Risk factors & Maternal Morbidity & Mortality of massive primary Postpartum Haemorrhage in Omdurman Maternity Hospital

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بسم الله الرحمن الرحيم

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كربه ووضعته
Contents

Dedication ....................................................................................... I
Acknowledgment ........................................................................ II
Abbreviation................................................................................ III
English abstract ........................................................................... IV
Arabic abstract ........................................................................... VI
List of figures .............................................................................. VIII
List of tables ............................................................................... IX

Chapter one

Introduction and literature review ........................................... 1
Objectives ..................................................................................... 41

Chapter two

Patients & Methods ...................................................................... 42

Chapter three

Results ........................................................................................ 44

Chapter four

Discussion .................................................................................... 67
Conclusion ..................................................................................... 71
Recommendations ......................................................................... 72
References ..................................................................................... 73
Appendix ....................................................................................... 82
Dedication

TO:

My Father

My Mother

&

My Husband
Acknowledgment

I am extremely grateful to my supervisor professor Abdel Salam Gerais for his continuous support and guidance; I wish to thank him for his suggestion, advices, and efforts throughout the period of this study.

One person has been present from the conception of this study to its completion, criticizing, reading, and advising. My words cannot express my gratitude to Dr. Mohammed Awad for his help.

I also must express my appreciation and thanks to Dr. Nabawia and Dr. Mutasim Abdul Raheem for their great help and support in the collection of the data.

I am grateful to Dr. Ahmed Hassan who stood patiently with me the long hours of writing and typing this thesis.

Finally, I am extremely grateful to my husband for his patience, understanding, encouragement, and continuous support.
# Abbreviation

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>BP</td>
<td>Blood pressure.</td>
</tr>
<tr>
<td>C.E.M.D</td>
<td>Confidential enquires into maternal deaths.</td>
</tr>
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<td>C.P.C.D</td>
<td>The coherence pregnancy and child birth data base.</td>
</tr>
<tr>
<td>C/S</td>
<td>Caesarian section.</td>
</tr>
<tr>
<td>C.N.S</td>
<td>Central nervous system.</td>
</tr>
<tr>
<td>C.T</td>
<td>Computer tomography.</td>
</tr>
<tr>
<td>C.V.P</td>
<td>Central nervous pressure.</td>
</tr>
<tr>
<td>D.I.C</td>
<td>Disseminated intravascular coagulation.</td>
</tr>
<tr>
<td>F.B.C</td>
<td>Full blood count.</td>
</tr>
<tr>
<td>G.T.I</td>
<td>Genital tract injury.</td>
</tr>
<tr>
<td>I.C.U</td>
<td>Intensive care unit.</td>
</tr>
<tr>
<td>I.M</td>
<td>Intra muscular.</td>
</tr>
<tr>
<td>I.V</td>
<td>Intra venous.</td>
</tr>
<tr>
<td>I.M.M</td>
<td>Intra myometrial.</td>
</tr>
<tr>
<td>M.R.I</td>
<td>Magnetic resonance imaging.</td>
</tr>
<tr>
<td>N.S</td>
<td>Normal saline.</td>
</tr>
<tr>
<td>N.T.G</td>
<td>Nitroglycerine.</td>
</tr>
<tr>
<td>P.P.H</td>
<td>Postpartum haemorrhage.</td>
</tr>
<tr>
<td>O.M.H</td>
<td>Omdurman maternity hospital.</td>
</tr>
<tr>
<td>R.C.O.G</td>
<td>Royal collage of obstetrician and gynaecologist.</td>
</tr>
<tr>
<td>R.P.O.C</td>
<td>Retained products of conception.</td>
</tr>
<tr>
<td>S.M.H</td>
<td>Saudi maternity hospital.</td>
</tr>
<tr>
<td>U.K</td>
<td>United kingdom.</td>
</tr>
<tr>
<td>U.S.A</td>
<td>United state of America.</td>
</tr>
<tr>
<td>V.D</td>
<td>Vaginal delivery.</td>
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<tr>
<td>V.S</td>
<td>Versus.</td>
</tr>
<tr>
<td>V.W.D</td>
<td>Von Willbran disease.</td>
</tr>
<tr>
<td>W.H.O</td>
<td>World health organization.</td>
</tr>
<tr>
<td>W.M.O.G.T.H</td>
<td>Wad Madani obstetrics and gynecology teaching hospital.</td>
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English Abstract

Postpartum haemorrhage is an important cause of maternal morbidity and mortality. In this study the incidence, risk factors, medical intervention, and short term maternal morbidity and mortality were studied.

This is a prospective, descriptive study carried out in Omdurman maternity hospital in the period of 1\textsuperscript{st} of December 2003 to thirty 1\textsuperscript{st} of March 2004.

Information was collected from the patients who developed postpartum haemorrhage (PPH) in the first 24 hours after delivery and needed blood transfusion in Omdurman maternity hospital using structural coded questionnaire.

Sixty-seven women were studied; those who developed PPH and did not need blood transfusion were excluded.

Most of the study population was in the age group between 30 and 39 years (47.76\%) and more than half were from urban areas (77.61\%).

The patients distributed almost equally in relation to parity (32.84\%- 34.32\%).

There was frequent association between PPH and anaemia (37.37\%), antipartum haemorrhage (28.36\%), pre-eclampsia (29.85\%), multiple pregnancy (13.43\%), and previous PPH (14.93\%).

There was no significant association between PPH and prolonged first stage of labour or augmentation of labour (P=0.199), (P=0.979) respectively.

Concerning the mode of delivery 47.76\% was spontaneous vaginal delivery, 11.94\% was instrumental delivery and 33.33\% was caesarian section.

Active management of the third stage of labour was done to 21(31.34\%) of the study population and the remaining 46(68.65\%) were managed conservatively.
The study found that uterine hypotonia is the major aetiological cause of PPH (65.67%), followed by genital tract trauma (23.88%) and retained placenta (10.45%).

Clinical intervention included both medical and surgical treatments. Oxytocin was the main drug used in the patients (95.52%). Ergometrine was used in 80.60% of the cases. The surgical treatment ranged between repair under general anesthesia (14.93%) and cesarean hysterectomy (13.43%).

Regarding the short term maternal morbidity and mortality, 2 patients (2.99%) developed acute renal failure, 8 (11.94%) patients developed DIC, 2 patients needed admission to the intensive care unit, and the maternal mortality was 3 (4.47%).
ملخص الاطروحة

تعتبر حالات التزيف مابعد الولادة من الاسباب المهمة لأمراض ووقائع الامهات.

تم في هذه الاطروحة دراسة نسبة حدوث، العوامل المؤثرة، التدخل العلاجي للفروض ما بعد الولادة.

هذه دراسة وصفية مستقبلية تم تنفيذها في مستشفى الولادة بامدرمان في الفترة ما بين 1-12-2003 حتى 31-3-2004.

جمعت المعلومات من المرضى الذين اصيبوا بالنزيف ما بعد الولادة خلال الأربع والعشرين ساعة الأولى من الولادة واحتاجوا لنقل دم وذلك باستخدام استبان في المستشفى المذكور.

تم جمع سبع وستون حالة من المرضى مع استبعاد حالات نزيف ما بعد الولادة والتي لم تحتاج لنقل دم.

معظم حالات الدراسة كانت في متوسط العمر مابين 30 - 39 زادت بنسبة 51.32%.

كثر من ثلثي الحالات كانت من المنطقت الطرفية وذلك بنسبة 77.61%.

لاحظ أن توزيع المرضي باعتبار الولادة كان متوازي وذلك بنسبة 32.84% - 34.32%.

وجد في الدراسة ان ثمة علاقة متكررة مابين نزيف ما بعد الولادة وكل من: فقر الدم، نزيف ما قبل الولادة، حالات ما قبل الاتكاس، حمل التوائم، والحالات التي سبق وأن أصيبت بنزيف ما بعد الولادة.

كما يوجد ان ليس هناك علاقة تربط بين طول المرحلة الأولي للولادة وترحيب الولادة وما بين نزيف ما بعد الولادة باعتبار آلية الولادة ونسبة نسبة: 67% كانت الولادة طبيعية، 11.29% كانت الولادة بالتدخل الأثري، 34.33% كانت ويلة قصيرة.

المعالجة الفعالة للمرحلة الثالثة للولادة أجريت في 21 (31.34%) من المرضى بينما 46 (68.65%) من المرضى خضعوا للعلاج التحفظي للمرحلة الثالثة للولادة.

وجدت الدراسة ان ارتفاع الحرم هو السبب الرئيسي للفروض ما بعد الولادة وذلك بنسبة 65.67% وقية الامراض كانت كما يلي: أذية الجهاز التناسلي بنسبة 39.5%، المشيمة المحتبة بنسبة 45.1%.

التدخل العلاجي شمل العلاج بالعقاقير والعلاج الجراحي، بالنظر الى العقاقير المستخدمة في حالات الدراسة: كان الاوكسيسوفين هو العقار الرئيسي حيث استخدم في 64 حالة بنسبة 95.52% واستخدم الارومترتين في 54 حالة بنسبة 80.60%.
بالنسبة للتدخل الجراحي: تراوح بين عمليات إعادة تأهيل تحت التخدير الكامل بنسبة 14.92% والأرازة القيسارية للرحم بنسبة 13.43%.

وأما باعتبار حالات وفيات وإمراض الأمهات كانت النسب كالآتي: حالتان (2.99%) اصيبتا بالفشل الكلوي الحاد، حالات (11.94%) اصيبوا باعتلال التجلط الوعائي المنتشر، حالتان (2.99%) تم نقلهم لوحدة العناية المكثفة، ثلاث حالات (4.47%) سجلت وفيات الأمهات.
List of figures

Figure 1: Age distribution among the study population. 47
Figure 2: Distribution of the study population according to the residence. 48
Figure 3: Distribution of the study population according to the educational level. 49
Figure 4: Distribution of the socioeconomic status of the study population. 50
Figure 5: Distribution of the study population according to the parity. 52
Figure 6: Distribution of the study population according to severity of anemia. 54
Figure 7: Distribution of the study population according to the active management of the third stage of labour. 56
Figure 8: Distribution of the study population according to the aetiological factors of PPH. 60
Figure 9: Distribution of the study population according to the amount of blood transfused. 63
List of tables

Table 1: Gestational age distribution among the study population. 51
Table 2: Distribution of antenatal risk factors predisposing to PPH among the study population. 53
Table 3: Distribution of the study population according to the intrapartum risk factors. 55
Table 4: Correlation between the duration of the first stage of labour and aetiological causes of PPH. 57
Table 5: Correlation between augmentation of labour and aetiological factorsof PPH. 58
Table 6: Distribution of the study population according to the mode of delivery. 59
Table 7: Distribution of the study population according to the type of genital tract injury. 61
Table 8: Distribution of the study population according to the fetal birth weight. 62
Table 9: Distribution of the study population according to the medical treatment modalities used. 64
Table 10: Distribution of the surgical treatment modalities used among the study population. 65
Table 11: Distribution of the study population according to the maternal Morbidity and mortality 66
Introduction

Postpartum haemorrhage is a potentially life-threatening complication of both vaginal & cesarean delivery. In spite of marked improvements in management, early PPH remains a significant contributor to maternal morbidity & mortality both in developing countries & in hospitals with all that modern medicine has to offer. Although maternal mortality rate have declined greatly in the developed world, the most recent report on the UK confidential enquiries into maternal deaths (CEMD) reveal no consistent fall in deaths related to haemorrhage. In developing countries, it is responsible for an annual mortality of approximately 150,000 women per year.

Prevention, early recognition & prompt appropriate intervention are the keys to minimizing its impact.

Persons providing intra-partum care should routinely take steps to prevent PPH. Practices should be established to facilitate the identification of women who may be at particularly high risk for PPH & allow prompt intervention should excessive bleeding occur.

Definition

PPH was defined as blood loss greater than 500 ml following vaginal delivery & more than 1000 ml following a cesarean delivery (1). The world health organization (WHO) defines primary PPH as bleeding in excess of 500 ml in the first 24 hours following delivery (WHO 1990) & this definition is generally adopted in the UK. In certain countries such as Australia & Zimbabwe, the minimum cut-off for defining PPH is 600 ml (2).

The use of this definition presents practical problem, nearly a half of all women who are delivered vaginally shed that amount of blood or more, when measured quantitatively. Therefore, blood loss somewhat in excess of 500 ml by accurate measurement is not necessarily an abnormal event for vaginal delivery. Pritchard & associates (1962) found that about 5 percent of women delivering vaginally loose more than 1000 ml of blood.
They also observed that estimated blood loss is commonly only about half the actual loss\(^3\). These findings resulted in adoption of a broader definition for PPH; Any bleeding that results in signs & symptoms of haemodynamic instability, or bleeding that could result in haemodynamic instability if untreated is considered PPH\(^4\).

A decrease in postpartum haematocrit level greater than 10% of the prenatal value also can be considered PPH\(^4\) but this change is dependent on the timing of the test & the amount of fluid resuscitation given. In one study, the mean postpartum haematocrit decline ranged from 2.6 to 4.3 vol %; one third of women had no decline or an actual increase (via Combs & Colleagues, 1991 b). Women undergoing caesarian delivery had a mean drop in haematocrit of 4.0 to 4.2 vol %, but 20 % had no decline (Combs and co-workers, 1991 a)\(^3\). but this is a retrospective approach which may be useful in research protocols to assess risk factors or compare the effectiveness of treatment but is not very helpful to a clinician faced with excessive bleeding.

Comb’s has suggested a clinical definition of (need for blood transfusion)\(^5\). This definition is complicated by large variation in practice patterns and attitudes towards transfusion by both patients and physicians\(^6\).

