

Bleeding Parturient-Anaesthetic Management

Dr. T. Murugan, MD DA
Professor of Anaesthesiology
Institute of child Health
Madras Medical college,
Chennai.

Obstetric haemorrhage a leading cause of maternal death and morbidity in developing countries as well as a cause for considerable concern in developed countries too. In India obstetric haemorrhage is responsible for more than 60% of maternal morbidity. Prevention, early recognition and prompt intervention are the keys to minimizing complications. Resuscitation can be inadequate because of underestimation of blood loss and misleading maternal response. Risk factors for haemorrhage should be identified antenatally, using all possible imaging modalities available, and utilizing multidisciplinary resources whenever possible.

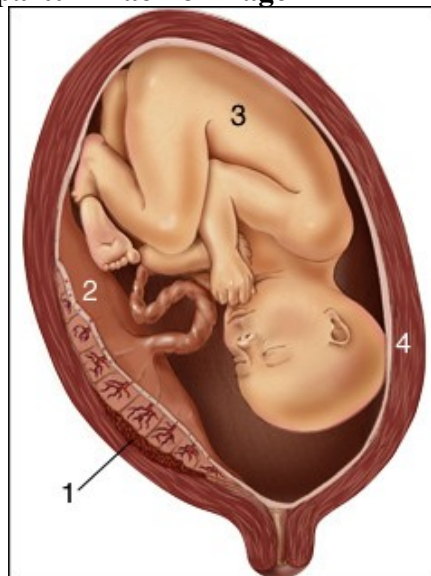
Aetiology of Obstetric haemorrhage

Causes of maternal haemorrhage are also classified by their timing of occurrence. Causes of antepartum haemorrhage (4% of pregnancies) include placenta previa, abruption placenta and uterine rupture. Postpartum haemorrhage (10% of deliveries), can be defined as a blood loss after delivery of more than 500 ml, or a 10% decrease in hematocrit from admission. Causes of early postpartum haemorrhage (first 24 hours) include uterine atony, genital laceration, retained placenta and uterine inversion. Parturients with antepartum haemorrhage are also at risk for postpartum bleeding.

Causes of Obstetric Haemorrhage

Early Pregnancy	Late Pregnancy
Abortions	Abruptio placenta
Molar Pregnancy	Placenta previa
Ectopic Pregnancy	Intra-/postpartum Uterine atony
	Retained placenta
	Rupture uterus
	Genital tract trauma
	Uterine inversion

