MANAGEMENT OF OBSTETRIC HEMORRHAGE

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Abstract

Major obstetric hemorrhage is an extremely challenging obstetric emergency associated with significant morbidity and mortality. Pharmacological treatment of uterine atony has not altered much in recent years apart from the increasing use of misoprostol, although controversy surrounds its advantages over other uterotonics. Placenta accreta is becoming more common, a sequel to the rising caesarean section rate. Interventional radiology may reduce blood loss in these cases. Uterine compression sutures, intrauterine tamponade balloons and cell salvage have been introduced in the last decade.

Keywords: Antepartum hemorrhage, postpartum hemorrhage, uterotonics.

Obstetric hemorrhage is the world's leading cause of maternal mortality¹. Postpartum hemorrhage (PPH) accounts for the majority of these deaths¹. The global maternal ratio of 402 deaths per 100,000 live births² obscures the fact that 99% of these deaths occur in the developing world³. In addition, even in many developed countries, it is also the maternal complication for which the highest rate of substandard care is observed⁴. Furthermore, Zeeman's⁵ study of obstetric critical care provision identifies hemorrhage as one of the most frequent reasons for admission to intensive care unit. Major obstetric hemorrhage accounts for 30% of cases⁶. Obstetric hemorrhage is often sudden, unexpected, and may be associated with coagulopathy. Blood loss can be notoriously difficult to assess in obstetric bleeds. Bleeding may sometimes be concealed and the presence of amniotic fluid makes accurate estimation challenging. Hence, early recognition and treatment are essential to ensure the best outcome.

Therefore, it is important to have a thorough knowledge of the pathophysiology, etiology, and management strategies of obstetric hemorrhage.

The initial assessment of a patient with an obstetric hemorrhage depends on its causes, but in general

- (1) Take a detailed medical and obstetric history and examine the patient to find the site and cause of the bleeding;
- (2) Empty the patient's bladder;
- (3) Ensure that there are no retained products of conception or genital tract lacerations (anesthesia may be necessary);
- (4) Estimate blood loss; and
- (5) Assess the patient's hemodynamic status and initiate appropriate resuscitation.

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Massive obstetric hemorrhage is variably defined as: blood loss more than 1500 ml, a decrease in hemoglobin more than 4 g/dl; or acute transfusion of more than 4 units; or patient receiving treatment for coagulopathy⁷.

The gravid uterus receives up to 12% of the cardiac output, thus obstetric hemorrhage can be rapidly become life threatening.

Mechanism of Hemostasis⁸

After disruption of vascular integrity, mechanisms of hemostasis include (a) platelet aggregation and plug formation, (b) local vasoconstriction, (c) clot polymerization, and (d) fibrous tissue fortification of the clot. Contraction of the uterus represents the primary mechanism for controlling blood loss at parturition. Endogenous oxytocics effect myometrial contraction after delivery.

Underestimation of peripartum hemorrhage is a frequent problem. Visual assessments typically underestimate the true amount of blood loss. Moreover, inadequate intravenous fluid administration is common. Hence, the primary problems were delayed recognition of hypovolemia and inadequate volume resuscitation.

Finding	% Blood loss
None	<15% to 20%
Tachycardia	
Mild hypotension	20% to 25%
Peripheral vasoconstriction	
Tachycardia (100 to 120 bpm)	
Hypotension (SBP 80 to 100 mmHg)	25% to 35%
Restlessness	
Oliguria	
Tachycardia (>120 bpm)	
Hypotension (SBP <60 mmHg)	>35%
Altered consciousness	
Anuria	

Table 1 Staging scheme for assessment of obstetric hemorrhage

bpm = beats per minute; SBP = Systolic blood pressure

Classification of Obstetric Hemorrhage

Antepartum Hemorrhage (APH)

This is bleeding after 24 weeks gestation and before delivery. The causes include:

- Placenta previa
- Placental abruption
- Trauma
- Uterine rupture

Primary Postpartum Hemorrhage

This is defined as blood loss within 24 hours of delivery, which is > 500 ml following a vaginal delivery and > 1000 ml following a caesarean delivery. The causes include:

- Uterine atony
- Retained product of conception
- Genital tract trauma
- Clotting defects
- Inverted uterus

Secondary Postpartum Hemorrhage

This is blood loss more than 24 hours after delivery. The causes include:

- Pre-eclampsia/HELLP syndrome
- Intrauterine sepsis
- Pre-existing coagulopathy
- Incompatible blood transfusion
- Retained dead fetus

ANTEPARTUM HEMORRHAGE

Antepartum hemorrhage is a relatively frequent problem, occurring in 5% to 6% of pregnant women⁸. Recent evidence suggests that antepartum hemorrhage of unknown origin does produce more premature labor and delivery and subsequently, more fetal and neonatal problems⁹. Cases with abnormal placentation, usually placenta previa or placental abruption, can result in serious complications for both mother and child. Antepartum hemorrhage can also result in postpartum hemorrhage. The antepartum bleeding secondary to placenta previa or abruption is responsible for perinatal mortality rates as high as 22% and 37%, respectively¹⁰.

Placenta previa

Placenta previa is abnormally low implantation of the placenta in the uterus. Three types of placenta previa are defined, depending on the relationship between the cervical os (rather than the fetal presenting part itself).

- 1. Complete previa (37%) the internal os is completely covered.
- 2. Partial previa (27%) the internal os is partially covered.
- 3. Marginal previa Part of the internal os is encroached on by the placenta.

The incidence varies between 0.5% to 1% of pregnancies, usually in association with prior uterine scarring such as a previous caesarean section, uterine surgery, or a previous placenta previa. Bleeding is caused by tearing of the placenta and its attachment from the decidua.

The classic sign of placenta previa include:

- 1. Painless vaginal bleeding during the second or third trimester.
- 2. With the first episode of bleeding, contractions typically are absent.
- 3. Onset of bleeding is not related to any particular event.

When the diagnosis of placenta previa suspected, the position of the placenta should be confirmed by ultrasonography¹¹.

Abruptio placentae

Abruptio placentae is separation of a normally implanted placenta from deciduas basalis after 20 weeks of gestation and prior to delivery (incidence 0.2% to 2%). It is classified as mild, moderate, or severe. Fetal distress occurs because of loss of area for maternal fetal gas exchange. When the separation involves only the placental margin, the escaping blood can appear as vaginal bleeding. Alternatively, large volumes of blood loss (1-2 litres) can remain entirely concealed in the uterus. Chronic bleeding and clotting between the uterus and placenta can cause maternal disseminated intravascular coagulation in 10% of cases. However, if fetal demise occurs, the incidence is much higher (up to $50\%)^{12}$.

Placenta accreta

Placenta accreta is defined as an abnormally adherent placenta. *Placenta accreta vera* is defined as adherence to the myometrium without invasion of or passage through uterine muscle. *Placenta increta* represents invasion of the myometrium. *Placenta percreta* includes invasion of the uterine serosa or other pelvic structures. Any of these placental implantations can produce a markedly adherent placenta that cannot be removed without tearing the myometrium.

The combination of a placenta previa and a previous uterine scar increases the risk significantly. In the general obstetric population, placenta accreta occurs in approximately 1 in 2500 and cannot be reliably diagnosed by ultrasonography. In patients with placenta previa and no previous caesarean sections, the incidence is 5% to 7%. With one previous uterine incision, the incidence of placenta accreta has been reported to be 24% to 31% and with two or more previous uterine incisions, the incidence rises to about 50% 13,14 .

Ultrasonography and MRI may be useful when abnormal placentation is suspected. However, they both have a poor sensitivity and the diagnosis is often made on opening the abdomen and uterus. Therefore, the anesthesiologist must keep in mind the possibility and be prepared to treat sudden massive blood loss.

