Managing Major Early (Primary) Postpartum Haemorrhage in Developing Countries.

Ayodele O. Arowojolu
Department of Obstetrics and Gynaecology, University College Hospital, Ibadan, Nigeria.

Abstract

Major primary postpartum haemorrhage continues to top the list of causes of maternal mortality worldwide. Delays in the correction of hypovolaemia, diagnosis and treatment of bleeding disorders and initiation of surgery are preventable factors responsible for majority of the deaths. The situation is worse in the developing countries where more deaths are reported due to poor facilities, lack of manpower and delay in seeking expert management. The clinical state of the patient rather than the amount of blood loss should guide the clinician to the urgency of resuscitation. The first priority is to rapidly correct hypovolaemia with crystalloids and red blood cells. It is probably safer to avoid the use of colloids as it has been found to be associated with an increased risk of maternal death. The use of blood components will depend on platelet count, coagulation tests, haematocrit and fibrinogen concentration. The various medical and surgical management modalities that are useful when there is a poor response to oxytocics are presented. Procedures that are possible in developing countries have been highlighted, with an emphasis on surgical intervention measures that are likely alternatives to hysterectomy. The role of intervention radiology in modern obstetrics is also discussed.

Key Words: Postpartum Haemorrhage, Labour, Maternal Death [Trop J Obstet Gynaecol, 2003, 20: 144-152]

Introduction

Worldwide, about half a million women die yearly of pregnancy-related causes [1]. Majority of maternal deaths occur in developing countries whose maternal death rates are about 1,000 times the rates in developed countries [2]. Shortage of medical facilities, blood transfusion services, manpower, anaesthesia, transportation and patients' willingness to seek help early when needed are major limitations in developing countries [3, 4, 5]. Massive postpartum haemorrhage (PPH) tops the list of causes of maternal deaths and continues to be a major cause of postpartum morbidity worldwide [5, 6, 7]. In Britain, the risk of maternal death from PPH is around 1 in 100,000 deliveries while in developing countries the risk is of the order of 1 in 1,000 deliveries [8, 9].

Definition of Post-Partum Haemorrhage

Primary postpartum haemorrhage has traditionally been defined as a loss of 500 ml or more of blood following vaginal delivery or 1,000 ml or more of blood following a caesarean section [6, 7]. Recently, major PPH requiring urgent attention has been defined as a loss of 1,000 ml or more postpartum [10]. This definition is based on the observation that a healthy pregnant woman at term can tolerate a blood loss of 1,000 ml without significant haemodynamic disturbance or a drop in haemoglobin [11]. However, absolute measurements present very little clinical relevance because blood losses recorded following childbirth are usually underestimates and unreliable for an accurate estimation of its effect on the cardiovascular status of the patient [12].

A well-conducted research to estimate blood loss at delivery has shown that relying upon clinical assessment alone leads to about 30 to 50 percent of PPH cases being missed [13]. Hence, the American College of Obstetricians and Gynaecologists (ACOG) defines PPH as a decrease in haematocrit of more than 10% from antepartum values [14]. This definition takes care of both vaginal and intraperitoneal blood losses that may not be evident at initial assessment. Unfortunately, information regarding pre-partum haematocrit is not always at hand during massive bleeding, and if available, it may not reflect the rapid haemodynamic changes taking place in the preceding 4 hours or more [15, 16].

In low-income communities, some pregnant women fall in labour already compromised by severe anaemia in pregnancy due to chronic nutritional deficiency or haemolysis. In such patients, a loss of less than 500 ml of blood may be devastating. Therefore, the overall clinical picture of the patient following a delivery will more reliably reflect her degree of cardiovascular compromise and the level of resuscitation she would need, rather than the subjective assessment of blood loss.

Correspondence: *Dr. Ayo Arowojolu, Department of Obstetrics & Gynaecology, University College Hospital, Ibadan, Nigeria.* **E-mail:** ayorowojolu@hotmail.com

Early (primary) PPH is that occurring within the first 24 hours following a delivery. It occurs in 1 to 2% of all deliveries and accounts for more than 75% of serious early postpartum complications [17].

Risk Factors Responsible For Major Primary Post-Partum Haemorrhage

Most episodes of PPH are due to atony of the uterus after the delivery of the infant [1, 7, 8, 18]. Other causes include retained placental fragments, lacerations of the lower genital tract and uterine rupture. Uterine inversion and blood coagulopathy are rare but can produce profound blood loss, shock and death [7 19]. In developing countries, poverty, ignorance, strong traditional beliefs and the fear of surgery, prompt some women to choose delivery at home, in religious houses or in other poorly-equipped facilities, under poor medical supervision with the attendant risks of precipitate labour, prolonged labour, intrauterine sepsis, uterine rupture, difficult instrumental deliveries and complicated third stage of labour, all of which can lead to PPH [20, 21].

Conditions like abruptio placentae, amniotic fluid embolism, pre-eclampsia, previous history of PPH, multiple pregnancies, retained dead fetus, grand-multiparity, uterine fibroids, aspirin ingestion, anaemia, polyhydramnios, maternal obesity, fetal macrosomia, episiotomy, injudicious use of tocolytics and some anaesthetic gases all predispose to various degrees of PPH [22, 23, 24]. Certain maternal medical diseases, such as acquired or inherited coagulation disorders and autoimmune thrombo-cytopenia increase the risk of PPH [1, 7].

