Post-partum haemorrhage (PPH) and active management of third stage of labour (AMTSL): Review

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Introduction

Postpartum hemorrhage (PPH) is commonly defined as blood loss exceeding 500 milliliters (mL) following vaginal birth and 1000 mL following cesarean.(1) Proposed alternate metrics for defining and diagnosing PPH include change in hematocrit, need for transfusion, rapidity of blood loss, and changes in vital signs, all of which are complicated by the urgent nature of the condition.(1) PPH is often classified as primary/immediate/early, occurring within 24 hours of birth, or secondary/delayed/late, occurring more than 24 hours post-birth to up to 12 weeks postpartum. In addition, PPH may be described as third or fourth stage depending on whether it occurs before or after delivery of the placenta, respectively.

PPH is one of the major causes of maternal mortality around the world with a reported incidence of 2–11%.(2) The exact rates may differ according to data source, country as well as assessment method with prevalence of 10.6% when measured by objective assessment of blood loss and 7.2% when measured by subjective techniques. According to a systematic review the prevalence of PPH with ≥500 ml of blood loss was 10.5% in Africa, 8.9% in Latin America and Caribbean, 6.3% in North America and Europe, and 2.6% in Asia.(2)

Various studies in different regions of India report different prevalence of PPH. A study in North east region of India reported 94 maternal deaths/102525 live births, out of these, 53.19% women died due to hemorrhage accounting for about 21.27% of total deaths.(3) Similar studies reported prevalence of 22.7% in Delhi and 28.57% in Orissa.(3) Hemorrhage was also found to be the major cause of maternal mortality in West India, accounting for 24.6% of maternal deaths in that region. In a study it was reported that 58% of maternal deaths due to hemorrhage were actually due to PPH, resulting from lack of provision of emergency transport at community level.(3)

The incidence of PPH has recently increased in most developed countries such as Canada, Australia, and the US and has been notably related to an increased use of oxytocin for labor augmentation and subsequent uterine atony.(4) PPH is a significant contributor to severe maternal morbidity and long-term disability as well as to a number of other severe maternal conditions generally associated with more substantial blood loss, including shock and organ dysfunction.

Causes of postpartum haemorrhage

PPH may result from failure of the uterus to contract adequately (atony), genital tract trauma (i.e. vaginal or cervical lacerations), uterine rupture, retained placental tissue, or maternal bleeding disorders. Uterine atony is the most common cause and consequently the leading cause of maternal mortality worldwide.

A number of case–control studies have identified antenatal and intrapartum risk factors for PPH although most cases of PPH have no identifiable risk factors. These risk factors have been summarised in a 2010 review.(5)

The four Ts	Risk factors/notes
Tone: abnormalities of uterine contraction	
Over distension of uterus	Polyhydramnios, multiple gestation, macrosomia
Intra-amniotic infection	Fever, prolonged rupture of membranes
Functional/anatomic distortion of uterus	Rapid labour, prolonged labour, fibroids, placenta praevia, uterine anomalies
Uterine relaxants, e.g. magnesium and nifedipine	Terbutaline, halogenated anaesthetics, glyceryl trinitrate
Bladder distension	May prevent uterine contraction
Tissue: retained products of conception	
Retained cotyledon or succenturiate lobe	
Retained blood clots	
Trauma: genital tract injury	
Lacerations of the cervix, vagina or perineum	Precipitous delivery, operative delivery
Extensions, lacerations at caesarean section	Malposition, deep engagement
Uterine rupture	Previous uterine surgery
Uterine inversion	High parity with excessive cord traction
Thrombin: abnormalities of coagulation	
Pre-existing states	
Haemophilia A	History of hereditary coagulopathies or liver disease

Idiopathic thrombocytopenic purpura	Bruising
von Willebrand's disease	
History of previous PPH	
Acquired in pregnancy	
Gestational thrombocytopenic	Bruising
Pre-eclampsia with thrombocytopenia e.g. HELLP	Elevated blood pressure
Disseminated intravascular coagulation	
a. Gestational hypertensive disorder of pregnancy with adverse conditions	Coagulopathy
b. in utero fetal demise	Fetal demise
c. severe infection	Fever, neutrophilia/neutropenia
d. abruption	Antepartum haemorrhage
e. amniotic fluid embolus	Sudden collapse
Therapeutic anticoagulation	History of thromboembolic disease
Abbreviations: HELLP haemolysis, elevated liver enzymes and low platelet count; PPH postpartum haemorrhage	

Interventions to prevent postpartum haemorrhage (WHO Guidelines)

In March 2012, the World Health Organization (WHO) held a technical consultation on the prevention and treatment of postpartum haemorrhage to review current evidence and to update previously published PPH guidelines.[6] The new guidelines combine previous documents to address both prevention and treatment—recognizing the importance of integrated care.