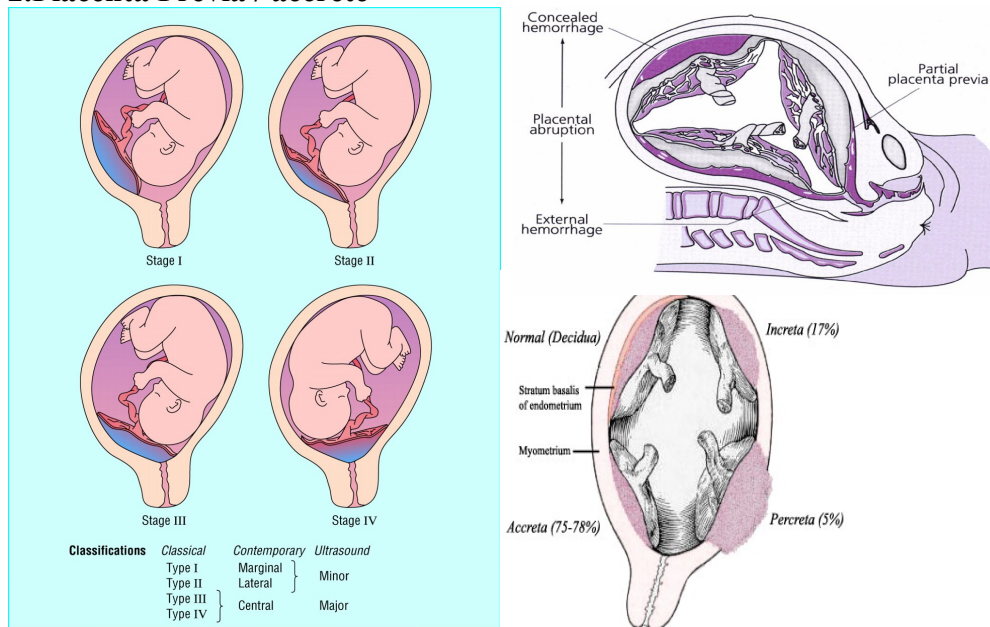
A. Ante partum haemorrhage



1. Placental abruption.

Placental abruption is defined as separation of the placenta from the deciduas basalis before delivery of the foetus. Associated conditions are hypertension (Chronic or PIH), premature rupture of membranes, previous abruption, high parity, smoking, cocaine abuse, trauma and sudden decompression of polyhydramnios. The foetal status, the maternal haemodynamics and coagulation status correlate with the degree of placental separation. Haemorrhage may be obvious or concealed (up to 2.5L). A concealed haematoma increases intrauterine pressure, and amniotic debris may be forced through open venous sinus, provoking disseminated intravascular coagulopathy (10%). The observed clotting defects has been hypothesised to be due to 1) Activation of circulating plasminogen, 2) Thromboplastin from placenta and deciduas triggers the activation of the extrinsic clotting pathway causing thrombin to convert fibrinogen to fibrin (DIC). The end result is hypofibrinogenaemia, platelet deficiency and decreased factors V and VIII. Once the clotting mechanism has been activated degeneration products of the fibrin-fibrinogen system also appear in the circulation. The patient then manifests widespread bleeding from the intravenous sites, gastrointestinal tract and subcutaneous tissues as well as the uterus.

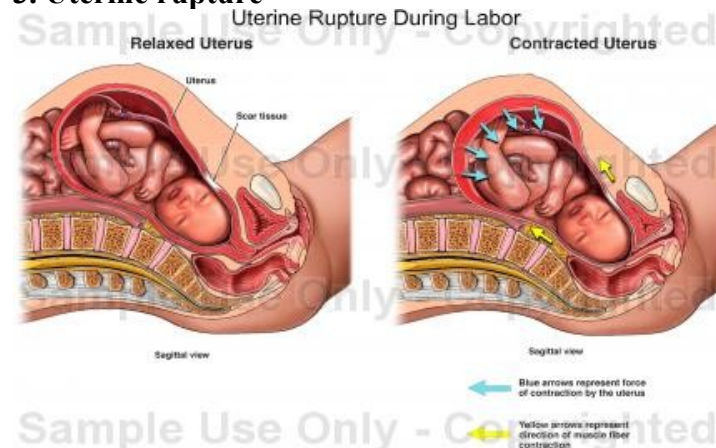
2.Placenta Previa / accrete



A placenta previa occurs when placental implantation takes place in the lower segment of the uterus in front of foetal presentation. It varies in degree and may be total, partial and marginal. Risk factors include prior placenta previa, uterine scar, advanced maternal age and multiparity. Placenta previa accounts for one third of antepartum bleeding but rarely puts the life of the mother or the foetus at risk because the bleeding usually stops spontaneously. Antepartum bleeding is the result of placenta separation during cervical dilation and lengthening of the lower uterine segment. Diagnosis is confirmed by ultrasound in more than 95% of cases a marginal previa from a low-lying placenta so a double set-up examination is rarely necessary. The double set-up refers to a vaginal examination in an operating room prepared with all personnel and equipments necessary to do and immediate caesarean section if profuse bleeding should occur. On occasions, it may still be utilised for definitive diagnosis. Placenta percreta is penetration through the full thickness of the myometrium. With this, implantations can occur on the bowel, bladder or other pelvic organs and vessels. These abnormal placental implantations occur more commonly in

placenta percreta are rare but can be diagnosed by ultrasound. Placenta Accreta is not reliably diagnosed until the uterus is open.

3. Uterine rupture



The most frequent variety of disruption is uterine scar separation. Uterine rupture involves separation of the old incision with possible extension and rupture of the foetal membranes, with either all or part of the foetus extruded into peritoneal cavity. The incidence of uterine rupture has increased from less than 1% to 4-5% in the last 10 years because of the increased incidence of vaginal birth after caesarean. The most common sign of uterine rupture is a non-reassuring foetal heart rate pattern, with variable decelerations that may evolve into late decelerations, bradycardia and loss of foetal heart rate/ severe abdominal pain is rarely a presenting symptom.