Adetoro [18], in a retrospective study of 204 women with primary PPH in Ilorin, Nigeria, concluded that both primiparity and multiparity were significant risk factors for PPH and attributed the main cause to mismanagement of labour. On the contrary, at Ile Ife, also in Nigeria, Selo-Ojeme and Okonofua [21] in a case-control study of a group of 101 women with singleton pregnancies who developed PPH following normal vaginal delivery and a comparable group of 107 women who did not, reported a number of risk factors for primary PPH from univariate analysis. After adjustment by logistic regression, only prolonged second and third stages of labour, and non-use of oxytocics after delivery were significant risk factors for PPH. Grand multiparity, primigravidity and previous episodes of PPH were not significantly associated with the occurrence of PPH in the study. In another study of 498 cases of major PPH in the UK, Stones *et al* [10] reported that placental abruption, placenta praevia, multiple pregnancies, obesity, macrosomic babies, induced labour and episiotomy but not grand multiparity were significant risk factors for primary PPH. These two latter studies confirmed that in well-managed labour, grandmultiparity, of itself, is not a risk factor for primary PPH in both developed and developing countries contrary to previous assumptions.

Management of Major Primary PPH

Prophylaxis

Although PPH occurs unpredictably in low risk women, cognisance of the risk factors should alert the clinician and prepare him for the management. Prevention of PPH is achievable as part of the process of active management of labour, which shortens the third stage by using oxytocics, cord clamping and controlled cord traction. This can reduce blood loss by two- to three-folds in high risk patients [25, 26]. A combination of 5 to 10 iu oxytocin and 500μg of ergometrine (Syntometrine®), or 10 iu oxytocin alone, given intramuscularly, is quite effective in preventing atony of the uterus [27]. Ergometrine has a more drastic effect than oxytocin but it may cause nausea and vomiting in a small number of women [27]. Routine oxytocin infusion after the third stage of labour in order to prevent primary PPH has its advocates [7].

Allowing patients to push against an incompletely dilated cervix, misplacement of episiotomy on a varicose vein and poorly performed instrumental vaginal delivery can cause massive PPH from genital lacerations and should be avoided. Access to blood transfusion facilities is essential. Blood storage facilities should be available in the labour ward for emergencies. When prenatal diagnosis of abnormal placenta is made, prophylactic placement of arterial catheter for uterine artery embolisation before delivery is more wide spread in advanced countries than the developing ones [13].

Treatment

General Treatment

The corner stone of effective management of a major PPH is rapid diagnosis and early intervention. Delays in correction of an impending haemorrhagic shock, surgical intervention, diagnosis and treatment of coagulopathy are avoidable causes of maternal deaths. The primary step is to examine the abdomen and palpate the uterus, which can be massaged to contract if atonic. A stable intravenous line with a large bore intravenous cannular (size F14 or 16) is required for fluid replacement or blood transfusion. It is essential to call for immediate help from obstetric colleagues, anaesthetists, haematologists,

midwives, and maids. The placenta should be carefully inspected for missing parts.

When the uterus is well contracted and bleeding persists, examination under anaesthesia is necessary to detect and repair lower genital tract lacerations. The uterine cavity should be explored for retained placental parts or a succenturiate lobe, which should be removed before repairing any genital lacerations.

Persistent bleeding from needle points or abdominal incisions will suggest coagulopathy. Laboratory tests for clotting profile such as prothrombin time, platelet count, fibrinogen level, fibrin degradation products and partial thromboplastin time should be carried out. Where such tests are not feasible, bedside tests like clot retraction test or clot observation tests will help to detect coagulation failure [7].

Resuscitation of Patients

Patients with hypovolaemia require urgent restoration of the blood volume to prevent irreversible shock. A second large-bore intravenous cannula is mandatory for transfusion of fluids. The initial step is to rapidly infuse a crystalloid solution (e.g. 0.9% normal saline, Hartmann's solution or Ringer's lactate) intravenously, giving 3ml for every millilitre of blood lost. Since most patients will tolerate a loss of 1,000 ml of blood without a significant haemodynamic change, anybody with major PPH showing signs of hypovolaemia will require at least 3000mls of crystalloid rapidly [7]. Dextrose solution is inappropriate as only 10% of the volume infused is maintained in the intravascular space [1], and it may affect platelet function and the outcome of blood compatibility tests. The use of colloids for resuscitation has been questioned since a systematic review of controlled trials of fluid replacement for hypovolaemia showed a 4% increase in absolute risk of maternal death when colloids were used for resuscitation in critically ill patients instead of crystalloid solutions [9, 28]. Alderson et al [29] in a similar study, reported that albumin infusion in critically ill patient with hypovolaemia was associated with 6% additional deaths. Both reports favoured crystalloid solutions rather than colloid solutions for critically ill patients with major PPH.

Red blood cell transfusion is necessary when about 2-3 litres of blood or 40% of the patient's blood volume has been lost. The most suitable replacement for blood lost in major PPH is a sterile fully cross-matched fresh whole blood, which is not always available in emergency situations. Otherwise, by the time up to 3.5 litres of blood is lost, rapid infusion of warmed uncrossmatched O Rhesus negative or group specific blood should be started through a giving set that has 170-200 μm filter. An experienced anaesthetist should be present to guide the process of resuscitation and fluid replacement should be regulated according to the patient's central venous pressure. Oxygen should be given by facemask at the rate of 8 litres per minute and the urinary output should be monitored with an indwelling urethral catheter.

In case of coagulation defects, platelet and fresh frozen plasma should be transfused in accordance with blood test results, or empirically if bleeding persists [9]. Four units of fresh frozen plasma is given rapidly, followed by an additional 4 units for each 6 units of packed red blood cells infused, with aim of maintaining the platelet count above 50 x 10^9/litre and the prothrombin/activated partial thromboplastin time lower than 1.5 times the control value [1]. Cryoprecipitate is required when the fibrinogen concentration is below 1g/dl. The management of disseminated intravascular coagulation should be in consultation with a haematologist.