PPH can be divided into:

1- Early PPH that occurs within 24 hours after delivery.

2- Late PPH that occurs 24 hours to 6 weeks after delivery\(^4\).

In general, early PPH involves heavier bleeding and greater morbidity\(^6\).

**Incidence:**

Life-threatening PPH occurs with a frequency of 1 per 1000 deliveries in the developed world\(^7\). Data from observational studies suggests that the incidence of primary PPH in developed countries is between 3.7 & 8.6%. Major PPH (defined as blood loss in excess of 1000 ml) occurs following 1.3% of all deliveries in the UK\(^2\). In the US the exact incidence of PPH is difficult to determine, a reasonable consensus is that 1-10% of pregnancies are complicated by PPH with the actual number in the range of 2-4%\(^4\).
Maternal mortality:

Obstetric haemorrhage was reported to be the fourth commonest cause of death i.e. 15 (11.6%) out of a total of 128 direct maternal deaths. Therefore, PPH still remains a significant contributor to maternal mortality in the UK. In UK, the number of maternal deaths from PPH has remained relatively unchanged from 1979 to 1993. In fact, the lowest figure for direct maternal death from this cause was six during the triennium 1985-1987 with mortality rate from PPH of 2.6 per million maternities, compared with eight during the lasts triennium (1991-1993) & mortality rate of 3.4 per million maternities (2). In the 1991-1993 triennium, 73% of haemorrhage related deaths were judged to involve substandard care (8). In the 1994-1996 triennial confidential inquire into maternal deaths in the UK primary PPH was responsible for 5 deaths (7). The direct pregnancy-related maternal mortality rate in the USA is approximately 7-10 women per 100.000 life births, national statistics suggest that approximately 8% of these deaths are caused by PPH (9).

One study of more than 2000 women in the United States revealed that obstetric haemorrhage is the cause of 13% of maternal deaths; PPH is thought to be the cause in one third of these cases (7).

In industrialized countries PPH ranks in the top 3 causes of maternal mortality (10).

Maternal mortality from PPH is much greater problem in developing countries. As many as 500.000 women die in child birth world wide each year at least 1 quarter of maternal deaths result from PPH, 99% of these deaths occur in developing countries (11). In developing world, several countries have maternal mortality rates in excess of 1000 women per 100.000 life births, & world health organization statistics suggest that 25% of maternal deaths are due to PPH accounting for more than 100.000 maternal deaths per year (12). A population-based survey conducted during 1982-1983 in rural Cambia has reported that PPH was the leading cause of maternal deaths, being responsible for 33% of such deaths. Adetoro (1987) also found that PPH was the largest single cause of maternal mortality (16% of direct maternal deaths) in a hospital based survey in Nigeria (2). A community-based investigation of causes of maternal mortality in rural Zimbabwe described the
leading cause of death as PPH (40 per 100.000). Another community based assessment of maternal mortality & causes of death in rural Senegal result in maternal mortality ratio range from 436-852 per 100.000, two-thirds of the maternal deaths were from direct obstetric causes haemorrhage being the most common \(^{(13)}\).

In Sudan, maternal mortality at OMH and SMH (1998 – 2001) was found to be 308 per 100.000 and obstetrical haemorrhage cause 21.1% of these deaths, PPH constitute 78.6% of haemorrhage \(^{(14)}\). Another study about maternal mortality in WMOGTH in five years (1998 – 2002), maternal mortality ratio was 802 per 100.000 life birth, obstetrical haemorrhage constitute 23.1% of all direct causes of death \(^{(15)}\). In Kasala hospital maternal mortality ration in two years (1997 – 1998) was 2351 per 100.000 life births and haemorrhage was responsible for 23.3% of direct maternal deaths \(^{(16)}\).

The difference in absolute mortality rates from PPH between non-industrialized & industrialized countries underscores the effectiveness of medical care in the reduction of mortality from this cause, and the need to improve this care further, as well as finding low-cost, implementable methods of reducing the problem in environments with limited medical facilities \(^{(17)}\).

**Aetiology of PPH:**

The aetiologies of early PPH are most easily understood as abnormalities or more of the four basic processes bleeding will occur if:

1- The uterus is not able to contact well enough to arrest the bleeding at the placental site.
2- Retained products of conception or blood clots.
3- Genital tract trauma may cause large blood losses postpartum
4- Coagulation abnormalities \(^{(6)}\).

These processes can be although of as the four T’s Tone, Tissue, Trauma & Thrombin \(^{(18)}\). It is not uncommon for more than one of these causes to be present in the same women with PPH \(^{(2)}\).
A. Uterine Atony:

The most common cause of postpartum haemorrhage is uterine atony (50% of cases) \(^{(19)}\), but placenta accreta is becoming more frequent. Failure of uterus to contract effectively will result in incomplete separation of the placenta, inhibition of contractibility of the uterine muscle to constrict the vascular channels in the placental bed & consequent excessive bleeding. The over-distended predicted uterus is very likely to be hypotonic after delivery. Thus, the woman with a large fetus, multiple fetuses, or hydramnios is prone to haemorrhage from uterine atony \(^{(3)}\). The aetiology of this phenomenon is unclear; overstretching may disrupt the bundles of actin-myocin in individual smooth muscle cells & decrease the efficiency of uterine contractions.

Metabolic factors may contribute to uterine atony. Hypoxia or acidosis from any cause may disturb myometrial metabolism. Patients who delivered after difficult or obstructed labour may suffer from uterine atony. The mechanism of uterine atony in these cases is complex & muscle exhaustion, lactate build up & glycogen depletion may be implicated \(^{(20)}\). The woman whose labour is characterized by uterine activity that is either remarkably vigorous or barely effectively is also likely to bleed excessively from uterine atony after delivery. Similarly, labour either initiated or augmented with oxytocin is more likely to be followed post delivery uterine atony & haemorrhage.

Another risk is if the woman has previously suffered PPH, mismanagement of the third stage of labour involves an attempt to hasten delivery of the placenta short of manual removal \(^{(3)}\). Drugs may have important effects on postpartum uterine tone \(^{(20)}\). Uterine atony causing haemorrhage can be anticipated whenever excessive concentrations of halogenated anesthetic agents are used that will relax the uterus \(^{(3)}\). Magnesium sulfate administered to prevent or treat seizures in preeclampsia or as a tocolytic agent, may result in uterine atony by impairing the calcium mediated activation of actin-myocin interaction. Beta-adrenergic tocolytic agents inhibit uterine contractility by increasing intracellular cyclic adenosine monophosphate \(^{(20)}\).
B. Haemorrhage from retained placental fragments:

Retained placenta is found in 2 percent of deliveries, the frequency of retained placenta is markedly increased (twenty-fold) at gestations < 26 weeks & even up to 37 weeks it remains three time more common than at term. At term 90 % of placentas will be delivered within 15 minutes. Once the third stage exceeds 30 minutes there is 10-fold increase in the risk of haemorrhage (21).

The increase in incidence of retained placenta affects predominantly women with induced preterm labour rather than women who deliver preterm after spontaneous labour. The reason for the failure of the placenta to separate in preterm deliveries is not known.

There are different clinical presentations of retained placenta, they vary from failure of placenta to be delivered spontaneously, requiring manual removal & causing minor blood loss to a placenta accreta that cannot be removed & causes sever bleeding requiring massive blood transfusion (20).

An abnormally adherent placenta, although an uncommon condition, assumes considerable significance clinically because of morbidity & at times, mortality from severe haemorrhage, uterine perforation & infection (3). Over the last 40 years the incidence of the placenta accreta has increase 10-fold.

This phenomenon is due to the fact that lower segment caesarian section appears to increase the risk of subsequent placenta praevia & these is a well-documented association between placenta praevia & previous caesarian section & placenta accrete (21). Zelop & Colleagues (1993) reported that abnormally adherent placentation caused 65 % of cases of intractable PPH requiring emergency peripartum hysterectomy at Prijham & woman’s hospital. Zaki & associated (1998) found an incidence of 1900 from 1990-1996 during which time there were over 23,000 deliveries at there hospital (3).

Abnormal placental adherence is found when residual formation is detected associated conditions include implantation in the lower uterine segment, over a previous caesarian section scar or other previous uterine incision or after uterine curettage. Zaki & associates 1998 found that 10 percent of 112 consecutive cases of placenta praevia had associated
accreta, Hardardottir & Colleagues 1996 observe that almost have of the placentas in women with a prior section delivery had adherent myometrial fibers detected microscopically (3).

C. Genital tract trauma:

Excessive bleeding from an episiotomy, laceration, or both causes are about 20% of PPH (19). Due to hypervascularity of the genital tract during pregnancy, any traumatic laceration can lead to excessive blood loss. The most troublesome cases commonly follow instrumental or operative delivery. The characteristic feature of bleeding from upper vagina & cervical tear is a steady loss of fresh red blood (2).

1. Perineal lacerations: usually result from extension of episiotomy incision (20), all except the most superficial lacerations are accompanied by varying degrees of injury to the lower portion of the vagina. Such tears may reach sufficient depth to involve the anal sphincter and may extend to varying depth through the walls of the vagina.

2. Vaginal lacerations: Isolated lacerations involving the middle & upper third of vagina unassociated with lacerations of premium or cervix are observed less commonly. Bleeding while the uterus is firmly contracted is strong evidence of genital tract laceration. Lacerations of the anterior vaginal wall in close proximity to the urethra are relatively common.

3. Injuries to levator ani: These are the result of over-distention of the birth canal. Muscle fibers are separated & diminution in their tonicity may be sufficient to interfere with the function of the pelvic diaphragm. If these injuries involve the pubococygens muscle urinary incontinence also may develop (3).

4. Injuries to the cervix: After vaginal delivery, the majority of women have lacerations &/or bruising of the cervix. Minor cervical lacerations are therefore extremely common. Deep lacerations & particularly those that involve the vaginal vault need to be managed in theatre under anesthesia. A laceration into the vault could extent forward to the bladder or
laterally towards the uterine artery at the base of the broad ligament \(^{(21)}\). Cervical lacerations are associated mainly with precipitous deliveries, the use of forceps, and with attempts to dilate the cervix manually \(^{(20)}\). In rare instances, however, the cervix may be entirely or partially avulsed from the vagina with coloporrhexis in the anterior, posterior or lateral fornices \(^{(3)}\).

5. Puerperal haematomas: Trauma during delivery may result in haematomas in the perineum or pelvis. These haematomas may be palpable and should be suspected if the patient has unstable vital signs & little or no external bleeding \(^{(4)}\). The incidence puerperal haematomas was found to vary from 1in 300 to 1 in 1000 deliveries (Gilstrap and Colleagues, 2001).

Nulliparity, episiotomy, and forceps delivery are the most commonly associated risk factors (Propst and Throp, 1998, Ridgway, 1995). However, haematomas develop following injury to a blood vessel without laceration of the superficial tissues. Puerperal haematomas may be classified as vulval, vulvovaginal, paravaginal, or retroperitoneal \(^{(3)}\). Also, haematomas are divided into those that lie above and those that lie below the levator muscle \(^{(21)}\). Vulval haematomas most often involve branches of the pudendal artery, including the posterior rectal, transverse perineal, or posterior labial artery, whereas paravaginal haematomas may involve the descending branch of the uterine artery (Zahn and Yeomans, 1990) \(^{(3)}\). A subperitoneal haematoma (broad ligament) is much less common 1 in 20,000 deliveries. They follow either spontaneous vaginal delivery, caesarian section or forceps operations \(^{(21)}\).

6. Ruptured uterus: Haemorrhage from a ruptured or lacerated uterus should be suspected of bleeding continues despite a contracted uterus and perineal, vaginal & cervical tears have been excluded \(^{(2)}\). Complete ruptured uterus can be a life-threatening emergency. Fortunately, the condition is rare in modern obstetrics, despite the increase in caesarian section rates. The incidence is about 0.3 percent.
Principle risk factors for ruptured uterus including:

- previous caesarian section (particularly "classical")
- multiparity
- oxytocic agents
- previous gynaecological surgery (e.g. myomectomy)
- congenital abnormality of the uterus (e.g. septate)

The risk of uterine rupture is significantly increased with a combination of risk factors. In one study in India 60 cases of rupture uterus managed in the last 5 years in the university hospital were reviewed, the factor responsible for rupture in admitted patients was either oxytocin induction / augmentation in scared uterus or obstetrical manipulation in unscarred uterus. Previous uterine scar was responsible for rupture in as high as 36.3 % cases. The commonest previous surgery was caesarian section mostly for non-recurrent causes. Obstructed labour was responsible for rupture in 26.6% cases while traumatic rupture was seen in 10 % of cases (22). Lower uterine segment dehiscence is the commonest current finding. The rupture of the lower segment may extend anteriorly into the back of the bladder or laterally towards the region of the uterine artery or even into the broad ligament plexus of veins, causing extensive haemorrhage & damage. Posterior rupture of the uterus is uncommon but can occur with previous uterine surgery or intrauterine manipulation (21).