B. Postpartum Haemorrhage

Postpartum haemorrhage (PPH) is a potentially life threatening complication of both vaginal and caesarean delivery. Traditionally, PPH was defined as blood loss greater than 1000ml in a caesarean delivery. Any bleeding that results in signs and symptoms of haemodynamic instability if untreated, is considered PPH. Blood loss of greater than 1,000ml with vaginal delivery or a decrease in postpartum haematocrit level greater than 10% of the prenatal value also can be considered PPH. Arbitrarily, PPH can be divided into 1) early PPH that occurs within 24 hours after delivery and 2) late PPH that occurs 24 hours to 6 weeks after delivery. Disorders of coagulation and thrombocytopenia pre-existing or occurring during the second or third stage of labour may be associated with excessive bleeding.

1. Uterine atony

Uterine Atony is the most frequent cause of PPH-a condition in which the uterine corpus does not contract properly, allowing continued blood loss from the placental site. It may occur alone or in association with placenta previa, placental abruption or retained placenta. Atony is the most common complication that leads to blood transfusion. Some of the factors associated with uterine atony are multiple gestation, macrosomia, polyhydramios, high parity, prolonged labour, excessive use of oxytocin and chorioamnionitis. An atonic uterus may contain up to 1L of blood.

2. Retained Placenta

Retention of part or all of the placenta which may be associated with immediate haemorrhage, delayed haemorrhage or both. Haemorrhage may be massive but is usually less than 1 litre and occasionally minimal. The incidence of retained placenta is approximately 1% of all vaginal deliveries and retained products of conception usually require manual exploration of the uterus. Retained products of conception are one of the leading causes of late haemorrhage but are rarely massive.

3. Genital tract trauma

Lacerations of the cervix and / or vagina are the second most frequent cause. Trauma during delivery may result in haematomas in the perineum or pelvis. These haematomas may be palpable and should be suspected if the patient has unstable vital signs and little or no external bleeding. In a bleeding parturient genital trauma must always be eliminated first if the uterus is firm. Genital lacerations are common cause of bleeding but rarely result in massive bleeding unless large vessels are involved or if the bleeding occurs after the patient has been transferred to the postpartum unit. Rarely bleeding from genital tract lacerations extends into the retroperitoneal space. Vaginal and vulval haematomas are usually self limiting but retroperitoneal haematomas may be extensive and life threatening.

4.Uterine inversion

This is an uncommon complication particularly seen in underdeveloped and developing countries. Uterine inversion may be associated with haemorrhage of approximately 2-litres. 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 also contribute to uterine inversion. Prompt repositioning of the uterus is mandatory and if this fails, a surgical intervention is mandatory because blood loss may be rapid.

5.)Uterine Rupture

Uterine rupture may be associated with vaginal bleeding but it should be considered in the presence of severe abdominal pain and unstable hemodynamic findings.

b) Lab Studies

Complete blood count: This is determined to evaluate the haemoglobin and haematocrit levels. In a patient with acute haemorrhage several hours may pass before levels change to reflect the blood loss and platelet count. Thrombocytopenia is frequently observed.

1. The prothrombin time (PT) and activated partial thromboplastin time (APPT) are assessed to determine if a coagulation disorder is present.
2. D-dimer tests (monoclonal antibody test) may be performed to determine if levels of serum fibrin degradation products are increased. This finding indicates a coagulation disorder.

C. Imaging Studies

Ultrasonography may be helpful in revealing abnormalities within the uterine cavity and occult haematomas. Angiography may be used occasionally, with possible embolization of bleeding vessels.

D.Management of PPH

The patient with suspected or obvious PPH requires immediate intervention, similar to any patient with haemorrhage. In addition to initiating resuscitative measures, carefully evaluate the patient to determine the cause of the haemorrhage and initiate specific treatments.

- Resuscitative measures include the following 1) administration of 100% oxygen, 2) Placement of several intravenous lines with large-bore catheters and infusion of crystalloid solutions (isotonic sodium chloride or lactated Ringer solution warmed, if possible) and 3) cardiac blood pressure, pulse, pulse oxymetry monitoring.
- Obtain sample for laboratory test, with special instructions to the laboratory personnel regarding determination of the cause of bleeding.
- Type and cross match packed red blood cells for transfusion. If the patient is in critical condition, warmers permitting rapid infusion are preferred.

