adjacent fibrin strands.

Two positive feedbacks should be noted in the coagulation cascade which can result in explosive thrombin generation. Factor IXa activates factor VII (linking the intrinsic and extrinsic systems), while thrombin promotes its own formation by activating factors V and VIII.

Figure 1: The factors involved in the formation of a stable fibrin clot and their interactions.



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