7. Uterine inversion: is a catastrophic complication of the third stage of labour. It is the prolapse of the fundus to or through the cervix so that the uterus is in effect turned inside out if the uterus is inverted but does not protrude through the cervix, the invasion is incomplete. In complete inversion, the fundus has prolapsed through the cervix. Occasionally, the entire uterus may prolapse out of the vagina. Puerperal invasion has also been classified on the basis of its duration. Acute inversion occurs immediately after delivery & before the cervix constricts. Once the cervix constricts, the inversion is termed subacute. Inversion is noted more than 4 weeks after delivery (19).
Uterine inversion occurs approximately once every 2000 deliveries. It is more likely to occur with vigorous cord traction and with Grede's maneuver (manual compression of the uterus to encourage delivery of the placenta) but can occur in the absence of mismanagement. It is more likely in primiparous patients and where there is a fundal placenta. Uterine inversion is associated with haemorrhage in over 90 percent of cases & shock is its most common complication (40 percent) (21). In the past, it was stated that shock tends to be disproportionate to blood loss. Careful evaluation of the effects from transfusion of large volumes of blood in such cases does not support this concept but instead makes it very apparent that blood loss in such circumstances was often massive but greatly underestimated (Watson & associates, 1980) (3).

D. Abnormalities of the coagulation system:

The second major mechanism of postpartum haemostasis involves the blood coagulation system. Coagulation is important in controlling bleeding from all spontaneous or surgical disruptions of the birth canal, small lacerations of the cervix, vagina or perineum, which are confined to the mucosa & seldom cause significant postpartum blood loss if blood coagulation is normal. However, if coagulopathy is present, even small lacerations may result in exsanguinations. Defects in the coagulation system are rather uncommon. However, it is important to detect them before delivery. The most common are disseminated intravascular coagulation (DIC), Von Willebrand’s disease, and alternation in platelets number or function. In study about obstetrical and gynaecological bleeding in the Royal Free hospital London, UK, that postpartum haemorrhage is very high in women with inherited bleeding disorders. In the centre, the incidence of primary PPH was 22% in carriers of haemophilia, 18.5% in VWD & 16% in FXI deficient women. Acquired haemophilia can be an unusual cause of sever and unexpected PPH with a very high mortality rate. Unexplained PPH that does not respond to general majors should alert clinicians to the possibility of bleeding disorders as a causative factor (23).

DIC is the most common bleeding disorder seen in obstetrics patients. In molar pregnancy, amniotic fluid embolism, endotoxic shock, and fetal death in utero, DIC is the
result of release of thromboplastin-like activity into the maternal circulation with subsequent activation of the coagulation & fibrinolytic systems. Consumption of coagulation factors probably initiates DIC in abruptio placentae and haemorrhagic shock. The etiology of DIC in severe pre-eclampsia is unclear but may involve, as a first step, activation of platelet aggregation at the site of endothelial damage.

The most common clotting factor deficiency in young women is Von Willebrand’s disease, an acquired autosomal recessive disorder resulting in relative, through not absolute, deficiency of factor VIII (20), which is a central component of haemostasis, serving both as carrier for factor VIII and adhesive link between platelets and the injured blood vessel wall (24).

Most patients have a family or personal history strongly suggesting coagulopathy, but a few patients have never experienced serious injury or surgery may be undiagnosed (20). The overall prevalence of VWD has been estimated to be as high as 1 percent of the general population, although the prevalence of clinically significant disease is probably closer to 1:1000 (24).

Deficiency in platelet number or function may occur in pregnancy (20). Asymptomatic thrombocytopenia occurs near or in the peripartum period in about 5 percent of normal pregnancies and thrombocytopenia sometimes severe, occurs in about 15% of women with pre-eclampsia (24). A form of severe pre-eclampsia, the HELLP (hemolytic anemia, elevated liver enzymes, low platelet count) syndrome, has thrombocytopenia among its characteristics features. Fortunately, seldom is the platelet count less than 50,000 / mm, a level at which thrombocytopenia result in poor haemostasis. Immune thrombocytopenic purpura, an autoimmune disease often diagnosed in young women, is caused by circulating IgG antibodies to platelet antigens. These patients may have profound thrombocytopenia & are at risk of delivering infants with thrombocytopenia. Defects in platelet function occur with aspirin ingestion; aspirin inhibits the enzyme cyclooxygenase and prevents the formation of thromboxane and often procoagulants that are potent stimulators of platelet aggregations (20).
Risk factors of PPH:

Due to possible rapid progression of massive blood loss following established PPH and the high case fatality rate (Greenwood et al 1987), preventive measures are preferable to interventions, a possible approach to reduce the risk of PPH is to identify risk factors associated with this complication in order to improve the effectiveness of antenatal screening (1993) \(^{(25)}\). Those women identified to be at the highest risk of having PPH should ideally be confined in a maternity unit staffed with trained obstetric personnel but early recognition of the risk factors for PPH may aid patient management without necessarily preventing PPH.

The study by Combs et al (1991) was a case-control study design on hospital-based populations. The cases had PPH defined either as an arbitrary decrease of haematocrit in excess of 10 between admission in labour and the first postpartum day or receipt of a blood transfusion irrespective of the haematocrit change. Although there were limitations to these definitions of PPH, this study used logistic regression models to define the significant predictor factors. The study found that the significant associations with PPH were \(^{(5)}\):

1- Prolonged third stage of labour \((\geq 30\text{ min})\).
2- Pre-eclampsia.
3- Mediolateral episiotomy.
4- Previous PPH.
5- Twin pregnancy.
6- Arrest of descent of the presenting part during the second stage of labour \((<1 \text{ cm/hr})\).
7- Soft tissue laceration.
8- Forceps or vacuum delivery.
9- Asian or Hispanic ethnicity.
10- Midline episiotomy.
11- Multiparity.
In one study in UK (1993), the factors associated with major obstetric haemorrhage were analyzed using data relating to 37,497 women delivered in 1988, UK. 498 cases (1.33%) were complicated by haemorrhage of 1000 ml or more. Intrinsic factors associated with significant risk ratios (99% confidence intervals) included placental abruption 12.6, placenta previa 13.1, multiple pregnancy (4.46) & obesity (1.64), but not high parity. Significant risk factors related to obstetric management & delivery included retained placenta (5.15), induced labour (2.22), episiotomy (2.06) & birth weight 4 kg or more (1.90) (25).

A population-based case control study of risk factor analysis of PPH in Zimbabwe (1993), relative risks were estimated by multivariate logistic regression, low parity, advance maternal age and antenatal hospitalization were among the strongest risk factors, with more modest associations for history of poor maternal or perinatal outcomes and borderline anemia at the time of booking. No association with grand multiparity was found. There conclusion: these findings confirm the importance of previously recognized factors such as low parity, poor obstetric history, anemia, and prolonged labour, but call into question the significance of grand multiparity (26).

The Scottish obstetric guidelines and audit project in the management of PPH (1998) recommendations, the risk factors associated with a substantial increase in the incidence of PPH include proven abruptio placentae (odds ratio 13), known placenta praevia (odds ratio 12), multiple pregnancy (odds ratio 5), pre-eclampsia / gestational hypertension (odds ratio 4). The risk factors are also associated with a significant (though smaller) increase in incidence of PPH, nulliparity (odds ratio 3), previous PPH (odds ratio 3), Asian ethnicity (odds ratio 2), obesity (odds ratio 2). The factors becoming apparent during labour and delivery and associated and increase risk of PPH, delivery by emergency caesarian section (odds ratio 9), delivery by elective caesarian section (odds ratio 4), retained placenta (odds ratio 5), mediolateral episiotomy (odds ratio 5), operative vaginal delivery (odds ratio 2), prolonged labour (>12 hours) (odds ratio 2), big baby (>4 kg) (odds ratio 2), pyrexia in labour (odds ratio 2) (8).
Leiomyomas may interfere with effective uterine contraction immediately after delivery; therefore, the possibility of PPH should be anticipated (19). Some of these factors are prolonged third stage of labour, episiotomy, forceps, anaesthesia, can be prevented or modified by the obstetrician attending the delivery, others cannot be changed, but their presence should alert the obstetrician to the potential for postpartum bleeding & make him take precautionary measures for adequate management of the problem (20).

**Prevention**

Prevention of haemorrhage is preferable to even the best treatment; all patients should be evaluated for risk of PPH.

"A" *Antenatally (during pregnancy):*

1. All women should be offered screening for anemia on at least one occasion during pregnancy
2. Repeat screening for anemia early in third trimester is recommended to assess the effect or supplementation &/or treatment of underlying disease.
3. Iron supplements (together with folic acid) should be offered to all pregnant women with anemia. In areas with high prevalence of malaria, prophylaxis or presumptive treatment for malaria should be provided in addition to these supplements.
4. All women should be screened for hypertension and proteinuria at each antenatal clinic visit—those at highest risk require more frequent screening (especially between 24-32 weeks).
5. It is unnecessary to screen all pregnant patients for thrombophilias but patients with a significant personal or family history should be assessed (27).
6. Hospital delivery for women with risk factors & consideration should be given to extra precautions such as i.v access, coagulation studies, cross-matching of blood and anesthesia backup. Referral to tertiary center should be recommended for some high risk patients (6).
“B” Intrapartum (during labour):

1- Avoid prolonged labour by proper monitoring to the progress of labour (26).

2- Avoid lacerations by proper management of the second stage of labour & follow the instructions for instrumental delivery (26).

3- Episiotomy: bleeding from episiotomies may contribute to PPH (17). Large episiotomies may result in blood loss approaching that found in caesarian section about 1000 ml (20). In one randomized study of routine versus selective episiotomy in which blood loss was evaluated after delivery, significantly less blood loss occurred in women allocated restrictive episiotomy policy. Avoidance of unnecessary episiotomies may contribute to the reduction in blood loss after delivery (15). RCOG recommended that routine episiotomy should be abundant (27).

4. Active management of third stage of labour:

One of the primary objectives of management of the third stage of labour is prevention of PPH. Active management of the third stage of labour should be offered to women since it reduces the incidence of PPH due to uterine atony. It consists of interventions designed to facilitate the delivery of the placenta by increasing uterine contractions & to prevent PPH by averting uterine atony. The usual components include: administration of uterotonic agents, controlled cord traction, uterine massage after delivery of the placenta, as appropriate.

How to use uterotonic agents:

Within one minute of the delivery of the baby, palpate the abdomen to rule out the presence of an additional baby & give oxytocin 10 units i.m. Oxytocin is preferred over other uterotonic drug because it is effective 2-3 minutes after injection, has minimal side effects and can be used in all women. If oxytocin is not available, other uterotonics can be used such as: ergometrine 0.2 mg i.m, syntometrine (1 ampoule) i.m or misoprostol 400-600 mcg orally. Oral administration of misoprostol should be reserved for situations when safe
administration and/or appropriate storage conditions for injectable oxytocin and ergot alkaloids are not possible.

*How to do controlled cord traction:*

Clamp the cord close to the perineum (once pulsation stops in a healthy newborn) and holed in one hand. Place the other hand just above the women's pubic bone and stabilize the uterus by applying counter-pressure during controlled cord traction, keep slight tension on the cord and await a strong uterine contraction (2-3 minutes), with the strong uterine contraction, encourage the mother to push and very gently pull downward on the cord to deliver the placenta, continue to apply counter-pressure to the uterus. As the placenta delivers, walled the placenta in two hands and gently turn it until the membranes are twisted. Slowly pull to complete the delivery. Look carefully at the placenta to be sure none of it is missing. If a portion of the maternal surface is missing or there are torn membranes with vessels, suspect retained placenta fragments and take appropriate action.

*How to do uterine massage:*

Immediately massage the fundus of the uterus until the uterus is contract. Palpate for contracted uterus every 15 minutes and repeat uterine massage as needed during the first two hours. Ensure that the uterus does not become relaxed after uterine massage (11).

Prendiville and Elbourne contributed a meta-analysis on “active VC conservative third stage management” to the CPCD (29). This incorporated the results of five trials and concluded: "active management is associated with important reduction in clinically estimated postpartum blood loss, low haemoglobin levels postpartum & blood transfusion". However, active management is also associated with an increased incidence of nausea, vomiting, headache & hypertension postpartum. In one trial, retained placenta & secondary PPH were also commoner in the active management group. A further meta-analysis by Prendiville & Elbourne covered 11 trials comparing any prophylactic oxytocic versus none, regardless of other co-interventions (30). The conclusion was that oxytocics significantly reduce the risk of PPH by about 60% & the need for therapeutic oxytocics by about 70% & that “the current routine use of prophylactic oxytocics in the third stage of labour in the UK is justified".
Further meta-analysis by the same authors have covered compressions between various possible prophylactic oxytocic agents\(^{(31,32)}\). The first of these included size trial comparing syntometrine VS oxytocin & concluded that syntometrine was superior in that it reduced the odds of PPH by about a quarter when compared with oxytocin alone. This benefit was gained at the expense of increased side effects, notably a five-fold increase in the incidence of vomiting. The remaining two meta-analysis\(^{(33,34)}\), compared syntometrine VS ergometrine (7 trials) and oxytocin VS ergometrine (6 trials) and, taken together, suggest that ergometrine used alone is the least satisfactory of the possible prophylactic agents.