Medical Treatment

Uterine atony is usually managed with oxytocics, giving 20iu to 40iu oxytocin in a litre of Ringer's lactate or normal saline. Further increment in the dose may be necessary in order to achieve the desired effect. Oxytocin is cheap and readily available in developing countries but requires refrigeration to maintain its potency. If the infusion is ineffective, 0.25mg to 0.5mg ergometrine, 0.2mg methylergonovine (Methergine®) or 0.25mg 15-methyl-prostaglandin F2 alpha (PGF2 alpha or Prostin®) can be administered intramuscularly. Ergometrine should be used with caution if alternatives are available. Its side effects include vomiting, headache, raised blood pressure and convulsions, but it can be very useful in dire emergencies. It is however important to note that the available brands of ergometrine lose about 90% of their potencies in the tropics after one year of storage exposed to light [30].

$PGF_{2\alpha}$ can be given repeatedly by the intramuscular route every 15 to 60 minutes or injected directly into the myometrium transabdominally or transcervically to achieve the desired effect [31, 32]. Clinical trials with $PGF_{2\alpha}$ showed that about 75% of women with primary PPH due to uterine atony responded adequately to a single dose [33]. However, the drug is expensive, heat labile (needs to be refrigerated) and scarce in most rural communities. Its use is also associated with rare side effects such as desaturation of arterial oxygen, myocardial infarction, broncho-spasm, and severe hypertension [7, 34], making the

drug unsuitable in patients with pre-existing pulmonary and cardiovascular problems.

An alternative is the prostaglandin E_1 [PGE_1] analogue (Misoprostol®). Misoprostol® is cheap (costs about $1 for a 200μg tablet), heat stable (requires no refrigeration), has a long shelf life [35,36] and is highly effective when administered orally, vaginally or rectally [30,37]. The usual dose is 400 μg to 1,000 μg. Misoprostol is well absorbed vaginally and orally with a peak level seen within 1 hour [38]. When 1,000 μg misoprostol was given rectally, uterine contraction occurred with 3 minutes [39]. The drug does not cause myocardial infarction, bronchospasm or changes in blood pressure, making it safe for asthmatic, hypertensive and pre-eclamptic patients [40]. A systematic review of 16 randomised controlled trials of misoprostol to prevent PPH in 28,138 women concluded that misoprostol is less effective than injectable oxytocin or oxytocin-ergometrine preparations as part of active management of the third stage of labour [41]. On the other hand, a more recent study by Wright and Newton [42] showed that misoprostol is as effective as oxytocin but less effective than a combination of oxytocin and methylergometrine in the prevention of primary PPH. The adverse effects of misoprostol that have been reported include vomiting, diarrhoea, shivering and pyrexia [40,41,42]. Misoprostol is useful to clinicians in most parts of the developing world where oxytocics are scarce due to difficulties with storage and it should be readily available in all delivery units.

Other useful prostaglandings include sulphoprostone given by intravenous infusion in normal saline, gemeprost pessary that can be placed in the uterine cavity to effect myometrial contraction and intramuscular injection of 0.25mg carboprost, an analogue of PGF2 alpha [43,44].

Surgical Treatment

Persistent bleeding despite all medical measures requires surgical treatment but the decision to undertake surgery is sometimes emotionally delayed to the detriment of the patients. Such delays rather than the lack of surgical expertise, accounts for most deaths [45]. Every obstetric unit should have protocols for the management of obstetric haemorrhage as illustrated in a review by Mousa and Walkinshaw [1]. The choice of surgical procedure depends on the patient's clinical state, age, parity, the cause and the severity of the bleeding, available facilities and the surgeon's expertise.

Exploration of the Uterus

Uterine exploration is usually carried out under anaesthesia when medical management has failed or genital lacerations are suspected. Blood is made available in the theatre before commencing, and transfused, if required, during the procedure. The patient's bladder is emptied with a catheter that is left *in situ* to monitor urinary output post-operatively. A stepwise inspection of the perineum, vaginal wall and cervix is performed for lacerations and bleeding vessels, which should be ligated. The uterine cavity is then explored digitally, removing retained placental fragments and clots manually, and checking for any uterine rupture that will require transabdominal repair. Lacerations in the lower genital tract are repaired in layers, after the uterine exploration.

Uterine Tamponade

The recent re-emergence of uterine packing after a long period of disrepute follows the report of successful treatment of 7 out of 9 women with PPH by Maier [46]. The earlier concerns about sepsis, concealed haemorrhage and uterine over-distension have been allayed by recent modifications to the procedure. Although Maier [46] used a special Torpin packer to pack the uterine cavity with a length of gauze, he emphasised that the use of special instruments is not essential. Importantly, the uterine cavity should be uniformly packed and the patient placed on broad-spectrum antibiotics post-operatively. The urinary bladder is drained continuously until the pack is removed some 24 to 36 hours after the operation.

Alternatives to gauze packs are Foley's catheter and Sengstaken-Blakemore tube [47,48]. A size 24 Foley's catheter with a 30 ml balloon inflated with 60 to 80 ml of normal saline or 110 ml of air in the uterine cavity is all that is required [48,49]. Occasionally, the use of more than one catheter may be necessary to arrest the bleeding. The risk of blood accumulating unnoticed in the excess space above the bulb of the Foley's catheter calls for concern, so the patient should be closely monitored after the procedure for continuing signs of hypovolaemia.