Uterine rupture

Rupture of the gravid uterus can be disastrous to both the mother and fetus. Fortunately, it does not occur often. Uterine rupture can be associated with separation of a previous caesarean section and healed incision (scar) in the uterus, rapid spontaneous delivery, or excessive oxytocin stimulation. Moreover, trauma during attempted forceps delivery also may cause uterine rupture in a patient with an unscarred uterus. Overall, however, more than 80% of uterine ruptures are spontaneous and without an obvious explanation.

The rupture of a classic uterine scar increases morbidity and mortality because the anterior uterine wall is highly vascular and also may include the area of placental implantation. Lateral extension of the rupture can involve the major uterine vessels and typically is associated with massive bleeding. Therefore, when vaginal birth is planned after a previous caesarean section, it is required that a surgical team, including an obstetrician, anesthesiologist, and nursing staff be available, so that emergency caesarean section can be initiated without delay should uterine rupture occur.

POSTPARTUM HEMORRHAGE

Bleeding after childbirth (postpartum hemorrhage) is an important cause of maternal mortality, accounting for nearly one quarter of all maternal deaths worldwide. Common causes for postpartum hemorrhage (PPH) include failure of the uterus to contract adequately after birth leading to atonic PPH, tears of the genital tract leading to traumatic PPH and bleeding due to retention of placental tissue. Atonic PPH is the most common cause of PPH and the leading cause of maternal death.

Uterine atony

Uterine atony is the leading cause of PPH, observed alone in 50% to 60% of cases, it presents as painless continuous bleeding, often developing slowly at the beginning. Blood can be concealed in the uterus and not exteriorized until external compression of the uterine fundus is performed. With up to 15% of maternal cardiac output at term supplying the gravid uterus, an atonic uterus can lose 2 liters of blood in 5 minutes. Atony is associated with "overdistension" of the uterus (multiparity, polyhydramnios, multiple gestation), as well as retained placenta, excessive oxytocin use during labor, and operative intervention. Initial management is medical – fluid resuscitation

to restore blood volume; oxygen supplementation (high flows via face mask); bladder emptying, uterine massage, and uterotonics. This may allow avoidance of operative intervention.

Among the uterotonics (Table 2) oxytocin is the initial therapy – up to 40 IU/ L infused as rapidly as possible¹⁵⁻¹⁸. Oxytocin is a systemic vasodilator, and may aggravate hypotension. Increasing this dose does not offer any benefit. Methylergonovine (Methergine) is a second line agent, though it is often bypassed in favour of the prostaglandin analog, carboprost (Hemabate). Neither methylergonovine nor carboprost should be administered intravenously - both are given intramuscularly. Methylergonovine is administered at a dose of 0.2 mg¹⁷; if two subsequent doses do not appropriately increase uterine tone, carboprost (0.25 mg) is administered either intramuscularly or into the myometrium. The total dose of carboprost should not exceed 1.5 mg. Both methylergonovine and carboprost have significant side effects. Methylergonovine is a peripheral vasoconstrictor; rapid intravenous injection can cause acute hypertension and cerebrovascular accident, and has been associated with pulmonary oedema and coronary vasospasm. Carboprost is a synthetic analog of prostaglandin F2 α ; it is a potent and pulmonary vasoconstrictor, systemic and bronchoconstrictor¹⁸. Intravenous administration can be associated with severe bronchospasm, and systemic and pulmonary hypertension, even intramuscular administration should be used with caution in asthma.