A final meta-analysis by Prendiville and Elbourne reviewed five trials comparing active VS conservative third stage management for “low risk” women only. The results were in line with those relating to active VS conservative management overall, and suggest that active management reduces the risk of PPH by as much as two thirds. Concerning the role of misoprostol for prevention of PPH, a double blind randomized trial conducted in Belgium, compared the effects of misoprostol (600 mcg orally) and methylergometrine (200 mcg IV) in 200 women after delivery of the infant. The authors conclude that methylergometrine and misoprostol provide nearly equal protection against PPH, but the misoprostol is associated with more side effects\(^{(35)}\). Other prospective, single-blind, randomized trial conducted in South Africa by Bamigboyea compared blood loss in 271 women receiving prophylaxis of 400 mcg of rectal misoprostol with 275 women receiving a non-identical placepo. PPH was defined as blood loss of at least 1000 ml and was detected in 4.8% of misoprostol group and 7% of the placepo group (a non-statistically significant deference)\(^{(36)}\). A third study conducted in the UK, it was uncontrolled, descriptive study evaluated oral administration of 600 mcg of misoprostol in 237 women, immediately after cord clamp, during term, singleton, and vaginal delivery. The authors concluded that misoprostol's efficacy in the prevention of PPH is comparable to that syntometrine, but that misoprostol is more stable and is associated with fewer side effects\(^{(37)}\). A new synthetic analogue of oxytocin, carbetocin is currently being studied to determine its place in prevention & treatment of PPH. This long-acting drug as a rapid
onset of action & a half life of 40 minutes as compared to oxytocin which is 4-10 minutes. Two Canadian randomized double blind studies have compared a single intravenous bolus of carbetocin to an infusion of oxytocin for women undergoing caesarian birth. Carbetocin was well tolerated & appeared to be effective or more effective than oxytocin as judged by the need for additional oxytocinic intervention \(^{(38-39)}\).

"C" postpartum:

Management of the forth stage of labour: Because many complications of birth occur or become evident during the first hour after delivery. This time has been referred to as the “fourth stage” of labour. The new mother should bee seen at least every 15 minutes by a trained labour & delivery nurse checking vital signs & looking for any evidence of uterine atony or PPH. The premium should be inspected for any signs of haematoma formation\(^{(40)}\). Early detection and prompt management without procrastination was the key to a successful outcome in the fourth stage of labour. Nearly tree-quarters were due to PPH \(^{(41)}\).

**Diagnosis and clinical presentation:**

The normal pregnant woman can withstand blood loss of 500 ml and even up to 1000 ml during delivery without obvious danger due to physiological cardiovascular and haematological adaptations during pregnancy, but certain women will become compromised with a relatively small blood loss \(^{(28)}\). This may include woman with gestational hypertension with proteinuria, women who are anemic or dehydrated and women of small stature \(^{(6)}\). In general, however, the degree of haemodynamic compromise or shock parallels the amount of blood loss:

1- phase of compensation:

Blood loss of 500-1000ml (10-15% of circulating volume). Sympathetic stimulation is the initial response to blood loss leading to peripheral vasoconstriction to maintain blood supply to vital organs.

Clinical symptoms & signs include palpitation, dizziness, tachycardia & maintain their blood pressure \(^{(6)}\).

2- Phase of decompensation:
Blood loss exceeds 1000–1500 ml (15-25% of circulating volume) lead to slight fall in blood pressure and symptoms of weakness, sweating and tachycardia (6).

Blood loss exceeds 1500-2000 ml (25-35%) of circulating volume lead to marked fall in blood pressure (systolic blood pressure 70-80 mmHg) and symptoms and signs of restlessness, pallor, oligouria (6).

3- phase of cellular damage:
This shock state is characterized by inadequate cellular perfusion leading to cellular damage & subsequent dysfunction or failure of major organ systems.

Losses of 2000-3000 ml (35-45% of circulating volume) will cause marked hypotension, with cardiovascular collapse, air hunger, anuria & sever shock (42).

Inadequately treated haemorrhagic shock results in prolonged tissue hypoxia & damage with the following effects:
1- Metabolic acidosis due to anaerobic metabolism initiated after lack of oxygen.
2- Arteriolar dilatation, caused by accumulation of metabolites leading to pooling & stagnation of blood in the capillaries & leakage of fluid into tissue.
3- Disseminated intravascular coagulation, caused by release of thromboplastin from the damaged tissues.
4- Cardiac failure: due to diminished coronary blood flow, in this phase death is imminent, transfusion alone is inadequate & if recovery from acute phase occurs residual tissue damage as renal &/or pituitary necrosis will occur (28).

PPH usually manifests with such rapidity that diagnostic procedures are almost entirely limited to a physical examination of the involved structures. Assessment of uterine tone & size is accomplished using a hand resting on the fundus & palpation the anterior wall of the uterus. The presence of a boggy uterus with either heavy vaginal bleeding or increasing uterine size establishes the diagnosis of uterine atony. The presence of uterine atony & resulting haemorrhage usually prevents the diagnosis of PPH from other causes because of an inability to visualize other sites. For this reasons, and because of the rapidity of blood loss secondary to atony, management & control of atony is paramount.
If the placenta has been delivered, inspection findings suggest whether portions of it have been retained, if it is undelivered or if retained clots or placental fragments are distending the uterus & bleeding is persisting despite appropriate ingoing treatment, manual exploration & removal should be undertaken. This is simultaneously therapeutic by emptying the uterus & permitting contraction while also aiding in the diagnosis of placenta accrete & uterine rupture. Cervical & vaginal lacerations may also be palpated at this time. If uterine atony has been controlled & bleeding from the uterus is minimal, careful inspection of the lower genital tract reveals bleeding sites in this area. Palpation & inspection may also reveal haematomas that require treatment. The cervix & vagina should be completely visualized following all operative vaginal deliveries (10).

**Management of established PPH:**

Early recognition of PPH is very important factor in management. Clinicians should routinely observed women after delivery for signs of excessive bleeding.

A previous established plan of action for the management of PPH is of a great value when a preventative measures failed. It is critical that these practices be familiar to all stuff in the maternity care facility and that precautions have been taken to ensure the availability of appropriate equipment, drugs & personnel in case of PPH (6).

Once PPH has been identified, management may be considered to involve four components. All of which must be undertaken simultaneously:

- Resuscitation.
- Monitoring & investigation.
- Arresting the bleeding.

**Resuscitation:**

The cornerstones of resuscitation following PPH are restoration of both blood volume & oxygen carrying capacity (8).

- At least two peripheral lines are established using cannulae of at least 14 gauge
• Setup central venous pressure monitoring & possibly an intra-arterial pressure display also

• Head down tilt.

• Oxygen by mask at 8 liters/min.

• Volume replacement must be undertaken with a acknowledgment that blood loss is often grossly under-estimated.

Compatible blood is the best fluid to replace blood loss & should be transfused as soon as available.

Perform the initial resuscitation with large volumes of crystalloid solution, either normal saline (NS) or ringer lactate solution, through peripheral sites. Normal saline is a reasonable solution in the labour ward setting because of its low cost & compatibility with most drugs & blood transfusions. The risk of hyperchloremic acidosis is very low in the setting of PPH. If large amounts (>10L) of crystalloid are being infused, a change to Ringer lactate solution can be considered. The loss of 1L of blood requires replacement with 4-5L of crystalloid because most of the infused fluid is not retained in the intravascular space but instead shift to the interstitial space. This shift, along with oxytocin use, may result in peripheral oedema in the days following PPH. Because a large portion of crystalloid fluid volume is lost to the interstitial space, the use of colloids in resuscitation has been examined. These solutions are largely retained within the intravascular space & include albumin, dextran, hydroxyethyl starch, & modified fluid gelatin. A meta-analysis in the Cochrane Library comparing resuscitation with colloid solutions versus crystalloid favored the use of crystalloids with respect to mortality (Choi, 1999: Alderson, 2000). For albumin or plasma protein fraction compared with NS, 18 trials reported data on mortality in 641 patients. The pooled relative risk from these trials was 1.52. The NS groups had a 1% mortality rate, versus an 11% mortality rate in the colloid group. For dextran compared to NS, 8 trials compared reported data on mortality in 668 patients. The pooled relative risk was 1.24. Two other recent meta-analysis on the same topic reach the same conclusion (10).

If fully x-matched blood is unavailable by the time 3.5 liters of clear fluid have been infused, then the best available alternative should be given group “O” negative blood may
be the safest way to avoid mismatched transfusion in an acute emergency, this might offer the only means of restoring oxygen carrying capacity within an acceptable time scale. The minimum number of units to be maintained on site should be agreed within local protocols & should reflect the likely period of delay in the arrival of further supplies should a dire emergency arise. While acknowledging the general principle that results of coagulation studies & the advice of a haematologist should be used to guide transfusion of coagulation factors.

Monitoring & investigation:

- A 20 ml blood sample should be taken for blood grouping, cross matching & coagulation studies\(^{(2)}\).
- The F.B.C will include estimation of haematocrit & platelet count. The clotting screen should include prothrombin time, thrombin time, partial thromboplastin time & fibrinogen assay. Fibrin degradation products should also be measured. Interpretation of the results of these tests should be undertaken collaboration with the haematologist and in the light of the physiological fall in prothrombin & partial thromboplastin times in the third trimester.
- The confidential enquiry reports\(^{(43,44)}\) have repeatedly emphasized the importance of central venous pressure (CVP) monitoring to guide volume replacement.
- All patients with significant PPH should have a Foley catheter placed to monitor urine output.
- Continuous pulse & blood pressure recording (using oximeter, ECG & automated BP recording).
- Consider transfer to intensive therapy unit.\(^{(8)}\).
Management of the underlying cause (arresting the bleeding)

Excessive bleeding may occur before delivery of the placenta or following delivery of the placenta. The management of the bleeding will be different depending on when the bleeding occurs.

Bleeding before delivery of the placenta (retained tissue):

Bleeding during third stage of labour is usually the result of retained placenta. When bleeding is brisk & the placenta is still inside the uterus, an attempt is now made to deliver it with controlled cord traction & uterine counter-traction. Care must be taken because the risk of uterine inversion is greater if the uterus remains poorly contracted. Intra-umbilical injection of oxytocin has been used to hasten placental separation in retained placenta. A randomized controlled trial was done on 35 consequent women; indicate that intra-umbilical vein injection of oxytocin is not clinically useful for the removal of a retained placenta. Another study about the role of misoprostol to facilitate placental separation, in Taiwan. Concluded that 800mcg of misoprostol per rectum is a safe & effective technique & may be a useful alternative to manual removal of retained placentas. Performed manual removal if the placenta is not easily delivered or the cord is avulsed. Perform manual removal with a level of analgesia that matches the clinical urgency of the situation. The safety, predictability, & ease administration of nitroglycerine (NTG) have been firmly documented. In recent years, intravenous NTG has come to the attention of the obstetrician as a potent uterine relaxant. Intravenous NGT has been used to relax the uterus during manual extraction of retained placenta. In Japan, they present report of the use of an isosorbide dinitrate tablet for the purpose of uterine relaxation for manual extraction of a retained placenta. The tablet administered sublingually proved to be a rapid & effective uterine muscle relaxant for manual removal of the placenta without overt adverse effects.

Retained placenta caused by abnormal attachment of the uterus, the majority of cases the placenta can be peeled off the uterus without major difficulties, in few cases the placenta is firmly attached to the uterine wall & it is impossible to find an adequate plan of cleavage for its removal. This is placenta accreta & in majority of patients the best
treatment is to perform hysterectomy. A review in 1972 of 622 published cases of placenta accreta shows a maternal mortality 4 times larger when conservative treatment consisting of “piecemeal” removal & uterine packing was used in stead of hystrectmy. Today with better antibiotics, chemotherapeutic agents and means of surveillance, conservative treatment may produce different results & may be indicated in selected patients who desire preservation of their fertility (20).

• Bleeding after delivery of the placenta (uterine atony):

Bleeding after delivery of the placenta is usually resulting of uterine atony. Management must be preceded by careful clinical examination to ascertain that the uterine is indeed atonic & that other source of bleeding such as genital tract lacerations have been excluded (8). Uterine atony can be treated immediately by uterine massage &/or compression in addition to the administration of oxytotic medications (6).

**Uterogenic agents:**

Several drugs are available to treat uterine atony. They should employ in a methodical fashion, while maintaining uterine massage & compression. The currently available products include:

1- oxytocin:

Oxytocin is a synthetic hormone identical to that produced in the posterior lobe of the pituitary. This medication causes contraction of the uterus with its effect increasing with the gestation as oxytocin receptors develop. In small doses oxytocin increases the tone & frequency of contractions but in larger doses can cause tetany. It can be given IV or IM. For patient with active bleeding, a continuous infusion of saline or Ringer’s lactate with 20 units of oxytocin per liter should be infused. If circulatory collapse has occurred, 10 units may be given intramyometrially (IMM). Very few side effects are noted with oxytocin a side from occasional nausea & vomiting. Water intoxication is a theoretical risk rarely encountered. There are no contraindications to the use of this drug for PPH prevention or treatment (4).
2- Methylergonovine maleate:
   Methylergonovine maleate is an ergot alkaloid which produces titanic contractions of
   the uterus within five minutes of intramuscular injection. It is given IM in doses of 0.25
   mg, which may be repeated up to every 5 minutes to maximum dose of 1.25 mg. It may be
   also being given directly into the uterine muscle if necessary or as an IV bolus of 0.125 mg.
   This drug is known to cause peripheral vasospasm & can exacerbate hypertension. It also
   may cause nausea & vomiting. It should not be used when the patient has hypertension.
   Also used with caution in heart disease, mitral valve stenosis, venoarterial shunts, sepsis,
   obliterative vascular disease, hepatic or renal impairment (4).