The balloon of the Sengstaken-Blakemore tube (designed to control bleeding from oesophageal varices) is inserted into the uterine cavity with a pair of forceps and partly inflated to assess its effect on the blood loss (tamponade test) before it is inflated to its full capacity [50]. If considerable bleeding is observed, the test is considered unsuccessful and laparotomy is performed. Like Foley's catheter, the Sengstaken-Blakemore tube has an opening at its tip that allows continuous uterine drainage but it has the advantage of a larger balloon that can be inflated with up to 300 ml of water over Foley's catheter. However, the Sengstaken-Blakemore tube is not readily available in most labour wards in developing

countries, and it may not be easily obtainable in emergency situations. After successful placement of either the tube or a Foley's catheter, an oxytocin infusion is set up to maintain uterine retraction and the vagina is packed with gauze to prevent expulsion of the catheter or tube. The patient is covered with broad-spectrum antibiotics and her urinary output is monitored by continuous bladder drainage. The pack in the vagina, Foley's catheter, or the tube should all be removed at the bedside 24 hours after insertion, using intravenous analgesia.

Uterine tamponade with a pack or balloon stops severe PPH in 50% of cases identifiable with the tamponade test or uterine compression [50, 51]. When unsuccessful, the procedure would have allowed enough time to replace blood loss, stabilise the patient and prepare for surgery.

The Rusch urology catheter has been reported in literature as being useful in controlling massive primary PPH [52]. The catheter has the advantage of a larger balloon capacity (400-500mls) than the Foley's catheter and has been used in a patient with intractable PPH where the Sengstaken-Blakemore tube had failed [52].

Selective Arterial Embolisation

Selective arterial embolisation involves the passage of a catheter under fluoroscopy, through the femoral artery into the target artery by an interventional radiologist, using conscious sedation or regional anaesthesia [53]. For PPH, internal iliac, uterine or ovarian arteries are potential target vessels. Once in the target vessel, embolising agents, such as gelfoam pledgets, coils, sponges, polyvinyl alcohol particles or balloon catheter, can be used to occlude the target vessels and stop the bleeding [50, 54, 55]. The gelfoam is slowly absorbed and recanalisation of the vessels occurs [56]. The procedure can be done within 30 to 60 minutes by experienced personnel and the patient's response is usually immediate. The success rate is between 95 and 97% [53, 57]. Successes have been reported in patients with coagulopathy, bleeding from the cervix, ectopic pregnancy, post-abortion and broad ligament bleeding [1, 53]. Reports indicate that recovery is usually uneventful, menstruation returns quickly within 3 to 8 months and fertility is retained [54, 58, 59]. The procedure should be considered before laparotomy.

Women with placenta accreta, previa or abdominal pregnancy may have the catheter inserted before surgery in anticipation of need [57, 58]. Such a prophylactic approach is reportedly associated with less blood loss than emergency insertions [58]. Sepsis, pyrexia, buttock ischaemia, haematoma and vascular perforation are potential complications of the procedure, which also carries a small risk of irradiation to the fetus when insertion is done before delivery [60, 61].

Selective arterial embolisation is not yet readily available in developing countries due to lack of expertise in interventional radiology. The prospect is very promising because of the cost and avoidance of major surgery while preserving the patient's reproductive potential. An added advantage is that patients recover quickly from both the surgery and the haemorrhagic disorder.

Replacement of Uterine Inversion

Replacement of uterine inversion is preferably carried out immediately the inversion occurs [62]. Occasionally, the inversion is partial and can be replaced manually with little effort [14, 62]. On other occasions, it is complete and difficult to replace [19]. Uterine inversion is associated with massive bleeding, thus the patient should be resuscitated before embarking on surgery. Replacement of total inversion can be achieved by either infusing a large volume of normal saline into the vagina (saline replacement) or surgically at laparotomy using Haultain or Hunlington procedures [14]. It is advisable to leave the placenta attached to the uterus during replacement to avoid excessive haemorrhage. Anaesthesia helps to relax the patient but halothane has an added advantage of causing uterine relaxation. After successful replacement, oxytocin infusion should be given to maintain uterine contraction.

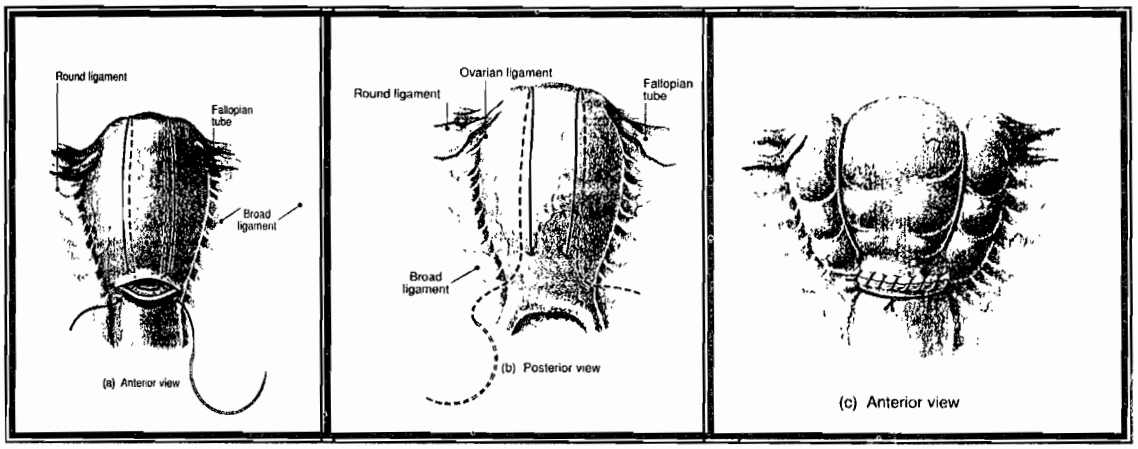
Surgical Compression Suture for Uterine Atony (B-Lynch Suture)

The B-Lynch suture was described in 1997 for surgical compression of the uterus in patients with intractable PPH [63]. It has since gained in popularity due to its simplicity and has brought reductions in hysterectomies, blood transfusions, maternal deaths and improvement in patients' satisfaction [63, 64, 65]. The B-Lynch suture is appropriate where bimanual uterine compression produces a decrease in vaginal bleeding before surgery. The procedure is simple and briefly described in Figure 1

A modified B-Lynch suture technique (Figure 2), which involves insertion of multiple uterine 'brace' haemostatic sutures, was recently used to control haemorrhage at caesarean section due to morbidly adherent placenta previa [50]. The advantages of the modified procedure are that it does not require hysterotomy and it applies pressure directly on the placental bed bleeding while reducing blood flow to the uterus. Using two separate sutures allows more tension to appose the uterine walls than can be achieved with a single suture. The horizontal lower

Figure 1

Illustration and Procedure for B-Lynch Brace Suture [63].