The new guidelines replace the 2009 WHO Guidelines for the Management of Postpartum Haemorrhage and Retained Placenta. Updates based on recent evidence include:

- Uterine massage is recommended for the treatment of PPH. Initiate uterine massage as soon as excessive bleeding/uterine atony is identified.
- Intravenous oxytocin alone is still the recommended uterotonic drug for the treatment of PPH. IV oxytocin is the first line drug of choice over other drugs (ergometrine and prostaglandins), including for women that have already received it for PPH prevention.
- If intravenous oxytocin is unavailable or if the bleeding does not respond to oxytocin, intravenous ergometrine, oxytocin-ergometrine fixed dose or a prostaglandin drug (including sublingual misoprostol, 800 mcg) should be given. This is an updated recommendation that considers all 3 second-line options, including prostaglandins.
- If PPH persists
  1. The use of intrauterine balloon tamponade is recommended for the treatment of PPH due to uterine atony. This recommendation is now stronger than the previous guidelines. It can be used for women who do not respond to uterotonics or if uterotonics are not available. This procedure can potentially avoid surgery and is appropriate while awaiting transfer to a higher-level facility.
  2. The use of uterine artery embolization is recommended as a treatment for PPH due to uterine atony, if other measures have failed. This recommendation is now stronger than the previous guidelines.
  3. If bleeding does not stop in spite of treatment (using uterotonics and other available interventions), the use of surgical interventions is recommended.
- For women experiencing PPH and awaiting transfer, the following are recommended as temporizing measures until appropriate care is available:
  1. Use of bimanual uterine compression for the treatment of PPH due to uterine atony after vaginal birth. This recommendation is now stronger than the previous guidelines.
  2. Use of external aortic compression for the treatment of PPH due to uterine atony after vaginal birth.
  3. Use of non-pneumatic anti-shock garments (NASGs). This is a new recommendation. Research is under way to evaluate the potential benefits and harms of NASGs for PPH treatment.
- For a retained placenta, IV/IM oxytocin (10 IU) in combination with CCT is still recommended. Ergometrine is still not recommended. While the 2009 guidelines included intraumbilical vein injection of oxytocin as a treatment for retained placenta, the updated version concludes that there is insufficient evidence to recommend its use.

What is new and Different about AMTSL in these recommendations?

AMTSL as a prophylactic intervention is composed of a package of three components or steps:

1. administration of a uterotonic, preferably oxytocin, immediately after birth of the baby;
2. controlled cord traction (CCT) to deliver the placenta; and
3. massage of the uterine fundus after the placenta is delivered.

In 2012, the results of a large WHO-directed, multi-centred clinical trial[7] were published and showed that the most important AMTSL component was the administration of a uterotonic.

The WHO trial also demonstrated that the addition of CCT did almost nothing to reduce haemorrhage. The women who received CCT bled 10 mL less (on average) than women who delivered their placenta by their own effort. There was a real difference, however, in terms of the length of the third stage: third stage was an average of six minutes longer among those women who did not receive CCT.

Considering data from this trial and the existing evidence concerning the role of routine uterine massage in the prevention of PPH, the WHO issued new recommendations clarifying that although administration of a uterotonic remains central to the implementation of AMTSL, the performance of CCT and immediate fundal massage are optional components.

PPH Management (RCOG Protocol)[8]

Resuscitation

Measures for MINOR PPH (blood loss 500–1000 ml, no clinical shock):

- Intravenous access (one 14-gauge cannula)
- Urgent venepuncture (20 ml) for:
- group and screen
- full blood count
- coagulation screen, including fibrinogen
- Pulse, respiratory rate and blood pressure recording every 15 minutes
- Commence warmed crystalloid infusion.

Measures for major PPH (blood loss more than 1000 ml and continuing to bleed OR clinical shock):

- A and B – assess airway and breathing
- C – evaluate circulation
- Position the patient flat
- Keep the woman warm using appropriate available measures
- Transfuse blood as soon as possible, if clinically required
- Until blood is available, infuse up to 3.5 l of warmed clear fluids, initially 2 l of warmed isotonic crystalloid. Further fluid resuscitation can continue with additional isotonic crystalloid or colloid (succinylated gelatin). Hydroxyethyl starch should not be used.
- The best equipment available should be used to achieve rapid warmed infusion of fluids
- Special blood filters should not be used, as they slow infusions.

A high concentration of oxygen (10–15 l/min) via a facemask should be administered, regardless of maternal oxygen concentration. If the airway is compromised owing to impaired conscious level, anaesthetic assistance should be sought urgently. Usually, level of consciousness and airway control improve rapidly once the circulating volume is restored.