3- Carboprost:
   Carboprost is a synthetic 15 methyl analogue of prostaglandin F2 alpha. It is given IM or
   IMM in doses of 0.25 mg which may be repeated every 15 minutes to a maximum dose of 2
   mg. it is an extremely effective agent for increasing uterine tone but may produce
   prostaglandin side effects including nausea, vomiting diarrhea, headache, hypertension &
   bronchospasm due to smooth muscle contraction. Carboprost also acts on the central
   nervous system thermoregulatory center, sometimes causing flushing, diaphoriesis,
   restlessness due to increased basal temperatures. It has been shown to cause oxygen
   desaturation due to increase pulmonary shunting. Carboprost should not used in patients
   with major cardiovascular, pulmonary, renal or hepatic dysfunction. In spite of these
   potential risks, serious side effects are rare & most are self limiting. Several case series
   reported in the literature show that the use of carboprost is extremely effective for
   persistent bleeding due to uterine atony. The success rate for controlling bleeding was
   between 84 & 96 percent in these reports (49,50).
   However, two case series from the US (51), comprising 26 & 237 cases respectively,
   report success in controlling haemorrhage, without resort to surgical means, in 85% & 95%
   of cases. Two of the four failures in the smaller series were associated with placenta
   accreta. Carboprost should currently be regarded as the prostaglandin of choice.
4- Misopristol:

The possible place for misopristol addition to conventional oxytocics for treatment of PPH requires further study \(^{(17)}\). In the study by O'Brien et al (1998), Haemorrhage that was unresponsive to oxytocin & ergometrine was controlled & contractions were produced within 3 minutes of rectal administration of 1000 mcg of misopristol \(^{(37)}\). The data obtained by El-Refaey et al. (1997) also demonstrate the effectiveness of 600 mcg of misopristol for the management of PPH \(^{(52)}\).

5- Vasopresin:

Vasopressin may be used at caesarean for control of placental site bleeding. It causes acute vasospasm, decreasing blood flow near the injection site allowing coagulation to occur. Lurie describes six cases of injection of dilute vasopressin for intractable bleeding due to placenta accrete, where bleeding ceased without need for further surgery \(^{(53)}\). Twenty units (one ml) of vasopressin is diluted with 100 ml normal saline giving a 0.2 units/ml solution, which s infiltrated one ml at a time subendometrially at the bleeding site. It is very important that the solution is correctly diluted & that the needle not be in a blood vessel because high doses or intravascular injection of vasopressin can cause arterial hypertension, bradycardia or death \(^{(54)}\).

Genital tract trauma:

Initial exploration may also reveal trauma including lacerations of cervix or lower genital tract, uterine rupture or uterine inversion. Genital tract trauma is the most likely cause if bleeding persists despite a well-contracted uterus. Use appropriate analgesia along with good lighting & positioning, which facilitates excellent exposure. If not already initiated, moving the patient to an operating room is reasonable at this time. Experienced assistants and an excellent circulating nurse are essential.

Directly visualize and inspect the cervix with the aid of ring forceps. Small, non-bleeding lacerations of the cervix do not need to be sutured. Suture any bleeding lesions longer than 2 cm. Use an absorbable, continuous interlocking stitch. Ensure that the stitch begins above the apex of the tear. Polyglycolic sutures have largely replaced catgut.
Lacerations of the vaginal vault must be well visualized & their full extent realized prior to repair. Lacerations high in the vaginal vault & those extending up from the cervix may involve the uterus or lead to broad ligament or retroperitoneal haematomas. The proximity of the ureters to the lateral vaginal fornices, & the base of the bladder to the anterior fornix, must be kept in mind when repair is undertaken in these areas. Poorly placed stitches can lead to genitourinary fistulas. Cervical & vaginal vault lacerations that continue to ooze or those that are associated with haematomas may be amenable to selective arterial embolization. Traumatic haematomas are rare & may be related to lacerations or may occur in isolation. Lower genital tract haematomas are usually managed by incision & drainage, although expectant management is acceptable if the lesion is not enlarging (Propst, 1998). Any bleeding vessels are tied off, & oozing areas may be oversewn. Vaginal packing may be useful following drainage & repair of a paravaginal haematoma. Remove the pack in 24-36 hours. Broad ligament & retroperitoneal haematomas are initially managed expectantly if the patient is stable & the lesions are not expanding (Lingam, 2000). Ultrasound, CT scanning, & MRI all may be used to assess the size & progress of these haematomas. Selective arterial embolization may be the treatment of choice if intervention is required in these patients. Use surgical procedures to evacuate the haematoma, & attempt to tie off any bleeding vessels. Consider involving a surgeon with extensive experience operating in the retroperitoneal space (10).

**Management of uterine rupture:**

Uterine rupture requires blood replacement & formal exploration by laprotomy under general anesthesia. Most uterine ruptures in the UK occur in the lower segment but may extent anteriorly into the bladder & uterus, laterally into the uterine artery or venous plexus in the blood ligament causing extensive bleeding. Having excluded any damage to the surrounding structures, uterine rupture is usually managed by performing hysterectomy. Although if may be possible to control the bleeding with repair of the rupture alone (2).
A decision to repair the defect or proceed with hysterectomy is made on the basis of the extent of the rupture, the patient desire for future child bearing, and the patient’s clinical deterioration (19).

**Management of uterine inversion:**

Uterine inversion is serious disorder can result in massive haemorrhage & maternal shock. If uterine inversion occurs & placenta remains attached to the uterus, it should be left in place until the uterus is restored to its normal position. Prompt recognition & restoration of the uterus to its anatomic position are the key elements for effective treatment. An attempt should be made to replace the uterus immediately with the placenta attached; the clinician should grasp the inverted fundus & gently exert upward force into the pelvis using the fingers & palm. If it appears that the lower uterine segment is contracted to a degree that impairs replacement, a uterine relaxing agent may be administered. Pharmacologic agents that may facilitate restoration of the uterine to its intrapelvic position include IV magnesium sulfate (2-gm bolus) or IV nitroglycerine (50-100 mcg). As relaxation is accomplished, the uterus may be manually replaced in its intra-abdominal position with gentle massage of the most cephalad portion of the exposed endometrial placenta unit. Once the entire uterus has been restored to its original position, the placenta may be manually extracted if the patient is haemodynamically stable. Once the placenta is safely removed, the likelihood of recurrent uterine inversion can be reduced by administration of uterotonic agent such as intravenous oxytocin or intramuscular methylated PGF2α (55). If simple repositioning fails, O'sullivan suggested treating uterine inversion with hydrostatic pressure. Two liters of saline at body temperature are placed on an infusion stand & kept approximately 2 meters above ground level. The nozzles of two long rubber tubes are placed in the posterior fornix of the vagina. Whilst fluid is allowed to follow quickly, it is escape is prevented by blocking the introitus by using the operator’s hand (21).

Failure of conservative steps to effect replacement of the uterus may necessitate surgery. The Huntington produce requires laparotomy with progressive traction on the
cervical ring to gradually restore the uterus to its normal position. The Hultain procedure also requires laparotomy with a posterior relaxing myometrial incision placed to facilitate correction of the inversion with subsequent repair of the posterior incision. It is unusual to need laparotomy to reduce uterine inversion\(^{(55)}\).

**Management of patient with hereditary coagulopathies:**

In case of mild hemophilia A & type I Von Willibraud, there is well established evidence that the use of desmopressin, before surgery or for treating severe hemorrhage, normalized bleeding time & factor VIII levels & is clinically efficacious\(^{(56)}\). Although the evidence is less clear, desmopressin has been used successfully to prevent or stop bleeding in patients with congenital defects of platelet function, with hemostatic abnormalities associated with chronic liver disease, & with those induced by therapeutics use of antiplatelet & anticoagulant\(^{(39)}\).

But if the patient has coagulopathy, consider the transfusion of fresh frozen plasma. And if the patient is thrombocytopenic, consider platelets transfusion\(^{(4)}\).

**Management of intractable PPH:**

For the proportion of women not responding to initial management steps, a multidisciplinary team should be assembled including a second obstetrician or surgeon, anesthesiologist, and the associated staff from the operating room, blood bank & intensive care unit. If invasive radiology services are available consideration may be given to angiographic embolization\(^{(6)}\).

While such arrangements are being made, bimanual compression and massage, uterotonic drugs should be continued and consider the following:

1- **Uterine packing:**

Uterine packing was first described in the 1800s & was practiced by many obstetricians & supported by most major obstetrical textbooks\(^{(57,58)}\). It fell out of favor in the 1950s because if was felt that it was not “physiologic” and that it may mask trauma &
ongoing bleeding and cause infection. There is very little evidence in the literature to support or refute these fears; several small retrospective reviews in the literature indicate it may be useful in certain PPH situations. The technique involves packing the uterine cavity completely and uniformly with mesh gauze. The patient is given antibiotics and the pack is left in place for 24 hours while fluid and blood component replacement is completed. Uterine packing may be particularly useful when surgical treatment is unavailable at the current site or when the women are too unstable to undergo surgery at that time. In recent years, however, several modifications of this procedure have allayed these concerns. Balloon tamponade using either a Foley catheter or Sengstaken-Blakemore tube has been shown to effectively control post partum bleeding & may be useful in several settings: uterine atony, retained placental tissues, & placenta accreta. Both the Foley catheter & Sengstaken-Blakemore tube have open tips, which permit continuous drainage from the uterus. Furthermore, if the concern for concealed haemorrhage still exists, ultrasound can more effectively detect a developing haematoma when the contrast is a fluid-filled balloon as opposed to blood –saturated gauze. Thus, this technique has the advantage of being not only therapeutic but also diagnostic when use in combination with ultrasound in differentiation the various etiologies. Additionally, if intrauterine blood loss exceeds 5 cm/sec, the actual site of arterial bleeding can be pinpointed sonographically using power angiography mode against the contrast of the fluid-filled balloon.

4- Uterine artery / internal iliac embolization:

Angiographic embolization techniques were first described for the management of bleeding with PPH in 1979. With more than 150 cases in the literature, selective arterial embolization has reported success rates of up to 97%. This technique has been described in the control of postpartum vaginal wall haematomas, abdominal pregnancy, retroperitoneal haematomas, and miscellaneous types of post partum & postcaesarian hysterectomy haemorrhage using apercutaneous approach under local anesthesia, an aortogram is performed to identify the anatomy of the pelvic vessels, as well as to identify specific bleeding sites. The internal
iliac artery is then entered under fluoroscopy and a specific bleeding site is identified by the appearance of a “blush” or pooling of the contrast media in an extra-vascular site. The bleeding vessel is then selectively catheterized & occluded. Hemostasis may be documented by fluoroscopy, multiple vessels including collaterals, may be occluded in this manner.

The primary disadvantage of angiographic technique involves the usual need to transfer a bleeding patient to the radiology suite. Thus such techniques are not useful in sitting of rapid life-threatening haemorrhage, where a surgical approach is mandated (62). Other complications include fever, buttock ischemia, haematoma, vascular perforation, and infection. One woman developed uterine necrosis and sepsis 53 days following SAE for PPH and required hysterectomy for definitive treatment (60).

**Surgical approaches:**

The usual approach to the management of PPH unresponsive to non-operative measure is sequence of surgical procedures of increasing invasiveness (17). These include laprotomy with:

1- Pressure occlusion of the aorta.
2- Uterine artery ligation.
3- Internal iliac artery ligation.
4- Lynch brace suture.
5- Transverse imbricating sutures.
6- Peripartum hysterectomy.

**1-pressure occlusion of the aorta:**

Immediate temporary control of pelvic bleeding may be obtained at laparotomy by pressure occlusion of the aorta, which will provide valuable time to treat hypotension, obtain experienced assistants, identify the source of bleeding, and plan the operative
procedure. In the young and otherwise healthy patient, pressure occlusion can be maintained for several minutes without permanent sequel\(^{(19)}\).

2-**Uterine artery ligation:**

Water's\(^{(63)}\) first described this procedure in 1952 and others have subsequently reported success rates of 18-19%\(^{(64,65)}\). Surgical ligation of the uterine artery is an important part of the obstetrician’s armamentarium. The ability to properly perform this procedure may reduce blood loss associated with uterine rupture or lacerations and may under certain circumstances, permit uterine conservation. The largest experience in the literature is O'leary's report of 30 years experience with 265 patients, with 96% success rate\(^{(66)}\). A large study reports 100% effectiveness in 103 patients with intractable PPH using a stepwise approach to uterine devascularization, which begins with unilateral uterine vessel ligation and may include subsequent ovarian vessel ligation\(^{(67)}\).

Uterine atony was the major indication for vessel ligation. The incidence of significant complications was approximately 1%, and appeared to be associated with operator inexperience. No urologic injury was observed\(^{(62)}\). In the most cases of intractable PPH, uterine vessel ligation should be among the first surgical steps attempted as it is simple to perform can be done quickly. Advantages over internal iliac ligation include easier dissection, lower complication rates, more distal occlusion of arterial supply with less potential for rebleeding because of collateral and high reported rates of success in controlling haemorrhage\(^{(6)}\).

3-**Internal iliac artery ligation:**

Ligation of the internal iliac arteries is a procedure originally described in 1800s for management of bleeding from pelvic tumors, and applied more recently in obstetrical haemorrhage\(^{(6)}\).

In the United States, internal iliac artery ligation was first described in the late nineteenth century by Kelly, who used it to control bleeding associated with carcinoma of
the cervix, subsequently, the procedure has been most commonly used in the control of PPH (Clark & colleagues, 1985 b; Evans & Mc Shane, 1985). Branches of the anterior division provide the major blood supply to organs of the female pelvis. Reports of term pregnancy after bilateral ligation of internal iliac, uterine and ovarian arteries attest to the abundant collateral blood supply of the female productive tract (Mengert and Colleagues, 1969) (62). In one study in India 1998, internal iliac arteries were ligated in 15 cases of PPH, 4 cases of secondary haemorrhage after hysterectomy over a period of 5 years in a general hospital at Calutta. Concluded that the method was effective in 75% of atonic PPH and in majority of PPH due to other causes including caesarian section. The procedure was simple and did not involve any hazard (68).