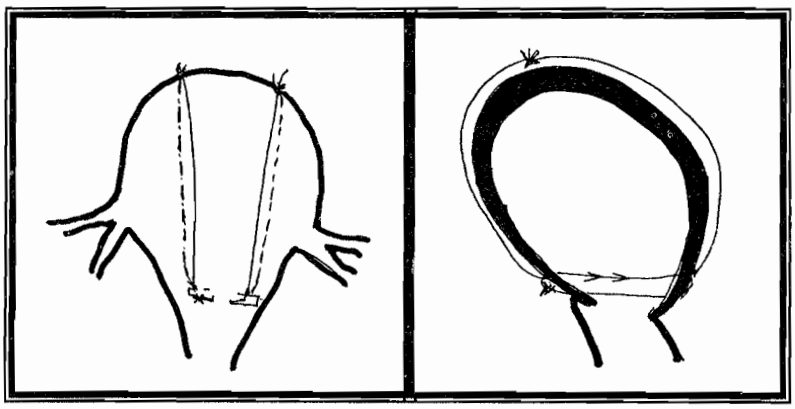


Procedure:

The patient is placed in the Lloyd-Davies position to enable vaginal assessment of blood loss. A Pfannenstiel or midline incision is made to open the abdomen, which is entered in layers. To test for potential efficacy of the procedure, the surgeon exteriorises the uterus and manually compresses it while the assistant checks the vagina for bleeding. If bleeding stops when the uterus is compressed, there is a good chance that application of the B-Lynch suture will be beneficial even in the presence of coagulopathy. A transverse hysterotomy is made in the lower uterine segment and the bladder is displaced inferiorly. The first stitch is inserted 3cm below the hysterotomy incision on the patient's left side and threaded through the entire anterior uterine wall into the cavity and brought out through the same wall 3cm above the upper incision margin, approximately 4cm from the lateral border of the uterus. The suture is carried outside the uterus over its anterior surface to the posterior surface, lying 4cm from the cornu, where it is inserted through the wall into the cavity at a point on a horizontal plane at the level of the uterine incision. It is then taken out through the posterior wall on the right side and brought anteriorly to enter the uterine cavity 4cm from the right lateral border of the uterus, 3cm above the hysterotomy incision, and then out of the cavity anteriorly 3cm below the incision opposite the first entry point. The assistant applies a constant pressure on the uterus, squeezing out blood clots and maintaining uterine contraction. The two ends of the suture are knotted anteriorly to compress the uterus using a double throw-knot.

Figure 2

Illustration and Procedure for the Uterine Haemostatic Suture of Tamizian and Arulkumaran [50]



Anterior View Saggital View

Procedure:

An isthmo-cervical stitch that controls bleeding from the lower uterine segment and cervix is inserted. A strong absorbable suture (No. 2 dexon or catgut) on a straight needle is used to puncture the uterine wall above the bladder reflection, 3cm below and 2cm medial to the edge of the lower uterine segment, and the needle is carried through the uterihe cavity and the posterior uterine wall. The needle is then carried through the posterior wall, uterine cavity and the anterior wall from a point about 1cm medial to its exit point posteriorly. The free ends of the suture are knotted anteriorly to complete the loop. The procedure is repeated on the opposite side, leaving free a central portion that connects the cervical canal for drainage. Any bleeding from the body of the uterus can be controlled with a modified brace suture applied to each side of the uterine body using a No. 2 chromic catgut suture. This is inserted just above the horizontal uterine brace sutures in the lower segment on each side of the uterus. The suture is carried through the anterior wall and uterine cavity to come out through the posterior wall. The free ends of the suture are then knotted over the fundus 3cm to 4cm medial to the cornu. The procedure is repeated on the opposite side while bimanual compression or oxytocics maintain uterine tone. The bladder should be emptied and checked for trauma.

segment suture prevents bleeding from ascending vessels from the vagina.

Another method that was recently described is the insertion of multiple square sutures from anterior uterine wall to the posterior wall through the cavity, to arrest PPH at caesarean section using No1 chromic catgut [66]. In a series of 23 women, 4 of whom later got pregnant, haemostasis was achieved and hysterectomy avoided [66]. Hysteroscopic and hysterosalpingographic examinations of the uterine cavity done later in 6 of the women who had the procedure, revealed no abnormality.

Ligation of Pelvic Arteries

Ligation of pelvic arteries reduces the pulse pressure in these vessels and allows coagulation to take place[7]. This is the rational for the use of arterial ligation in the treatment of PPH as an alternative to hysterectomy when medical treatment has failed. The choice of this method depends on the patient's parity, her desire for more children, severity of the bleeding and the surgeon's expertise. The procedure is very useful for bleeding from placenta praevia and lower uterine segment of an intact uterus [50]. The objective is to place a single absorbable suture around the uterine vessels just above the bladder flap, encompassing a portion of the myometrium. A second stitch is placed beneath the ovarian ligament adjacent to the uterus if required to arrest further bleeding from this source. The stitches should be inserted on both sides of the uterus to minimize blood loss from collateral circulation [50]. Deep bites into the uterine muscle provide a firm anchor and occlude the vessels. O'Leary quoted a success rate of 95% in a series of 265 cases [67]. Despite a reduction in arterial pulse pressure by 80% in the patients who had bilateral ligation of both uterine and ovarian vessels, recanalisation of the vessels with subsequent normal menstrual flow and fertility occurred.