Establish two, 14-gauge intravenous lines; a 20 ml blood sample should be taken and sent for diagnostic tests, including full blood count, coagulation screen, urea and electrolytes, and to cross-match packed red cells (4 units). The urgency and measures undertaken to resuscitate and arrest haemorrhage need to be tailored to the degree of shock (Table 1).

Table 1: Fluid therapy and blood product transfusion

Crystalloid	Up to 2 l isotonic crystalloid
Colloid	Up to 1.5 l colloid until blood arrives.
Blood	If immediate transfusion is indicated, give emergency group O, rhesus D (RhD)-negative, K-negative red cell units. Switch to group-specific red cells as soon as feasible.
Fresh frozen plasma (FFP)	Administration of FFP should be guided by haemostatic testing and whether haemorrhage is continuing: • If prothrombin time (PT) or activated partial thromboplastin time (APTT) are prolonged and haemorrhage is ongoing, administer 12–15 ml/kg of FFP. • If haemorrhage continues after 4 units of red blood cells (RBCs) and haemostatic tests are unavailable, administer 4 units of FFP.
Platelet concentrates	Administer 1 pool of platelets if haemorrhage is ongoing and platelet count less than 75×10^9/l.
Cryoprecipitate	Administer 2 pools of cryoprecipitate if haemorrhage is ongoing and fibrinogen less than 2 g/l.

If pharmacological measures fail to control the haemorrhage, initiate surgical haemostasis sooner rather than later.

Conservative surgical interventions may be attempted, depending on clinical circumstances and available expertise:

- Balloon tamponade
- Haemostatic brace suturing (such as using procedures described by B-lynch or modified compression sutures)
- Bilateral ligation of uterine arteries
- Bilateral ligation of internal iliac (hypogastric) arteries
- Selective arterial embolisation.

Resort to hysterectomy Sooner rather than later (especially in cases of placenta accreta or uterine rupture).

Haemostatic Suturing

Several case series have been published describing success with haemostatic brace sutures. The best known version, described by B-Lynch in 1997, requires hysterotomy for its insertion and is particularly suitable when the uterus has already been opened for a caesarean section. In 2002, Hayman et al. described a modified compression suture which does not require hysterotomy, and success in 10/11 women managed with this suture has been reported.[9] Other authors have described variants on these techniques. Double vertical compression sutures have proved effective in treating PPH due to atony and placenta praevia. This may have a dual action of reducing uterine blood flow and compressing the bleeding surface.

Stepwise uterine devascularisation and internal iliac artery ligation

Stepwise uterine devascularisation describes the successive ligation of (i) one uterine artery, (ii) both uterine arteries, (iii) low uterine arteries, (iv) one ovarian artery and (v) both ovarian arteries, in the management of PPH.[10] The original case series of 103 patients with intractable PPH not responding to medical management was effective in all cases without the need for hysterectomy, leading some clinicians to propose that stepwise uterine devascularisation should be the first-line conservative surgical treatment to control PPH.

A systematic review[11] of fertility outcomes following the surgical management of PPH concluded that uterine devascularisation techniques, including internal iliac artery ligation, did not adversely affect future fertility, although, the number of studies and quality of evidence was limited.

Selective arterial occlusion or embolisation by interventional radiology

A large retrospective study[11] has evaluated arterial embolisation in 251 patients after PPH. It was successful in arresting the bleeding in 86.5%. The analysis suggested that caesarean section delivery, disseminated intravascular coagulation and transfusion of more than 10 units of packed red cells were related to failed embolisation.

The logistics of performing arterial occlusion or embolisation where the equipment or an interventional radiologist may not be available mean that uterine balloon tamponade is a more appropriate first-line treatment.

Hysterectomy

The decision for hysterectomy should be made by an experienced consultant clinician and the decision preferably discussed with a second experienced clinician when feasible. Early recourse to hysterectomy is recommended, especially where bleeding is associated with placenta accreta or uterine rupture.[12] Hysterectomy should not be delayed until the woman is in extremis or while less definitive procedures with which the surgeon has little experience are attempted. The procedure should be carried out by a surgeon who is experienced in carrying out hysterectomies. Subtotal hysterectomy is the operation of choice in many instances of PPH requiring hysterectomy, unless there is trauma to the cervix or a morbidly adherent placenta in the lower segment.

Conclusion

PPH is a common complication of childbirth and a leading cause of maternal morbidity and mortality. Clinicians should identify risk factors before and during labor so that care may be optimized for high-risk women. However, significant life-threatening bleeding can occur in the absence of risk factors and without warning. All caregivers and facilities involved in maternity care must have a clear plan for the prevention and management of PPH. This includes sound resuscitation skills and familiarity with all medical and surgical therapies available.

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