Retained placenta

This is the second most important etiology of postpartum hemorrhage (roughly 20% - 30% of cases),

Medication	Class	Administration	Dosing	Side effects	Comments
Oxytocin	Neurohypophyseal	Infusion	Up to 40 IU/l	Hypotension with rapid infusion	Initial therapy
Methylergonovine	Ergot alkaloid	Intramuscular	0.4 mg IM; repeat once	Hypertension	Sustained increase in uterine tone
Carboprost	Prostaglandin	Intramuscular	0.25 mg 1M repeat up to 1.0 mg total	Systemic & pulmonary hypertension, bronchospasm	Never administer intravenously

Table 2 Commonly used uterotonics

but it must be systematically investigated first, because uterine atony is frequently associated and can be misleading¹². It is suggested by the finding of an absent or incomplete delivery of placenta. Hence, uterus will not contract, and arteries of the deciduas basalis will continue to bleed. The degree of hemorrhage is often not severe, but it may be insidious, and visual estimates are inaccurate. The condition usually necessitates manual exploration of the uterus.

Genital tract lacerations

The most common injuries incurred at child birth are lacerations and hematomas of the perineum, vagina, and cervix. Most injuries have minimal consequence, but some puerperal lacerations and hematomas are associated with significant hemorrhage, either immediate or delayed. Genital tract lacerations should be suspected in all patients who have vaginal bleeding despite a firm, contracted uterus. The cervix and vagina must be inspected carefully in these patients. Retroperitoneal hematomas are dangerous because they can become large and develop insidiously, and treatment often requires exploratory laparotomy, blood transfusion, and possibly hysterectomy.

Uterine inversion

An atonic uterus and an open cervix allow the uterus to "turn inside out" through the birth canal. Fundal pressure and inappropriate traction on the umbilical cord to hasten placental delivery contribute to uterine inversion. The diagnosis is usually obvious. Clinical features include abdominal pain and often severe hemodynamic instability. Along with that a reflex bradycardia can be immediate by the effect of traction on the ligaments supporting the uterus. Uterine relaxation needed for reduction of the uterus with short-time tocolysis (trinitrite) as first line, using a potent vasopressor intravenously at the same time to counteract hypotension (phenylphrine or adrenaline)¹².

MANAGEMENT

General

The initial assessment of the patient with an obstetric hemorrhage depends on its cause, but in general (a) take a detailed medical and obstetrical history and examine the patient to find the site and cause of the bleeding; (b) empty the patient's bladder; (c) ensure that there are no retained products or genital tract lacerations; (d) estimate blood loss; and (e) assess the patient's hemodynamic status and initiate appropriate resuscitation.

Monitor

- 1. Monitor ECG, blood pressure and oxygen saturation continuously.
- 2. Monitor urine output hourly.
- 3. Consider invasive monitoring if the patient is hemodynamically unstable or repeated venepunture is anticipated.

Resuscitate

The aim of resuscitation is to restore circulating blood volume and to maintain tissue perfusion: (a) high-flow oxygen (8L/min), (b) head-down tilt and left lateral tilt to avoid aorto-caval compression (if not yet delivered); (c) intravenous access (*two* 14 or 16 gauge cannula) and take blood for complete blood count, clotting and cross-match; (d) fluids: crystalloids, colloid (avoiding dextrans) and if necessary (not to exceed 3.5 liters prior to blood transfusion), blood; (e) O Rhesus negative blood should be immediately available, type specific cross-match will be time consuming; (f) try to avoid dilutional coagulopathy with emperical administration of clotting products, (g) accept hemoglobin of 8 gm/dl.

As hypothermia impairs coagulation and shifting of oxygen dissociation curve to left (impair oxygen release at tissue level), fluids should be warned and the patient kept warm with active warming devices or warmed blankets. Appropriate correction of acidosis and hypocalcaemia is also required.

Because timing is the essence, all members of the obstetric team should be familiar with the protocol (**Box 1**).

Stop the Bleeding

Pharmacological

 Oxytocin is the drug of choice for the prevention and treatment of atonic PPH. Five to 10 units are given slowly by intravenous bolus followed by an infusion of 10 units per hour. Vasodilation produced by oxytocin may cause hypotension in cardiac or hemodynamically unstable patients.