If bleeding persists after inserting theses stitches, both internal iliac arteries should be ligated before hysterectomy is considered. Such situations occasionally occur in women with uterine rupture or broad ligament haematoma [50]. The artery is located beneath the peritoneum over the common iliac artery parallel and lateral to the ureter. A single non-absorbable suture is inserted around the anterior trunk of the internal iliac artery distal to the origin of the posterior division, 2cm to 3cm below its origin from the common iliac artery. The vessel is not divided and the procedure is repeated on the other side as a rule [7, 68]. Success rate is about 42% [50, 68]. A successful pregnancy and delivery by a woman who had both internal iliac arteries ligated has been reported in the literature [69]. Bilateral occlusion of the internal iliac arteries results in about 85% reduction in arterial pulse pressure, leaving a venous flow pattern, which is subject to the control of intrinsic coagulation and pressure mechanisms [7]. Technical difficulties aside, the procedure takes a longer time, and there is a risk of ureteric injury [50]. It should therefore be performed only when uterine preservation is very necessary and the patient is in a clinical state conducive to doing such a procedure.

Hysterectomy

Peripartum hysterectomy is performed in patients with persistent bleeding which has not responded to medical measures or conservative surgery. It is commonly performed in developing countries for ruptured uterus and placenta accreta[70, 71, 72]. Peripartum hysterectomy can be subtotal or total. Subtotal hysterectomy is quicker and somewhat safer than total hysterectomy, and can be performed by general practitioners or middle grade surgeons. It is recommended for severe bleeding from ruptured lower uterine segment, especially in multipara and those who have completed their families. The decision to perform hysterectomy is often delayed, resulting in maternal death. For women in shock who continue to bleed and in situations where blood replacement is not available, hysterectomy should be considered early in the management of the patient.

Conclusion

Major PPH continues to be an important cause of maternal deaths, with diverse diagnostic and management problems. In developing countries, these problems are compounded by several factors that include shortage of manpower, preponderance of substandard medical facilities and delay by the patients in seeking medical attention in emergency situations. Optimum management depends on the identification of women at risk, initiation of appropriate prophylactic measures and timely intervention in those apparently normal women who present suddenly with massive haemorrhage. Contemporary obstetric practice is replete with modalities of medical and surgical treatment that provide the clinician the opportunities to prevent hysterectomy but are yet to be fully deployed in the developing countries. Advantages of misoprostol and those of conservative surgery have remained almost totally untapped. Therefore, the need for continuous training of clinicians and to review the various management modalities cannot be over-emphasised in such countries.