Unilateral iliac artery ligation reduces distal ipsilateral blood flow by only half. A more important clinical effect is an 85% diminution of pulse pressure distal to the ligation, thus changing the haemodynamics of the distal arterial tree to one more resembling those of a venous system and amenable to haemostasis via simple clot formation (Burchell 1968). These are several uncontrolled reports that have examined the potential efficacy of bilateral internal iliac artery ligation to control obstetric haemorrhage and to avoid hysterectomy. (Clark & Colleagues, 1985 b; Evans & Mc Shane, 1985) reported success rates have ranged from 25-60%. Data from recent series indicate that ligation is successful in avoiding hysterectomy in approximately half of the cases associated with uterine atony & placenta accreta. Ligation may be far less successful when a uterine or broad ligament laceration is encountered, although occasional efficacy may be seen in these women as well. In some cases, identification & ligation of lacerated vessels is facilitated by first performing internal iliac ligation.

In series of 19 women undergoing bilateral iliac artery ligation for control of otherwise intractable obstetric haemorrhage, there was an increased incidence of ureteral injury and cardiac arrest from blood loss of women undergoing unsuccessful ligation followed by hysterectomy as compared with these undergoing primary hysterectomy without prior vessel ligation. However, ureteral injuries appear to have been surgically related to the hysterectomy rather than to artery ligation. The authors concluded that the
prolonged operative time & extensive blood loss after unsuccessful artery ligation may have led to less meticulous surgical technique during the subsequent hysterectomy resulting in ureteral injury (Clark & Colleagues, 1985b). Thus, complications associated with unsuccessful internal iliac artery ligation may be related to delay in hysterectomy rather than to the surgical procedure itself. Although extensive collateral circulation generally prevents ischemic complications, central pelvic ischemia, breakdown of the perineal skin & episiotomy site, and post ischemic lower motor neuron damage, with weakness of the lower extremities have been reported as sequelae of internal iliac artery occlusion (Braf & Knootz, 1979; Greenwood & associates, 1987). Although these complications are rare, the possibility of atypical collateral circulation must be kept in mind when evaluating the appropriateness of this procedure.

**Complications:**

1- Misidentification & ligation of the external iliac artery.
2- Laceration of the internal & external iliac vein.
3- Ureteral injury.
4- Retroperitoneal haemtoma.

The risk / benefit ratio of this procedure in cases of obstetric haemorrhage is probably acceptable only if three criteria are met. The women must be an hemodynamically stable, future child bearing must be an over whelining concern for the patient, and an experienced operator must be available, otherwise, hysterectomy may be the preferable procedure (62).

4-**Lynch brace suture:**

The B-Lynch suture initially described by Christopher B-Lynch in 1997. The theory behind this technique is the mechanical compression of uterine vascular sinuses prevents further engorgement with blood and continued haemorrhage. When used to treat atony and haemorrhage that does not respond to pharmacologic intervention, the B-Lynch appears to be very effective. To date, this technique has been used in a series of 11 patients, in each case successfully averting hysterectomy. Subsequently, two of these women had
uncomplicated pregnancies and were delivered at term. One of these patients had an elective C/S, and no adhesions or uterine defects were identified at the time of surgery. A third patient had an MRI and hysterosalpingogram following the B-Lynch suture and was noted to have normal uterine anatomy. A woman meets the criteria for the B-Lynch compression suture if bimanual compression decreases the amount of uterine bleeding by abdominal and perineal inspection (60). With this technique, an absorbable suture is placed in the lower uterine segment & then looped over the fundus when it is threaded through the lower segment posteriorly to the opposite site. The suture is then looped back over the uterus & passed through the lower segment anteriorly & then tied. This suture results in uterine compression (62).

5-Transverse Imbricating sutures:

Gilstrap & Colleagues (1999) described transverse, imbricating suture technique for the control of PPH, starting at the fundus, interrupted suture of non chronic are placed at approximately 3-4 cm intervals down to and including the lower uterine segment. When these individual sutures are tied, the uterus is compressed and gives the appearance of a contracted uterus. It may be necessary to place these sutures posteriorly also. This technique should only be done as a last resort prior to performing a hysterectomy. This technique may also prove useful for lower segment bleeding secondary to placenta praevia (62).

6-Emergency peripartum hysterectomy:

Emergency hysterectomy is the most common treatment modality when massive PPH requires surgical intervention. The incidence of emergency peripartum hysterectomy reported in the literature varies from 7 to 13 per 10,000 births (69,70), and is much higher after caesarian section than vaginal delivery. The most common indication for the procedure was placenta acreta or percreta. In a retrospective review of 123 cases of emergency peripartum hysterectomy from 1985-1990 in Los Angeles country, the most common indication was placenta accreta or percreta (49.6%) (69). The association of
placenta praevia & prior caesarian section with placenta accreta & risk of hysterectomy is well documented in the literature. Other frequently cited indications for emergency hysterectomy are rupture of the uterus, severe extension of caesarian section on incision; broad ligament haematoma after forceps, lacerated cervix/vagina after forceps or ventose and chorioamnionitis & subtotal hysterectomy has been advocate to reduce operative time & blood loss. In a retrospective review of emergency obstetric hysterectomy in Hong Kong 1993-1997, the indications for hysterectomy were uterine atony and placental disorders. There were one case of urinary bladder injury and two cases of DIC. There was no maternal mortality.

The advantages of emergency hysterectomy in the situation of massive haemorrhage are the ability to remove the source of bleeding and the familiarity of the obstetrician with the procedure of hysterectomy, which albeit more technically difficult in this situation is still familiar operation to any obstetrician/gynaecologist. The disadvantage of hysterectomy may include the loss of uterus in a woman who wishes to continue child bearing.

**Post hysterectomy bleeding:**

Unfortunately hysterectomy does not guarantee control of blood loss in severe PPH, bleeding may persist for the pelvic surfaces due to decreased coagulation combined with the trauma from prolonged manipulation. These small sites may be difficult or impossible to isolate & coagulate or suture. Bleeding vessels may retract deep into the pelvic retroperitoneal space and be difficult or impossible to isolate surgically.

Intra-abdominal packs have been used for continued bleeding from peritoneal surfaces when hysterectomy has been done, a consumptive coagulopathy exists, and there is continued wide spread bleeding. In this situation the pelvis is packed firmly with large laparotomy packs, which are then removed 24 hours later after correction of the coagulopathy. A variation in this method involves the use of a trans-vaginal pressure pack, in which Kerlix gauze is held in place in the pelvis by a sterile plastic bag & brought out through the vagina. Traction on the pack produces pressure against the pelvic floor.
pack can later be removed through the vagina (71). Specific vessels which haemorrhage persistently may be controlled with embolization procedures.

COMPLICATIONS OF PRIMARY PPH

1- Complications of blood transfusion:

   (A) Transfusion reactions:

   These may be the following problems:

   • **Incompatibility**:
   
   They are caused usually by the transfusion of red cells which are incompatible with an antibody present in the plasma of the patient. The most severe reactions occur with rapid intravascular destruction of incompatible red cells. This accompanied by fever & rigors even after the infusion of a small quantity. Haemoglobin is also excreted by kidney and whilst haemoglobin per se doesn’t produce renal damage, the presence of red cell stroma & other products resulting from antigen-antibody reaction, together with shock & poor perfusion, may result in renal failure. When extravascular lysis occurs, jaundice may become apparent after the transfusion (73).

   • **Simple pyrexial reaction**:
   
   In which the patient develops pyrexia, rigor and some increase in pulse rate. These are the result of “pyrogens” in the donor apparatus and are largely avoided by the use of plastic disposable giving sets.

   • **Allergic reactions**:
   
   The patient develops mild tachycardia & an urticarial rash, rarely an acute anaphylactic reaction may occur. This is the result of allergic reaction to plasma products in the donor blood. The reaction is treated by stopping the transfusion & giving an antihistamine drug.

   • **Sensitization to leucocytes & platelets**:
   
   The individual develops antibodies to donated white cells or platelets, which cause reactions with each transfusion. They may be minimized by giving packed red cells.
from which plasma & a puffy coat layers have been removed or by washing of donor cells. Aspirin, antihistamines or steroids may also be given to the recipient if necessary.

- **Immunological sensitization:**
  
  Only the ABO, Kell, & Rh (D) groups are considered for blood transfusion. Immune antibodies may be stimulated by transfusion, and may give rise to difficulties with compatibility tests or to haemolytic transfusion reactions.

(B) **Effects of massive transfusion:**

A massive transfusion, although a relative term, can be defined as the administration over a short period of ten units of blood to an adult.

Changes occur in stored blood which may result to decrease oxygen transport, stored blood is depleted of platelets and certain coagulation factors & dilution in the patient’s plasma may be a contributory cause to persist bleeding, acidosis may result from transfusion of large quantities of stored blood due to it is lower PH consequent on the citric acid present and the accumulation of lactic acid as a result of red cell metabolism, citrate ions are removed rapidly from the body by nucleated cells & the average adult can with stand 500 ml blood every 5 minutes without the need for supplementary calcium, the rapid administration of large quantities of blood at 4 degrees centigrade may significantly lower the blood temperature. The resultant hypothermia may impair citrate metabolism & may increase the rate of release of developing acidosis & hypocalcaemia (73).

(C) **Infections:**

There are four main reasons for blood transfusion causing infection in the recipient.

- Serum hepatitis virus may be from the donor and is usually a sever hepatitis arising approximately 3 months after the transfusion. It should be avoided by adequate verbal screening of the blood donor & by testing for the presence of the hepatitis. Associated antigen in the blood prior to transfusion. Post transfusion hepatitis may be also be caused by the transmission of viruses collectively known as non-A non-B, for which no assay procedure is available as yet (73).
• HIV infection can be transmitted by blood & blood products. All donors must be screened.

• Bacterial infection may result faulty storage. This arises most commonly from the donor blood being left in a warm room for some hours before the transmission is commenced. This allows proliferation of any bacteria, and transfusion of such infected blood may result in severe septicemia in the recipient & rapid death.

• Malaria can be transmitted by blood transfusion in areas where the disease is endemic. Malaria parasites remain viable in stored blood for 7-14 days (73). Whenever possible, donors should be screened & the disease eradicated before blood is obtained or given.

2- Disseminated intravascular coagulation:
This syndrome arises from the intravascular activation of procoagulant factors, chiefly thrombin and platelet aggregation adhesion leading to widespread thrombosis of the microcirculation of several organs, a consumptive coagulopathy and a secondary activation of plasminogen to plasmin causing a concurrent fibrinolysis. The net result is multiple organ failure (adult respiratory stress syndrome, acute renal failure, hepatic insufficiency and CNS changes). This is accompanied by a generalized bleeding tendency manifested as petechiae, ecchymosis and bleeding from the mucous membranes (gastrointestinal haemorrhage, haematuria, epistaxis).

The severity of the organ dysfunction & extent of haemostatic failure have been associated with grave prognosis. Libratory features include thrombocytopenia, reduced fibrinogen level, elevated levels of D-dimer & fibrin (0-gen) degradation products & prolonged partial thromboplastin, prothrombin & thrombin times (24).

3- Sheehan’s syndrome:
Severe intrapartum or early PPH is on rare occasions followed by Sheehan syndrome, which the classical case is characterized by failure in lactation, amenorrhea, atrophy of the breasts, loss of public and axillary hair, superinvolution of the uterus, hypothyroidism & adrenal cortical insufficiency. The exact pathogenesis is not well understood, because such endocrine abnormalities are not evident in most women who haemorrhage severely, in
some but not all instances of Sheehan syndrome, varying degrees of anterior pituitary
necrosis with impaired secretion of one or more atrophic hormones account for endocrine
abnormalities. The anterior pituitary of the some women who develop hypopituitarism after
puerperal haemorrhage does respond to various releasing hypothalamic function. Moreover,
whitehead (1963) identified specific atrophic changes in hypothalamic nuclei
histologically in some cases. Lactation after delivery usually, but not always, excluded
extensive pituitary necrosis. In some women, failure to lactate may not be followed until
many years later by other symptoms of pituitary insufficiency. In the series reported by
Ammin & Mathur (1994), the average duration of onset of symptoms was 5 years.

The incidence of Sheehan syndrome was originally estimated to be 1 per 10,000
deliveries (Sheehan & Murdoch, 1938). It appears to be even rarer today. Application of
tests of hypothalamic & pituitary function now available should identify milder forms of
the syndrome & define their prevalence (Grimes & Brooks, 1980). Bakiri & colleagues
(1991) used computed tomography to study 54 women with documented Sheehan
syndrome. In all these, the appearance of the pituitary was abnormal; the sella turcica was
either totally or partially empty (2).

**Prognosis**

The prognosis depends on the cause of PPH, its duration, the amount of blood loss, co
morbid conditions, and the effectiveness of treatment (4).
Objectives

1-To determine the incidence of massive primary postpartum haemorrhage in Omdurman maternity hospital.

2-To identify the risk factors predisposing to massive primary PPH.

3-To assess the level of clinical intervention in massive primary PPH.

4-To study short term maternal morbidity and mortality associated with massive primary PPH.
Patients & methods

Study design:
This is a prospective descriptive hospital based study carried out at Omdurman maternity hospital to study the incidence, the risk factors and short term maternal morbidity and mortality of massive primary PPH.

Study area:
Omdurman maternity hospital is a governmental hospital, located in the center of Omdurman city established in 1957 (Bayomi 1978), it has been the main separate maternity hospital in Sudan where the federal ministry of health supervises the training of doctors and midwives; it represents the different Sudanese social strata from rural to more affluent societies.

The hospital capacity is 13 labour observational beds, six delivery tables, 122 inpatients beds, and it is covered by senior obstetricians, obstetrics and gynaecology registrars, house officers, midwives, nurses and other paramedical staff. Also there is a consultant paediatrician and anaesthetists.