- Perform manual or bimanual massage. This stimulates uterine contractions and frequently stops uterine haemorrhage. Exercise caution not to use excessive pressure on the fundus of the uterus, this may increase the risk of uterine inversion.
- Oxytocic agents, such as oxytocin, ergonovine, methylergonovine and 15-methyl-prostaglandin are used to stimulate uterine contraction and control haemorrhage. These agents are routinely used in PPH.
- Direct pressure over laceration in the perineum, cervix, vagina or uterus may help control bleeding.
- Check the placenta for evidence of missing placental tissue, which still may be attached to the wall of the uterus, causing excessive bleeding. Removal of retained tissue can be difficult and painful.
- If uterine inversion occurs, gently push the uterus back into position. Fortunately, when this inversion occurs on an emergency basis, the cervix generally does not have time to contract firmly around the inverted uterus.
- If the patient has coagulopathy, consider the transfusion of fresh frozen plasma. If the patient is thrombocytopenic consider platelet transfusion.
- In cases of uterine rupture, emergent laparotomy is required.

Postpartum haemorrhage usually responds to limited conservative therapy such as intrauterine manual examination, uterine massage and uterotonic maintenance of maternal haemodynamics. However, when the haemorrhagic process continues and when either clotting abnormalities or haemodynamic instability develop, the next step is often to either opt for a conservative approach by embolizing the selective pelvic vessels or ligating the hypogastric arteries or proceed to perform a hysterectomy. Other measures which have been tried successfully in the management of severe postpartum haemorrhage include uterine balloon tamponade using a foley's catheter, intrauterine irrigation with prostaglandins. Angiographic selective embolisation (ASE) for obstetric haemorrhage are mainly reported in pelvic lacerations and atony of the uterus with 90% and 100% success rate respectively

MANAGEMENT OF OBSTETRIC HAEMORRHAGE

a) Maternal response to blood loss

The gravid uterus receives 12% of the cardiac output and thus when haemorrhage occurs it can be extremely rapid. The foetus is at greater risk from maternal haemorrhage than the mother. Physiological increase in blood volume and cardiac output, along with decrease in the systemic vascular resistance resulting from vasodilator effects of pregnancy hormones and low-resistance utero-placenta circulation, allow the pregnant women to tolerate more blood loss than the non-pregnant. But massive blood loss (>500) can be dangerous to the mother because of lack of vascular auto-regulation, as the normal vasoconstrictor response to elevate systemic vascular resistance is impeded by arteriovenous fistula of uteroplacental circulation. Hypotension reduces uteroplacental blood flow and severe anaemia will further reduce oxygen delivery in addition. Abruption may directly compromise blood supply. Foetal mortality may be as high as 35%.

Within 4 hours of a significant haemorrhage in pregnancy, fluid shifts occur from interstitial space to reduce the degree of hypovolemia. As the blood volume deficit approaches 25%, compensatory mechanisms become inadequate to maintain cardiac output and arterial pressure, resulting in shock and loss of capillary membrane integrity with additional loss of intra vascular fluid volume into extra vascular space. Shock also alters electrolyte transport resulting in an increase in the intercellular

sodium and chloride concentrations which tend to pull fluid from interstitial space in to the cell. Therefore haemorrhage shock is associated with decreased in both intravascular and interstitial fluid volumes.

The parturient is also at high risk for pulmonary oedema because of decreased colloid osmotic pressure, pulmonary vasodilatation and decreased gradient between colloid osmotic pressure. In addition the enlarging abdomen may cause reduced functional residual capacity which along with higher oxygen consumption during pregnancy increases the risk of hypoxia.

If local tissue hypoxia and metabolic acidosis are not corrected properly shunting of blood from renal and splanchnic beds may result in acute tubular necrosis and contributes to pulmonary endothelial damage and ARDS even if resuscitation is eventually successful. Hypovolemic shock can also result in platelet aggregation, release of vasoactive substances and impaired microcirculatory perfusion resulting in disseminated intravascular coagulation.

Utero- Placental blood flow is diminished along with reduced splanchnic perfusion as a compensatory response to hypovolemia resulting from massive haemorrhage. This may reduce foetal oxygenation causing foetal distress even before the mother shows clinical signs of shock. Thus foetus may recover as the maternal hypoxia, acidosis and under perfusion of uteroplacental unit are corrected.