References

1. Mousa HA, Walkinshaw S. Major postpartum haemorrhage. *Curr Opin Obstet Gynecol,* 2001; 13: 595-603.
2. Rosenfield A. Maternal mortality in developing countries. An ongoing but neglected 'epidemic'. *JAMA* 1989; 262: 376-379.
3. Okonofua FE, Abejide A, Makanjuola RO. Maternal mortality in Ile-Ife, Nigeria: a study of risk factors. *Stud Fam Plann,* 1992; 23: 319-324.
4. Mantel GD, Buchmann E, Rees H, Pattinson RC. Severe acute maternal morbidity: a pilot study of a definition for a near-miss. *Br J Obstet Gynaecol,* 1998; 105(9):985-90.
5. Etuk SJ, Asuquo EE. Effects of community and health facility interventions on postpartum hemorrhage. *Int J Gynecol Obstet,* 2000; 70: 381-383.
6. Jouppila P. Postpartum haemorrhage. *Curr Opin Obstet Gynecol,* 1995; 7: 446-450.
7. Roberts WE. Emergent obstetric management of postpartum hemorrhage. *Obstet Gynecol Clin North Am,* 1995; 22: 283-302.
8. Drife J. Management of primary postpartum haemorrhage. *Br J Obstet Gynaecol,* 1997; 104(3): 275-277.
9. Bonnar J. Massive obstetric haemorrhage. *Baillieres Best Pract Res Clin Obstet Gynaecol,* 2000; 14: 1-18.
10. Stones RW, Paterson CM, Saunders NJ. Risk factors for major obstetric haemorrhage. *Eur J Obstet Gynecol Reprod Biol* 1993; 48: 15-18.
11. Hofmeyr GJ, Mohlala BK. Hypovolaemic shock. *Best Pract Res Clin Obstet Gynaecol,* 2001; 15: 645-662.
12. Prasertcharoensuk W, Swadpanich U, Lumbiganon P. Accuracy of the blood loss estimation in the third stage of labor. *Int J Gynecol Obstet,* 2000; 71: 69-70.
13. Gilbert L, Porter W, Brown VA. Postpartum haemorrhage--a continuing problem. *Br J Obstet Gynaecol,* 1987; 94: 67-71.
14. American College of Obstetricians and Gynecologists. ACOG Educational Bulletin. Postpartum hemorrhage. Number 243, January 1998 (replaces No. 143, July 1990). *Int J Gynaecol Obstet,* 1998; 61: 79-86.
15. Robson SC, Boys RJ, Hunter S, Dunlop W. Maternal hemodynamics after normal delivery and delivery complicated by postpartum hemorrhage. *Obstet Gynecol* 1989; 74: 234-239.
16. Nicol B, Croughan-Minihane M, Kilpatrick SJ. Lack of value of routine postpartum hematocrit determination after vaginal delivery. *Obstet Gynecol,* 1997; 90: 514-518.
17. St George L, Crandon AJ. Immediate postpartum complications. *Aust NZ J Obstet Gynaecol,* 1990; 30: 52-56.
18. Adetoro OO. Primary post-partum haemorrhage at a university hospital in Nigeria. West Afr J Med 1992; 11(3):172-8.
19. Ogueh O, Ayida G. Acute uterine inversion: a new technique of hydrostatic replacement. *Br J Obstet Gynaecol* 1997; 104: 951-952.
20. Combs CA, Laros RK Jr. Prolonged third stage of labor: morbidity and risk factors. *Obstet Gynecol,* 1991; 77: 863-867.
21. Selo-Ojeme DO, Okonofua FE. Risk factors for primary postpartum haemorrhage: a case-control study. *Arch Gynecol Obstet,* 1997; 259: 179-187.
22. Fakeye O. The incidence, sociobiological factors and obstetric complications associated with large infants at Ilorin, Nigeria. *Int J Gynecol Obstet,* 1988; 27: 343-347.
23. Reubinoff BE, Eldor A, Laufer N, Sadovsky E. Maternal hemorrhagic complications following prophylactic low-dose aspirin and dipyridamole therapy. *Gynecol Obstet Invest,* 1992; 33: 241-243.
24. Tsu VD. Postpartum haemorrhage in Zimbabwe: a risk factor analysis. *Br J Obstet Gynaecol,* 1993; 100: 327-333.
25. Prendiville WJ, Harding JE, Elbourne DR, Stirrat GM. The Bristol third stage trial: active versus physiological management of third stage of labour. *BMJ* 1988; 297: 1295-1300.
26. Begley CM. A comparison of 'active' and 'physiological' management of the third stage of labour. *Midwifery* 1990; 6: 3-17.
27. Choy CM, Lau WC, Tam WH, Yuen PM. A randomised controlled trial of intramuscular syntometrine and intravenous oxytocin in the management of the third stage of labour. *BJOG* 2002; 109: 173-177.
28. Schierhout G, Roberts I. Fluid resuscitation with colloid or crystalloid solutions in critically ill patients: a systematic review of randomised trials. *BMJ* 1998; 316: 961-964.
29. Alderson P, Schierhout G, Roberts I, Bunn F. Colloids versus crystalloids for fluid resuscitation in critically ill patients. *Cochrane Database Syst Rev* 2000; (2): CD000567.
30. el-Refaey H, O'Brien P, Morafa W, Walder J, Rodeck C. Use of oral misoprostol in the prevention of postpartum haemorrhage. *Br J Obstet Gynaecol,* 1997; 104: 336-339.
31. Bigrigg A, Chissell S, Read MD. Use of intra myometrial 15-methyl prostaglandin F2 alpha to control atonic postpartum haemorrhage following vaginal delivery and failure of conventional therapy. *Br J Obstet Gynaecol* 1991; 98(7): 734-736.
32. Kupferminc MJ, Gull I, Bar-Am A *et al.* Intrauterine irrigation with prostaglandin F2-alpha for management of severe postpartum hemorrhage. *Acta Obstet Gynecol Scand* 1998; 77: 548-550.
33. Buttino L Jr, Garite TJ. The use of 15 methyl F2 alpha prostaglandin (Prostin 15M) for the control of postpartum hemorrhage. *Am J Perinatol,* 1986; 3: 241-243.
34. Hankins GD, Berryman GK, Scott RT Jr, Hood D. Maternal arterial desaturation with 15-methyl prostaglandin F2 alpha for uterine atony. *Obstet Gynecol* 1988; 72: 367-370.
35. Kararli TT, Catalano T, Needham TE, Finnegan PM. Mechanism of misoprostol stabilization in hydroxypropyl methylcellulose. *Adv Exp Med Biol,* 1991; 302: 275-289.
36. Toledo-Velasquez D, Gaud HT, Connors KA. Misoprostol dehydration kinetics in aqueous solution in the presence of hydroxypropyl methylcellulose. *J Pharm Sci* 1992; 81: 145-148.
37. Bamigboye AA, Hofmeyr GJ, Merrell DA. Rectal misoprostol in the prevention of postpartum hemorrhage: a placebo-controlled trial. *Am J Obstet Gynecol* 1998; 179: 1043-1046.
38. Zieman M, Fong SK, Benowitz NL, Banskter D, Darney PD. Absorption kinetics of misoprostol with oral or vaginal administration. *Obstet Gynecol,* 1997; 90: 88-92.