Study period:
The study was conducted in the period 1st of December 2003 to thirty 1st of March 2004.

Inclusion, exclusion criteria:
The study population was all patients who developed postpartum haemorrhage within 24-hours after delivery and required blood transfusion due to haemodynamic instability, the patients who developed PPH which didn’t necessitate blood transfusion excluded.
**Data collection:**

A structured questionnaire was designed; it includes information about the personal history (age, occupation, residence, education, and the socioeconomic status), obstetrical history, intrapartum and postpartum period. All these patients had been followed up during their stay in the hospital till they were discharged by daily morning enquiries and general medical examinations.

**Sample size:**

Total coverage of all patients who developed postpartum haemorrhage – during the study period - within 24 hours after delivery and required blood transfusion were included in the study which constitutes 67 patients.

**Data analysis:**

Some appropriate descriptive statistics like frequency tables, graph, cross tabulation to give summery and idea about the study population had been done.

The statistical analysis of the data was done using the computer SPSS program and a P-value < 0.05 at 95% confidence interval was used as the level of significance, also chi-square test was used.
Results

The total number of deliveries in Omdurman Maternity Hospital in the period from 1.12.2003 to 31.3.2004 (4 months) was 5649, out of this number, those who developed PPH which required blood transfusion were 67 patients which accounts for 1% from the total.

The majority of the study population (47.76%) were in the age group 30-39 years, (35.82%) in the age group 20-29 years, while (11.94%) in the age group <20 & only (4.48%) in the age group >40 (figure 1).

Most of the study population (77.61%) was from urban area especially those living in Omderman attachments area, (22.39%) were from rural areas (figure 2).

Figure 3: showed the educational level of the study population, (37.31%) was illiterate, (26.87%) had primary school level, (28.36%) reach secondary school level, & only 7.46% were graduated.

Concerning the socioeconomic state of the study population, the majority of patients were from low socioeconomic class (67.16%) while (31.34%) were from moderate socioeconomic class, just (1.49%) were high social class (figure 4).

Regarding the gestational age of the study population, the majority (67.16%) were in the gestational age 37-42 weeks, (17.91%) were less than 37 weeks, (1.49%) were more than 40 weeks gestational age, while 9(13.43%) were uncertain of their gestational age & they have no antenatal care (table 1).

In accounts to parity (32.84%) were nulliparous, (34.32%) were multipara & (32.48%) were grandmultipara (figure 5).

Table 2: showed the frequency & percentage of the antenatal risk factors predisposing to PPH among the study population, (37.37%) of the patients were anemic (Hb <11g/dl), (29.85%) were diagnosed cases of preeclampsia or gestational hypertension, (28.36%) had antipartum haemorrhage either sonographically proven case of placenta previa or clinically diagnosed case of placental abruption, (14.93%) had previous history of PPH and
(13.43%) were cases of multiple pregnancy; 9 sets of twins and one triplet pregnancy. Five patients (7.46%) had premature rupture of membranes, two patients (2.99%) had polyhydramnious & only one patient (1.49%) had fibroid with pregnancy diagnosed by ultrasound, there was no patients with hereditary coagulopathy among the study population.

Figure 6: Classifying the study population according to the degree of anaemia (68%) of them had moderate anemia, (20%) had mild anemia & (12%) had sever anemia according to the Indian council of medical research categories of anaemia\(^{(74)}\). When reviewing the intrapartum risk factors of PPH in the study group, (35.82%) had prolonged first stage of labour (more than 12 hours), while 914.93% had delayed in the second stage of labour (more than 2 hours) & (10.45%) had prolonged third stage of labour (more than 30 minutes)(table 3).

Figure 7: showed that (31.34%) had active management of the third stage of labour, (68.65%) had expectant management of the third stage of labour.

Table 4: correlate the duration of the first stage of labour to the aetiological causes of PPH, (97.17%) of the patients with first stage of labour more than 12 hours developed uterine hypotonia, (12.5%) developed genital tract injury, and (8.33%) had retained placenta, were as in patients with first stage of labour less than 12 hours (58.14%) had uterine atonia, (30.23%) developed genital tract injury, and (11.63%) had retained placenta. However, the difference between the two groups did not reach statistical significance (P=0.199).

In relating different aetiological factors of PPH to augmentation of labour, (66.67%) of augmented labour resulted in uterine hypotonia, (23.08%) had genital tract injury, and (10.25%) had retained placenta, while (64.29%) of patients who did not receive augmentation developed uterine hypotonia, (25.0%) developed genital tract injury and (10.71%) had retained placenta. However, the difference between the two groups did not reach statistical significance (P=0.970) (table 5).

Concerning the mode of delivery in the study population,(47.76%) were spontaneous vaginal delivery,(5.97%) were induced vaginal delivery, instrumental vaginal delivery were
of them (7.46%) were forceps delivery & (4.48%) were ventose delivery, cesarean section constitute (34.33%) of the study population (table 6).

Figure 8: showed the main aetiological factors of PPH in the study group, uterine hypotonia was the main cause constituting (65.67%), followed by genital tract injury (23.88%), retained placenta caused (10.45%) of the PPH in the study group.

Those patients who suffered from genital tract injury had different types of injury as shown in (table 7), cervical tears & lacerations accounted for (50.00%) of the injuries, (18.75%) were extended or malsutured episiotomy, (12.5%) were vaginal tears or lacerations, also perineal tears constituted (12.5%) of the injuries, and one patient (6.25%) had rupture uterus secondary to obstructed labor, no patient in the study group developed broad ligament haematoma.

When looking to the fetal birth weigh of the study population, (74.03%) had birth weigh between 2.5-4 Kg, (20.78%) were less than 2.5Kg and only (5.19%) were above 4Kg (table 8).

The amount of blood transfusion needed for the treatment of the patients varied as shown in figure 9; (53.73%) of the patients received 1-2 units of blood, (20.89%) received 3-4 units, and (14.93%) needed 5-6 units of blood, (10.45%) needed more than 6 units of blood.

Regarding the medical treatment required, syntocinon IV or infusion used in (95.52%) of the patients, Ergometrine IV or IM used in (80.60%) of the patients, while rectal misopristol was used in (7.48%), carboprost & prostaglandin E2 were not used for any of the patients (table 9).

Surgical treatment modalities required in the study group ranged from repair under general anesthesia in (14.93%), cesarean hysterectomy in (13.435) and ligation of the internal iliac artery in (1.50%) of the study population (table 10).

Table 11: showed the maternal morbidity & mortality of the study population, (11.94%) of patients developed DIC, (2.99%) had renal failure, (2.99%) of the patients admitted to ICU, and the maternal mortality caused directly by massive PPH was (4.47%).
Figure 1: Age distribution among the study population
Figure 2: Distribution of the study population according to the residence

- 77.61% rural
- 22.39% urban
Figure 3: Educational level distribution of the study population

- Illeterate: 27.34%
- Primary school: 26.87%
- Secondary school: 28.36%
- Graduated: 7.46%
Figure 4: Distribution of socioeconomic status of study population

- Low: 31.34%
- Moderate: 1.49%
- High: 67.16%

Legend:
- □ low
- □ moderate
- □ high
Table 1: Gestational age distribution of the study population

<table>
<thead>
<tr>
<th>Gestational Age</th>
<th>Frequency</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 37 weeks</td>
<td>12</td>
<td>17.91%</td>
</tr>
<tr>
<td>37 – 42 weeks</td>
<td>45</td>
<td>67.16%</td>
</tr>
<tr>
<td>&gt; 42 weeks</td>
<td>1</td>
<td>1.49%</td>
</tr>
<tr>
<td>Uncertain date</td>
<td>9</td>
<td>13.43%</td>
</tr>
<tr>
<td>Total</td>
<td>67</td>
<td>100%</td>
</tr>
</tbody>
</table>
Figure 5: Distribution of the study population according to the parity
**Table 2: Distribution of antenatal risk factors predisposing to PPH among study population**

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Frequency</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia with pregnancy</td>
<td>25</td>
<td>37.37%</td>
</tr>
<tr>
<td>Pre-eclampsia / gestational hypertension</td>
<td>20</td>
<td>29.85%</td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td>19</td>
<td>28.36%</td>
</tr>
<tr>
<td>Previous PPH</td>
<td>10</td>
<td>14.93%</td>
</tr>
<tr>
<td>Multiple pregnancy</td>
<td>9</td>
<td>13.43%</td>
</tr>
<tr>
<td>History of PROM</td>
<td>5</td>
<td>7.46%</td>
</tr>
<tr>
<td>Polyhydramnious</td>
<td>2</td>
<td>2.99%</td>
</tr>
<tr>
<td>Fibroid with pregnancy</td>
<td>1</td>
<td>1.49%</td>
</tr>
<tr>
<td>History of hereditary coagulopathy</td>
<td>0</td>
<td>0.00%</td>
</tr>
</tbody>
</table>
Figure 6: Distribution of the study population according to severity of anaemia

- Mild (10.0-10.9 g/dl) - 68%
- Moderate (7.0-10.0 g/dl) - 12%
- Severe (< 7.0 g/dl) - 20%
**Table 3: Distribution of the study population according to the intrapartum risk factors of PPH**

<table>
<thead>
<tr>
<th>Intrapartum risk factor</th>
<th>Frequency</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prolonged 1st stage of labour (&gt; 12 hrs)</td>
<td>24</td>
<td>35.82%</td>
</tr>
<tr>
<td>Prolonged 2nd stage of labour (&gt; 2 hrs)</td>
<td>10</td>
<td>14.93%</td>
</tr>
<tr>
<td>3rd stage of labour &gt; 30 minutes</td>
<td>7</td>
<td>10.45%</td>
</tr>
</tbody>
</table>
Figure 7: Distribution of the study population according to active management of the third stage of labour

68.65%

31.34%

active management  expectant management
Table 4: Correlation between the duration of the 1st stage of labour & aetiological causes of PPH.

<table>
<thead>
<tr>
<th>Duration of 1st stage of labour</th>
<th>Aetiology of PPH</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Atonia</td>
<td>GT injury</td>
</tr>
<tr>
<td>&gt; 12 hours</td>
<td>19 (79.17%)</td>
<td>3 (12.50%)</td>
</tr>
<tr>
<td>&lt; 12 hours</td>
<td>25 (58.14%)</td>
<td>13 (30.23%)</td>
</tr>
<tr>
<td>Total</td>
<td>44</td>
<td>16</td>
</tr>
</tbody>
</table>

P=0.199
Table 5: Correlation between augmentation of labour and aetiological factors of PPH

<table>
<thead>
<tr>
<th>Augmentation of labour</th>
<th>Aetiology of PPH</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Atonia</td>
<td>GT injury</td>
</tr>
<tr>
<td>Augmented</td>
<td>26 (66.67%)</td>
<td>9 (23.08%)</td>
</tr>
<tr>
<td>Not augmented</td>
<td>18 (64.29%)</td>
<td>7 (25.00%)</td>
</tr>
<tr>
<td>Total</td>
<td>44</td>
<td>16</td>
</tr>
</tbody>
</table>

P=0.979
Table 6: Distribution of the study population according the mode of delivery

<table>
<thead>
<tr>
<th>Mode of delivery</th>
<th>Frequency</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous VD</td>
<td>32</td>
<td>47.76%</td>
</tr>
<tr>
<td>Induced VD</td>
<td>4</td>
<td>5.97%</td>
</tr>
<tr>
<td>Forceps VD</td>
<td>5</td>
<td>7.46%</td>
</tr>
<tr>
<td>Ventose VD</td>
<td>3</td>
<td>4.48%</td>
</tr>
<tr>
<td>Cesarean section</td>
<td>23</td>
<td>34.33%</td>
</tr>
<tr>
<td>Total</td>
<td>67</td>
<td>100%</td>
</tr>
</tbody>
</table>
Figure 8: distribution of the study population according to the aetiological factors of PPH.
**Table 7: Distribution of the study population according to the type of genital tract injury**

<table>
<thead>
<tr>
<th>Type of GT injury</th>
<th>Frequency</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical tear / laceration</td>
<td>8</td>
<td>50%</td>
</tr>
<tr>
<td>Vaginal tear / laceration</td>
<td>2</td>
<td>12.5%</td>
</tr>
<tr>
<td>Extended / malsutured episiotomy</td>
<td>3</td>
<td>18.75%</td>
</tr>
<tr>
<td>Perineal tears</td>
<td>2</td>
<td>12.5%</td>
</tr>
<tr>
<td>Broad ligament haematoma</td>
<td>0</td>
<td>0.00%</td>
</tr>
<tr>
<td>Ruptured uterus</td>
<td>1</td>
<td>6.25%</td>
</tr>
<tr>
<td>Total</td>
<td>16</td>
<td>100%</td>
</tr>
</tbody>
</table>
Table 8: Distribution of the study population according to the fetal birth weight

\[ N=77 \]

<table>
<thead>
<tr>
<th>Fetal birth weight</th>
<th>Frequency</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 2.5 kg</td>
<td>16</td>
<td>20.78%</td>
</tr>
<tr>
<td>2.5 – 4 kg</td>
<td>57</td>
<td>74.03%</td>
</tr>
<tr>
<td>&gt; 4 kg</td>
<td>4</td>
<td>5.19%</td>
</tr>
<tr>
<td>Total</td>
<td>77</td>
<td>100%</td>
</tr>
</tbody>
</table>
Figure 9: Distribution of the study population according to the amount of blood transfused

- 1 to 2: 53.73%
- 3 to 4: 20.89%
- 5 to 6: 14.93%
- > 6: 10.45%

No. of blood units
**Table 9: Distribution of the study population according to the medical treatment modalities used**

<table>
<thead>
<tr>
<th>Medical treatment</th>
<th>Frequency</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syntocinon (IV, infusion)</td>
<td>64</td>
<td>95.52%</td>
</tr>
<tr>
<td>Ergometrin (IV, IM)</td>
<td>54</td>
<td>80.00%</td>
</tr>
<tr>
<td>Misoprisol (rectally)</td>
<td>5</td>
<td>7.46%</td>
</tr>
<tr>
<td>Carboprost</td>
<td>0</td>
<td>0.00%</td>
</tr>
<tr>
<td>Prostaglandin E2</td>
<td>0</td>
<td>0.00%</td>
</tr>
</tbody>
</table>
Table 10: Distribution of the surgical treatment modalities used among the study population

<table>
<thead>
<tr>
<th>Surgical treatment</th>
<th>Frequency</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repaired under GA</td>
<td>10</td>
<td>14.92%</td>
</tr>
<tr>
<td>caesarian hystrectomy</td>
<td>9</td>
<td>13.43%</td>
</tr>
<tr>
<td>Ligation of the internal iliac artery</td>
<td>1</td>
<td>1.49%</td>
</tr>
</tbody>
</table>
Table 11: Distribution of the study population according to 
Maternal morbidity & mortality

<table>
<thead>
<tr>
<th>Morbidity</th>
<th>Frequency</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute renal failure</td>
<td>2</td>
<td>2.99%</td>
</tr>
<tr>
<td>DIC</td>
<td>8</td>
<td>11.94%</td>
</tr>
<tr>
<td>Admission to ICU</td>
<td>2</td>
<td>2.99%</td>
</tr>
<tr>
<td>Maternal mortality</td>
<td>3</td>
<td>4.47%</td>
</tr>
</tbody>
</table>
Discussion

The incidence of primary PPH in developed countries is about 1.3 % of all deliveries (2), but there are limited studies in the developing countries including Sudan, which makes it difficult to determine the exact incidence.