- 2) Ergometrine (0.5 mg intravenously or intramuscularly) is as effective as oxytocin, however, intravenous administration possess undesirable effects. The agent is contraindicated in hypertensives due to its blood pressure raising effects.
- 3) Prostaglandins:
 - A) Hemabate (250 mcg intramuscularly or intramyometrially) contracts the walls of uterus. However, the agent has potential liability to cause life threatening bronchospasm¹⁹.
 - B) Misoprostol can be administered orally, sublingually or rectally. Oral or sublingual prostaglandin may be useful in cases in which injectable uterotonics are not available or practical. However, the World

Box 1 Bleeding parturient management

- Provide early diagnosis, treat the cause
- Follow general principles of resuscitation (airway, breathing, and circulation)
- Call for help
- Begin second large-bore intravenous line
- Order blood tests (hemoglobin, coagulation screen, cross-match)
- Order blood (hematology consultation?)
- Provide crystalloid/colloid (Pentastarch) to maintain isovolaemia
- Start high-pressure infusion system
- Begin arterial line (serial hemoglobin, coagulation studies)
- Provide air warming blanket
- Provide auto transfusion device?
- Begin central venous pressure line (after stabilization)
- Begin prompt treatment of clotting disorders
- Monitor urine output
- Consider use of vasopressors

Health Organization takes a different view, concluding that oral misoprostol is more expensive, has more side effects, and, crucially, is less effective than oxytocin²⁰.

Invasive/surgical iliac artery or uterine artery ligation (bleeding unresponsive to oxytocics)

There is little in the literature to guide practice when pharmacological treatment fails. However, bimannual compression, uterine balloon tamponade, arterial embolization, uterine compression sutures, and uterine artery or internal iliac artery ligation are commonly practiced procedures. The review by Doumouchtsis et al²¹ of the conservative management of PPH found no statistical difference between the various methods, and the Cochrane collaboration²² failed to identify any relevant randomized control trials.

(1) Bimannual compression

Compression of the uterus between one hand in the vagina and, another on the anterior abdominal wall, thereby reducing the bleeding.

(2) Uterine balloon tamponade

In the case of intractable PPH, Doumouchtsis et al²¹ proposes balloon tamponade as the most straight forward, rapid, and least invasive surgical option. An overall success rate of 84% has been reported ^{21,23}. Various balloon devices have been used, with the Sengstaken–Blakemore esophageal catheter being the most frequently employed²¹.

(3) Uterine arterial embolization

It has become a well-recognized alternative method of treatment in the conservative management of PPH in association with local or medical treatment, or in the event of their failure. This therapeutic approach avoids morbidity associated with peripartum hysterectomy and preserve fertility.

The reported success rate of uterine artery embolization in the literature is more than 90% $^{24-27}$. In most patients, fertility is preserved and normal menstruation returns almost $100\%^{21}$. Minor complications such as pain and fever due to inflammation are rare $(0\% - 10\%)^{27}$. More severe

complications like pelvic infection, pulmonary embolism, or necrosis of uterus and bladder have been reported but are extremely rare^{28,29}.

Transfer to the radiology suite is usually done when vital signs are stable because facilities equipped to handle major bleeding are often much better in the operating room. Placement of arterial catheters and occlusion balloons before delivery is currently the treatment options of choice whenever major haemorrhage is highly suspected. These balloons can be used to reduce blood loss significantly while the patient is prepared to undergo embolization.

(4) Uterine compression suture (B-Lynch suture)

These are useful for patients with uterine atony who respond to bimannual compression. B-Lynch et al³⁰ first described the compression suture in 1997 and others have since developed the technique using vertical³¹, transverse³², or multiple square suture^{33,34} to oppose the anterior and posterior walls of the uterus. Successful subsequent pregnancies have been reported, although evidence of fundal grooves from the sutures has been noted³¹. Uterine necrosis, intrauterine fibrous bands, abdominal adhesions, and pyometria are other noted complications³⁵.