Whatever be the cause of haemorrhage, primary goal is quick restoration of blood volume, with adequate oxygen carrying capacity, so as to maintain perfusion and oxygenation, till the definitive treatment for blood loss is instituted. Mortality in obstetric haemorrhage is often attributed to errors in management resulting from inadequate fluid and blood replacement or overzealous fluid infusion as well as unreasonable delay in termination of pregnancy or surgical intervention. Intensivist and anaesthesiologist should manage these patients jointly with the obstetrician and a consultant haematologist should also be involved in patient with coagulation disorders or for women requiring massive blood transfusions. Wide bore IV access should be established for rapid transfusion and blood sample should be taken for grouping and cross matching, haemoglobin, hematocrit, platelet count and coagulation profile. Bedside bleeding time, clotting time, clot retraction time can be tested but they do not provide accurate estimate and full coagulation profile must be done. Oxygen should be delivered by facemask or IPPV (if airways are not patent or inadequate tidal volume) to maintain adequate SpO₂ until haemoglobin can be replaced.

b)Assessment of blood loss

Clinical assessment of volume loss can be made according to the classification suggested by Baker. But the assessment during pregnancy can often be fallacious due to compounding factors as pregnancy induced hypertension, where associated intravascular volume depletion may result in haemodynamic instability, even with class I haemorrhage. On the contrary, severe hypovolemia may go undetected in a patient with pre-existing hypertension, which may show normal blood pressure despite significant haemorrhage.

Classification of Haemorrhage in Pregnancy

Class	Acute blood loss(ml)	%Lost	Clinical findings
-------	----------------------	-------	-------------------

I	<1000	15	None
II	1200-1500	15-25	Orthostatic blood pressure changes, positive tilt test, pulse pressure < 30mmHg, reduced peripheral perfusion with prolonged capillary refill time.
III	1500-2000	25-35	Cold clammy skin, tachycardia, tachypnea, hypotension.
IV	<2000	>35	Profound shock, nonpalpable blood pressure.

c) Diagnosis of haemorrhage

Diagnosis of haemorrhage is usually self evident, although one should be aware that concealed bleeding may occur, especially with placental abruptions. In addition, sign of cardiovascular decompensation may be delayed, as women are usually young, fit and start with a pregnancy induced expansion of their intravascular volume. Also the anaesthesiologist should be cautious of the woman with cold peripheries as this sign is also abnormal in pregnancy. Hypotension is a late and ominous sign.

Detection of anaemia more than physiologic anaemia at delivery increases the likelihood of a woman requiring blood transfusion. Iron studies may demonstrate deficiency. Coagulation studies may be required in the presence of congenital or acquired coagulation defects. Elevated creatine kinase may be a marker of invasion in cases of probable abnormal adherence of the placenta.

Imaging investigations are useful in the detection of placental abnormalities, with detection of placental abnormalities, with placenta praevia and placenta accreta the most important identifiable risk factors for massive haemorrhage. Ultrasound studies identify placenta accreta has been well examined and reviewed.

Predelivery treatment of known coagulation defects may be required. Examples include the use of desmopressin acetate (DDAVP) in von Willebrand's disease type I, Haemophilia carriers with low factor VIII levels, and other specific factor treatment in which deficiency exists. Pregnant women with procoagulant condition often are treated with non-steroidal anti-inflammatory drugs, including aspirin, oral anticoagulants such as warfarin, or more commonly, unfractionated or low molecular weight heparin. Haemorrhage risk may be reduced by either stopping or reducing therapy or by switching therapy to unfractionated heparin, which provides more measurable and reversible anticoagulation.

Bleeding Parturient Management-Protocol

-
- Provide early diagnosis, treat the cause
 - Follow general principles of resuscitation (Airway, breathing, circulation)
 - Call for help
 - Begin large-bore intravenous line (14G/16G omr or two cannulae)
 - Order blood test (haemoglobin, coagulation profile, cross match)
 - Order blood (haemoglobin, coagulation, if possible)

- Oxygen by mask at 8lit/min.
 - Infuse crystalloid/colloid(Pentastarch/Polygeline) to maintain isovolemia
 - Start high- pressure infusion system
 - Pulse and BP monitoring with Oxymeter and Non-invasive BP monitor
 - Insert arterial line (serial Haemoglobin, coagulation studies)
 - Insert a central venous pressure line (After stabilization)
 - Begin prompt treatment of clotting disorder Consider use of Vasopressors
 - Monitor urine output (Foley’s catheter)
 - Maintenance of Intravascular volume and normotension with crystalloid and colloid solutions
 - Maintenance of normothermia helps prevent coagulopathy, metabolic acidosis and even ventricular fibrillation.
-

Because obstetric haemorrhage can occur unexpectedly, certain measures may be needed to limit acute torrential haemorrhage, thereby allowing time or resuscitation and treatment or transfer to a site where definitive treatment may be expedited.