39. Kundodyiwa TW, Majoko F, Rusakaniko S. Misoprostol versus oxytocin in the third stage of labor. *Int J Gynecol Obstet,* 2001; 75: 235-241.
40. el-Refaey H, Templeton A. Early abortion induction by a combination of mifepristone and oral misoprostol: a comparison between two dose regimens of misoprostol and their effect on blood pressure. *Br J Obstet Gynaecol,* 1994; 101: 792-796.
41. Villar J, Gulmezoglu AM, Hofmeyr GJ, Forna F. Systematic review of randomized controlled trials of misoprostol to prevent postpartum hemorrhage. *Obstet Gynecol,* 2002; 100: 1301-1312.
42. Wright JM, Newton W. Is rectal misoprostol as effective as oxytocin in preventing postpartum hemorrhage? *J Fam Pract,* 2003; 52: 281-282.
43. Poeschmann RP, Doesburg WH, Eskes TK. A randomised comparison of oxytocin, sulprostone and placebo in the management of the third stage of labour. *Br J Obstet Gynaecol* 1991; 98: 528-530.
44. Woodhouse DR. The use of gemeprost pessaries to arrest postpartum haemorrhage. *Aust NZ J Obstet Gynaecol,* 1998; 38: 239.
45. Orji EO, Ogunlola IO, Onwudiegwu U. Brought-in maternal deaths in south-west Nigeria. *J Obstet Gynaecol* 2002; 22: 385-388.
46. Maier RC. Control of postpartum hemorrhage with uterine packing. *Am J Obstet Gynecol* 1993; 169: 317-321; discussion 321-323.
47. Katesmark M, Brown R, Raju KS. Successful use of a Sengstaken-Blakemore tube to control massive postpartum haemorrhage. *Br J Obstet Gynaecol,* 1994; 101: 259-260.
48. Marcovici I, Scoccia B. Postpartum hemorrhage and intra-uterine balloon tamponade: a report of three cases. *J Reprod Med* 1999; 44: 122-6.
49. Bowen LW, Beeson JH. Use of a large Foley catheter balloon to control postpartum hemorrhage resulting from a low placental implantation: a report of two cases. *J Reprod Med* 1985; 30: 623-625.
50. Tamizian O, Arulkumaran S. The surgical management of postpartum haemorrhage. *Curr Opin Obstet Gynecol,* 2001; 13: 127-131.
51. Chan C, Razvi K, Tham KF, Arulkumaran S. The use of a Sengstaken-Blakemore tube to control post-partum hemorrhage. *Int J Gynecol Obstet,* 1997; 58(2): 251-252.
52. Johanson R, Kumar M, Obhrai M, Young P. Management of massive postpartum haemorrhage: use of a hydrostatic balloon catheter to avoid laparotomy. BJOG 2001; 108(4):420-2.
53. Badawy SZ, Etman A, Singh M, Murphy K, Mayelli T, Philadelphia M. Uterine artery embolization: the role in Obstetrics and Gynecology. *Clin Imaging,* 2001; 25: 288-295.
54. Corr P. Arterial embolization for haemorrhage in the obstetric patient. *Best Pract Res Clin Obstet Gynaecol* 2001; 15: 557-561.
55. Oei SG, Kho SN, ten Broeke ED, Brolmann HA. Arterial balloon occlusion of the hypogastric arteries: a life-saving procedure for severe obstetric hemorrhage. *Am J Obstet Gynecol,* 2001; 185: 1255-1256.
56. Yamashita Y, Harada M, Yamamoto H *et al.* Transcatheter arterial embolization of obstetric and gynaecological bleeding: efficacy and clinical outcome. *Br J Radiol,* 1994; 67: 530-534.
57. Hansch E, Chitkara U, McAlpine J, El-Sayed Y, Dake MD, Razavi MK. Pelvic arterial embolization for control of obstetric hemorrhage: a five-year experience. *Am J Obstet Gynecol* 1999; 180: 1454-1460.
58. Mitty HA, Sterling KM, Alvarez M, Gendler R. Obstetric hemorrhage: prophylactic and emergency arterial catheterization and embolotherapy. Radiology 1993; 188(1):183-7.
59. Goldberg J, Pereira L, Berghella V. Pregnancy after uterine artery embolization. *Obstet Gynecol* 2002; 100: 869-872.
60. Ledee N, Ville Y, Musset D, Mercier F, Frydman R, Fernandez H. Management in intractable obstetric haemorrhage: an audit study on 61 cases. *Eur J Obstet Gynecol Reprod Biol* 2001; 94: 189-196.
61. Pirard C, Squifflet J, Gilles A, Donnez J. Uterine necrosis and sepsis after vascular embolization and surgical ligation in a patient with postpartum hemorrhage. *Fertil Steril,* 2002; 78: 412-413.
62. Bassaw B, Roopnarinesingh S. Postpartum shock and uterine inversion. *West Indian Med J* 1990; 39: 178-179.
63. B-Lynch C, Coker A, Lawal AH, Abu J, Cowen MJ. The B-Lynch surgical technique for the control of massive postpartum haemorrhage: an alternative to hysterectomy? Five cases reported. *Br J Obstet Gynaecol* 1997; 104: 372-375.
64. Ferguson JE, Bourgeois FJ, Underwood PB. B-Lynch suture for postpartum hemorrhage. *Obstet Gynecol,* 2000; 95: 1020-1022.
65. Dacus JV, Busowski MT, Busowski JD, Smithson S, Masters K, Sibai BM. Surgical treatment of uterine atony employing the B-Lynch technique. *J Matern Fetal Med* 2000; 9: 194-196.
66. Cho JH, Jun HS, Lee CN. Hemostatic suturing technique for uterine bleeding during cesarean delivery. *Obstet Gynecol,* 2000; 96: 129-131.
67. O'Leary JA. Uterine artery ligation in the control of post-caesarean haemorrhage. *J Reprod Med* 1995; 40: 189-193.
68. Clark SL, Phelan JP, Yeh SY, Bruce SR, Paul RH. Hypogastric artery ligation for obstetric hemorrhage. *Obstet Gynecol,* 1985; 66: 353-356.
69. Casele HL, Laifer SA. Successful pregnancy after bilateral hypogastric artery ligation. A case report. *J Reprod Med* 1997; 42: 306-308.
70. Wong WC, Kun KY, Tai CM. Emergency obstetric hysterectomies for postpartum haemorrhage. *J Obstet Gynaecol Res,* 1999; 25: 425-430.
71. Yamamoto H, Sagae S, Nishikawa S, Kudo R. Emergency postpartum hysterectomy in obstetric practice. *J Obstet Gynaecol Res,* 2000; 26: 341-345.
72. Bakshi S, Meyer BA. Indications for and outcomes of emergency peripartum hysterectomy. A five-year review. *J Reprod Med* 2000; 45: 733-737.