(5) Arterial ligation

Although ligation of internal iliac, uterine arteries can reduce pelvic blood flows and pulse pressure (85%) distal to the ligation, it may be ineffective if there are extensive collaterals. It can be useful in cases of uterine atony with a success rate of 84% has been described provided they are implemented quickly²¹. Bilateral ligation of these arteries does not appear to interfere with subsequent reproduction.

(6) Peripartum hysterectomy

As a last resort, but decided on quickly when all

other interventions have failed, hysterectomy may be required to control bleeding and save life. The decision, however difficult, should be made sooner rather than later and prior to the development of coagulopathy.

(7) Adjuncts

There are some new techniques that are currently controversial and not universally accepted. These include the use of interventional radiology, methotrexate in placenta accreta, cell salvage as a means of reducing donor blood transfusion, tranexamic acid and factor VIIa to aid coagulation.

Anesthesia

Anesthetic management – both in choice and technique – should be based on a thorough understanding of the physiology of pregnancy and also on the pathophysiology of the problems.

The existence of some of the obstetric risk factors may be known early in pregnancy from history and examination (Table 3).

- (A) Detection of anemia more than physiologic anemia of pregnancy is important because anemia at delivery increases the likelihood of blood transfusion.
- (B) Coagulation studies may be required in the presence of congenital (Von Willebrand's disease) or acquired (DIC, dilutional coagulopathy, heparin) coagulation defects.
- (C) Imaging investigations are useful in the detection of placental abnormalities, with placenta previa and placenta accrete, the most important identifiable risk factors for massive hemorrhage.
- (D) Ultrasound studies identify placental location, and their ability to detect placenta accreta.

In elective placenta previa cases, Royal College

Table 3
Antenatal investigations and associated conditions

Investigations	Associated conditions
Full blood count (including hemoglobin, platelets)	Anaemia, thrombocytopenia
Clotting screen (including fibrinogen, D-dimers) Abdominal ultrasound MRI	Anticoagulants, DIC, Dilutional coagulopathy Placenta previa/accreta/increta/percreta Placenta accreta/increta/percreta

of Obstetricians and Gynaecologists states that the choice of anesthetic is at the discretion of the anesthesiologist but there is increasing evidence to support the safety of regional anesthesia. If regional anesthesia is used, consideration should be given to a combined spinal epidural technique to allow time for surgery. As general anesthesia may be necessary, the patient should be fully prepared for conversion. For placenta accreta cases, although some centers advocate the use of regional anesthesia, general anesthesia may allow for more control¹¹.

During general anesthesia, prolonged induction – delivery and uterine incision – delivery intervals were associated with a higher incidence of low Apgar scores and acidotic babies. On the other hand, with spinal anesthesia in the absence of hypotension, a longer induction – delivery interval did not alter either the Apgar score or the acid-base values of the neonates. However, a uterine incision – delivery interval of more than 180 seconds was associated with a high incidence of low Apgar scores and acidotic infants; thus, might be related to reduced placental circulation.

Advantages and disadvantages of regional vs. general anesthesia

A. Regional Anesthesia

- 1. Advantages
 - Less blood loss.
 - Awake patient with less chance of aspiration. Moreover, parturient will be able to experience delivery of baby.
- 2. Disadvantages
 - Peripheral vasodilation may exacerbate hypotension.
 - General anesthesia may be necessary for patient's comfort if a hysterectomy is necessary.
- B. General anesthesia
 - 1. Advantages
 - Hemodynamic stability.
 - Security of the airway from the onset of the surgery.
 - Comfortable patient.

- 2. Disadvantages
 - Chance of difficult intubation, inability to intubate, and possible gastric aspiration.
 - Unconscious patient.

In emergencies, hemodynamic instability and concerns over coagulopathy make general anesthesia the technique of choice. Nevertheless, if a working epidural is in place then cautious top-ups may be appropriate.

A high-dependency setting is appropriate for at least half of those with a major obstetric haemorrhage¹¹. The majority who do require intensive care do so only for mechanical ventilation and usually for less than 48 hours.