This is a prospective descriptive hospital based study included all patients (67) who developed massive primary PPH at Omdurman maternity hospital during the study period, the incidence of PPH in this study was 1%, which is low in comparison to the international figure, this may be due to the fact that selection of patients required the need for blood transfusion was the only objective diagnostic criterion of PPH, so those patients who developed primary PPH and did not need blood transfusion were not included in the study.

Stones (25) and Combs (5) have used different definitions of PPH in their studies. Stones included all women with a recorded blood loss of > 1000 ml in the 24 hours after delivery as cases of PPH. Using this definition, 498 of 37.497 (1.3%) women studied suffered PPH. Combs included as cases all women with a fall in haematocrit of more than 10 points between admission and post delivery plus all women receiving blood transfusion. On this basis, 374 of 9598(3.9%) women suffered PPH. This explains the difference in incidence according to the definition used.

There was direct relation between primary PPH and increased maternal age, as 51.31% of the study group were in the age more than 30 years, this goes with the population based case control study in Zimbabwe (26) which concluded that advance maternal age was one among the strongest risk factors for PPH.

Most of the patients in the study group (77.61%) were from urban areas, this could be explained by the fact that, this hospital has been the main location of maternal care since 1957 and located in the center of Omdurman city, and referred cases were from rural areas.
No association between gestational age and risk of PPH was found in the literature. The only association is that the frequency of retained placenta is markedly increased (twenty-fold) at gestations < 26 weeks and even up to 37 weeks it remains three times more common than at term \(^{(21)}\). In this study the small number of patients (7) who developed retained placenta will not allow accurate correlation with the gestational age.

Obstetrics textbooks and reviews tend to perpetuate a list of numerous risk factors of PPH with no indication of the relative frequency or importance of the various factors, recently however, authors from the US \(^{(5)}\), UK \(^{(25)}\) and Zimbabwe \(^{(26)}\) have applied statistical methods to data from case-control series in order to confirm and quantify the level of risk associated with various factors, the risk factors quoted were all associated with odds (risk) ratios which reach statistical significance and have been rounded to the nearest whole number, which included placenta previa (13.1), placental abruption (12.6), multiple pregnancy (4.46), pre-eclampsia (4), previous PPH (3), and anemia.

In the study there is high frequency of, anemia (37.37%), pre-eclampsia and gestational hypertension (29.85%), antipartum haemorrhage including placenta previa and placental abruption (28.36%), multiple pregnancy (14.93%), previous PPH (14.93%), which goes with the strong association as in the literature \(^{(5)}\) \(^{(25)}\) \(^{(26)}\).

Concerning the relation between parity and development of PPH, Tsu-VD’s study confirmed the association between low parity and PPH, but no association with grand multiparity was found, so called into question the significance of grandmultiparity \(^{(26)}\). In this study there was almost equal distribution of the parity among the study group, (32.84%) nulliparous, (34.32%) multiparous, and (32.84%) grand multiparous. This might not reflect the association between parity and PPH but may be due to the different prevalence of nulliparous and grand multiparous among the background population.

The studies of Stones et al and Combs et al also provide data about factors becoming apparent during labour and delivery and associated with an increased risk of PPH, there is two-fold increased risk of PPH in prolonged labour (>12 hours) and five-fold increased risk in retained placenta. In this study, (35.82%) of patients had prolonged first
stage of labour, (14.93%) had prolonged second stage of labour, and (10.45%) had retained placenta.

The frequency of primary PPH is related to the management of the third stage of labour. Data from several sources, including several large randomized trials performed in industrialized countries; indicated that the prevalence of PPH of more than 500 ml is approximately 5% when active management is used versus 13% when expectant management is used. The prevalence of PPH of more than 1000 ml is approximately 1% when active management is used versus 3% when expectant management is used (Rogers, 1998; Prendiville, 2000). In this study, only (31.34%) of patients had active management of third stage of labour, while (68.65%) had expectant management of third stage of labour. This reflects the importance of active management of the third stage of labour in the prevention of PPH.

Cunningham FG et al (3) concluded that barely effective uterine contractions, induction or augmentation of labour are risk factors to PPH. In contrast to this the study did not find statistically significant relationship neither between prolonged labour nor augmentation of labour and PPH (P = 0.199), (P = 0.979) respectively. This may be due to the small sample size.

There is nine-fold increase in risk of PPH associated with caesarian section over vaginal delivery (8). The incidence of caesarian section in the study population was (34.33%); this figure is relatively high because the incidence of caesarian section in the study hospital is about one fifth of all deliveries.

Regarding the aetiological factors of primary PPH, uterine atony was the most common cause of PPH in the study group (65.67%), which is higher than the figure in the literature (50%) (19) Which can be explained by high percentage of patient who had expectant management of the third stage of labour? followed by genital tract injury (23.88%), this is in consistence with that found in the literature (20%) (21).

Most of the study population (53.73%) received 1-2 units of blood while (10.45%) needed massive blood transfusion, neither central venous pressure monitoring nor measurement of haematocrit as an important indicator for the amount needed for
transfusion was practiced in our hospitals and the amount of blood transfused is mainly
determined by the clinical parameters of pulse, blood pressure, and state of pallor as
indications of hypovolaemia.

The majority of the study population received oxytocin (95.52%) and ergometrin
(80.60%) as the medical treatment of choice. Because prostaglandins are expensive and
hardly available, only five patients (7.46%) received rectal misoprostol and no patient
received carboprost or prostaglandin E2.

The range of surgical treatment done in the management of some of the cases might
reflect the experience of the staff as 9 cases (13.43%) underwent caesarian hysterectomy
compared to only one patient managed by internal iliac artery ligation.
PPH still a significant contributor to maternal mortality in the developed and developing
countries (2.6-3.4% per million maternities), (40 per 100,000) respectively \(^{(2,17)}\).
In this study, 3 maternal deaths due to PPH occurred in a total delivery of 5649, this gives a
mortality rate of 53.1 per 100,000 deliveries. This figure is slightly more than that of the
developing countries \(^{(17)}\).
Conclusion

Obstetric haemorrhage is among the leading causes of maternal mortality worldwide.

The study concluded that maternal mortality form PPH is higher even in comparison to the rates in developing countries.

The study found that about half of the study group was in the age more than 30 years. There is high incidence of anemia, pre-eclampsia and gestational hypertension in the study population as well as increase in incidence of antepartum haemorrhage including placenta previa and placenta abruption, multiple pregnancy, and previous PPH among the study population.

In the study population active management of the third stage was done only in about one third of the patients.

The study did not find statistically significant relationship between prolonged or augmented labour and subsequent development of PPH.

The incidence of caesarian delivery is slightly higher than the incidence in the general population in the study hospital as the study carried out at central hospital to which most high risk pregnancies are referred.

The main aetiological factor of PPH in the study population was uterine atony constituting about two thirds of cases.

Oxytocin and ergometrin have been used almost exclusively as the medical treatment of choice for PPH. Prostaglandins used in a minority of cases.

Caesarian hysterectomy was done to considerable number of the study population to only one patient managed conservatively by internal iliac artery ligation.
**Recommendations**

- All hospitals should introduce low-cost, evidence-based practices and guidelines to prevent and manage PPH which can improve provider’s performance and quality of care.

- Patient at high risk of PPH should be cared for by skilled professionals on comprehensive emergency obstetric services at labour and the postpartum period.

- Careful use of both forceps and ventouse to minimize maternal injuries and well training of the staff to basic rules for instrumental delivery.

- Blood transfusion facilities and different types of plasma expanders should be available in all centers that provide comprehensive health care to the mothers.

- Obstetricians should be trained in simple conservative techniques of the treatment of PPH such as compression sutures and devascularization.
References


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6-Schuurmans Nan; Makinnon C; Lane C; Etches D. Prevention and Management of Postpartum Haemorrhage. SOGC clinical practice guideline. No. 8, April 2000.


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48- Okawa T; Takano Y; Takahashi H; Morimura Y; Yanagida K; Sato A. In: Arch – Gynaecol-Obstet. 2002 Jan; 266: 50 – 2.


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Massive Primary postpartum haemorrhage: Risk factors and maternal outcome

**Questionnaire**

1- Age: <20 ○ 20-29 ○ 30-39 ○ 40-49 ○

2- Residence: Rural ○ Urban ○

3- Education: Illiterate ○ Primary ○ Secondary ○ Graduated ○

4- Occupation:.................................

5- Husband Occupation:...........................

**Antenatal Factors**

6- Gestational age at delivery:............................Weeks

7- Parity: Nulliparous ○ 1-4 ○ >4 ○

8- Order of pregnancy: Single ○ Multipe ○

9- Past history of PPH: Yes ○ NO ○

10- Polyhydramnios: Yes ○ NO ○

11- Fibroid with pregnancy: Yes ○ NO ○

12- History of bleeding disorder: Yes ○ NO ○

13- History of APH: Yes ○ NO ○

14- History of Pre-eclampsia: Yes ○ NO ○

15- History of PROM: Yes ○ NO ○

16- Anaemia with pregnancy: Yes ○ NO ○

If yes Haemoglobin concentration..............................gm/dl
Intrapartum Factors

17- Mode of delivery: NVD  ○  Induced VD  ○  Forceps  ○  Ventose  ○  C/S  ○  C/S

18- Duration of the first stage of labour: Hours

19- Augmentation with oxytocin:  Yes  ○  NO  ○

20- Duration of the second stage of labour: Hours

21- Duration of the third stage of labour: Minutes

22- Active management of the third stage of labour:  Yes  ○  NO  ○

Aetiology of PPH

23- Genital tract injury: Yes  ○  NO  ○

If yes: Cervical tear/Laceration  ○  Vaginal tear/Laceration  ○

Extended episiotomy  ○  Perineal tear  ○  Broad ligament haematoma  ○

24- Uterine hypotonia:  Yes  ○  NO  ○

25- Retained placenta (partially or wholly):  Yes  ○  NO  ○

26- Morbidly adherent placenta:  Yes  ○  NO  ○

27- Malsutured episiotomy:  Yes  ○  NO  ○

Fetal Outcome

28- Birth outcome: Alive  ○  Fresh still birth  ○  Macerated still birth  ○
29- Birth WT:………………………………..Kg

**Maternal outcome & treatment modalities used**

<table>
<thead>
<tr>
<th>30- Need for blood transfusion:</th>
<th>No</th>
<th>1-2 units</th>
<th>3-4 units</th>
<th>5-6 units</th>
<th>&gt;6 units</th>
</tr>
</thead>
<tbody>
<tr>
<td>31-Medical treatment:</td>
<td>Syntocinon (IV or infusion)</td>
<td>☐</td>
<td>Ergometrin</td>
<td>☐</td>
<td>Carboprost</td>
</tr>
<tr>
<td>32-Surgical treatment:</td>
<td>Repair under general anesthesia</td>
<td>☐</td>
<td>Ligation of uterine arteries</td>
<td>☐</td>
<td>Ligation of internal iliac arteries</td>
</tr>
<tr>
<td>33- Acute renal failure:</td>
<td>Yes</td>
<td>☐</td>
<td>NO</td>
<td>☐</td>
<td></td>
</tr>
<tr>
<td>34- DIC:</td>
<td>Yes</td>
<td>☐</td>
<td>NO</td>
<td>☐</td>
<td></td>
</tr>
<tr>
<td>35- Admission to ICU:</td>
<td>Yes</td>
<td>☐</td>
<td>NO</td>
<td>☐</td>
<td></td>
</tr>
<tr>
<td>36- Maternal mortality:</td>
<td>Yes</td>
<td>☐</td>
<td>NO</td>
<td>☐</td>
<td></td>
</tr>
</tbody>
</table>