An alternative to blood transfusion in the recovery period is intravenous iron. Ferrous sucrose has an excellent safety profile³⁶.

Accurate diagnosis and appropriate management of obstetric hemorrhage can reduce maternal morbidity and mortality.

Conclusion

Obstetric hemorrhage, a leading cause of maternal as well as fetal morbidity and mortality worldwide, can be masked by early pregnancy related physiologic adaptation and complicated by abnormal placentation, ammiotic fluid emboli and infections etiologies. Obstetric hemorrhage is often sudden, unexpected and may be associated with coagulopathy. Because approximately 600-700 ml blood flows through the placental intervillous spaces each minute, obstetric hemorrhage can rapidly result in severe signs of shock.

Imaging technologies like ultrasound and magnetic resonance imaging have allowed earlier identification of the cause and institution of therapy for women at risk of hemorrhage. However, limitations in diagnostic sensitivity and specificity as well as the underestimation of blood loss and inadequate resuscitation remain common problems. Early recognition and treatment are essential to ensure the best outcome from these life threatening conditions.

References

- 1. LOLONDE A, DAVIS BA, ACOSTA A: Postpartum haemorrhage today: ICM/FIGO initiative 2004-2006. *UGO*; 2006, 94:243-253.
- HILL K, THOMAS K, ABOUZAHAR C, ET AL: Estimates of maternal mortality worldwide between 1990 and 2005: an estimate of available data. *Lancet*; 2007, 370: 1311-1319.
- BAID T: No woman should die giving life. *Lancet*; 2007, 370: 1287-1288.
- BOUVIER-COLLE MH, OULD EL JOUD D, VARNOUX N, ET AL: Evaluation of the quality of care for severe obstetrical haemorrhage in three French regions. *BJOG*; 2001, 108:898-903.
- ZEEMAN G: Obstetrical Critical Care: a blue print for improved outcomes. Crit Care Med; 2006, 34: 208-214.
- International Federation of Gynecology & Obstetrics. Prevention and treatment of postpartum haemorrhage. New advances for low score settings. ICM/FIGO. Joint Statement; November 2006.
- Scottish Confidential audit and severe maternal morbidity. SPCERH Publication No. 27, 2006.
- MAYER DC, SPIELMAN FJ, BELL EA: Antepartum and postpartum hemorrhage. In: Chestnut DH (editor). Obstetric anesthesia. Principles and practice; 3rd edition. Philadelphia: Elsevier Mosby, 2004, pp. 662-82.
- CHAN CC, To WW. Antepartum haemorrhage of unknown origin what is its clinical significance? *Acta Obstet Gynecol Scand*; 1999, 78:186-190.
- NIELSON EC, VARNER MW, SCOTT IR: The outcome of pregnancies complicated by bleeding during the second trimester. *Surg Gynecol Obstet*; 1991, 173:371-374.
- 11. WISE A, CLARK V: Strategies to manage major obstetric haemorrhage. *Curr Opin Anaesth*; 2008, 21:281-287.
- MERCIER FJ, DE VELDI MV: Major obstetric hemorrhage. Anesthesiol Cl N Am; 2008, 26:53-66.
- CHATTOPADHYAY SK, KHARIF H, SHEERBANI MM: Placenta Previa and accreta after caesarean section. *Eur J Obstet Gynecol Reprod Biol*; 1993, 52:151-156.
- CLARK SL, KOONINGS PP, PHELAN JP: Placenta accreta and prior caesarean section. *Obstet Gynecol*; 1985, 66:89-92.
- PALMER CM: Hemorrhage in Obstetrics. In: Palmer CM, D'Angelo R, Paech MJ (editors). *Handbook of Obstetric Anesthesia*; 2002, Bios Scientific Publishers Limited, Oxford. pp. 139-152.
- STOELTING RK: Pharmacology and physiology in anesthesia practice; 1987, J. B. Lippincott Co. Philadelphia, pp. 394-414.
- DOUGLAS WJ, WARD ME: Current pharmacology and the obstetric anesthesiologist. Int Anesth Clin; 1994, 32:1-10.
- Drug information for the Health Care Professional, 19th edition, vol. 1, 1999. World Color Book Services, Taunton, MA. pp. 782-784.
- HARBER C, LEVY D, CHIDAMBARAM S, MACPHERSON M: Life threatening bronchospasm after intramuscular carboprost for postpartum haemorrhage. *BJOG*; 2007, 114:366-368.

- 20. World Health Organisation. WHO recommendations for the prevention of postpartum haemorrhage, 2007.
- 21. DOUMOUCHTSIS S: Papageorghiou A, Arulkumaran S. Systemic review of conservative management of postpartum hemorrhage: what to do when medical treatment fails: *Obstetr Gynecol Surv*; 2007, 62:540-547.
- 22. MOUSA H, ALFIREVIC Z: Treatment for primary postpartum hemorrhage. The Cochrane Collaboration; January 2007.
- 23. DABELEA VG, SCHULTZE PM, MC DUFFIE RS: Intrauterine ballon tamponade in the management of postpartum hemorrhage. *J Obstet Gynaecol*; 2006, 107:38S.
- 24. DEUX JF, BAZOT M, LE BLANCHE A F, ET AL: Is selective embolization of uterine arteries a safe alternative to hysterectomy in patients with postpartum hemorrhage? *A J R*; 2001, 177:145-149.
- MITTY HA, STERLING K M, ALVAREZ M, ET AL: Obstetric haemorrhage prophylactic and emergency arterial catheterization and embolotherapy. *Radiology*; 1993, 188:183-187.
- 26. HONG TM, TSENG HS, LEE RC, ET AL: Uterine artery embolization: an effective treatment for intractable obstetric haemorrhage. *Clin Radial*; 2004, 59:96-101.
- SONCINI E, PELICELLI A, LARINI P. ET AL: Uterine artery embolization in the treatment and prevention of postpartum hemorrhage. *Int J Gynaecol Obstet*; 2007, 96:181-5.
- COTTIER JP, FIGNON A, TRANQUART F, ET AL: Uterine necrosis after arterial embolization for postpartum hemorrhage. *Obstet Gynecol*; 2002, 100:1074-1077.
- 29. PORCU G, ROGER V, JACQUIER A, ET AL: Uterus and bladder necrosis after uterine artery embolization for postpartum haemorrhage. Br J Obstet Gynecol; 2005, 112:122-123.
- B-LYNCH C, COKER A, LAWAL A, ET AL: The B-Lynch surgical technique for the control of massive postpartum haemorrhage: an alternative to hysterectomy? Five cases reported. *BJOG*; 1997, 104:372-375.
- 31. GHEZZI F, CROMI A, UCCELLA S, ET AL: The hayman technique: a simple method to treat postpartum haemorrhage. *BJOG*; 2007, 114:362-365.
- OUAHBA J, PIKETTY M, HUEL C, ET AL: Uterine compression suture for postpartum bleeding with uterine atony. *BJOG*; 2007, 114:619-622.
- CHO J, JUN H, LEE C: Haemostatic suturing technique for uterine bleeding during caesarean delivery. *Obstetr Gynecol*; 2000, 96:129-131.
- BASKETT T: Uterine compression sutures for postpartum haemorrhage. Efficacy, morbidity and subsequent pregnancy. *Obstetr Gynecol*; 2007, 101:68-71.
- OCHOA M, ALLAIRE A, STITLEY M: Pyometria after haemostatic square suture technique. *Obstetr Gynecol*; 2002, 99:506-509.
- BHANDAL N, RUSSELL R: Intravenous versus oral iron therapy for postpartum anaemia. *BJOG*; 2006, 113:1248-1252.

A. RUDRA ET. AL