Obstetric Haemorrhage: Fluid Therapy

1.Initial therapy

- Crystalloid (Ringer lacted /0.9%saline)maximum 2 liters.
- Colloid (hetastarch, polygeline, human albumin, 4.5%- maximum 1.5 litres
- Do not use dextran

2. Transfuse blood as soon as possible

- If cross-matched blood is still unavailable and 3.5 litres of crystalloid/ colloid is infused then
- Give ‘O’ negative blood or
- Uncross matched, own group blood

3. If bleeding is unrelenting and results of coagulation studies are still unavailable

- Give I litre fresh frozen plasma
 - Give cryoprecipitate empirically
 - Use best equipment available to achieve rapid warmed infusion of fluids
 - Do not use special blood filters: they slow infusions.
-

Once haemorrhage has been controlled, the anaesthesiologist must consult with obstetrician and other regarding further care. Patients who have received massive transfusion are best managed in a high dependency or intensive care unit for close monitoring for complications. Removal of packs may involve risk of further haemorrhage, necessitating appropriate planning.

Uterotonics

As the patient is being stabilized, underlying cause of haemorrhage has be diagnosed and steps have to be taken simultaneously to arrest bleeding. Because uterine hypotonia leads to haemorrhage, prophylactic or early administration of oxytocics assists in minimizing blood loss. A range of uterotonic drugs is given to treat hypotonia.

Commonly used Uterotonics and their Doses

Drug	Doses	Information
Oxytocin -Synthetically	5-10 IU bolus 30-50UI in 500ml	causing uterine contraction, peripheral vasodilation and has very

produced hormone (syntocinon)		mild anti diuretic hormone action.
Ergometrine	0.5 mg IM or 0.125mg Slow IV injection	An ergot alkaloid derivative. Produces effective uterine contraction but nausea and vomiting are very common. Systemic vasoconstriction may produce dangerous hypertension in at risk groups(e.g.per-eclampsia, specific cardiac disease)
Carboprost (15 Methyl Prostaglandin F _{2alpha})	0.25 mg intramyometrially or IM. IM every 10-15min To a max. of 2mg	Effective uterine constrictor also causes nausea, vomiting and diarrhoea. May produce severe broncho spasm, alters pulmonary shunt fraction and induce hypoxia

III- Anaesthetic considerations for a bleeding Parturient

A) Anaesthesia Planning:- General or Regional Anaesthesia??

Regional Anaesthesia for anaesthesia of the bleeding parturient may be contraindicated because of hypovolemia or coagulopathy. For planned caesarean section without such contra-indications, some retrospective studies suggest that general anaesthesia with a volatile agent is associated with greater blood loss than regional techniques although some other disagree. A retrospective analysis of 541 parturients with placenta previa who underwent caesarean section identified general anaesthesia as a risk factor for greater blood loss and a need for transfusion. There was no difference in the incidence of intraoperative or anaesthetic complications, and regional anaesthesia was found to be a safe alternative to general anaesthesia. Using regression analysis of data collected during a review of 350 cases of placenta previa regional anaesthesia was associated with a significantly reduced estimated blood loss and need for transfusion. Sevoflurane, isoflurane and halothane all cause inhibition of the uterine contraction in a dose related manner and should be used cautiously. Other issues relevant to planning and expediting the anaesthetic management of the potentially haemorrhaging parturient include preparing transfusion apparatus: adequate large bore intravenous access, Fluid warmer, rapid infuser, warming mattress, warm air blanket and staff to porter blood work and blood products and assist with fluid therapy, Providing appropriate monitoring arterial line for beat to beat blood pressure and frequent blood sampling, central venous line and urometer to monitor renal function and adequate facilities and staff with sufficient expertise in management to massive haemorrhage.

B. Surgical Planning

When major haemorrhage is anticipated at caesarean section, a surgical plan is necessary. Placement of femoral artery cannulae with internal aortic or bilateral iliac balloons allows intra operative balloon inflation to help control major haemorrhage. The cannulae also can be used as access for later embolization. A urologist may place ureteric stents preoperatively to reduce the risk of urologic injury if hysterectomy is planned or required and the urologist is invaluable if the placenta invades the bladder. A vascular or specialist gynaecologic oncologic surgeon may be needed if hypogastric

artery ligation or aortic dissection is required. Ligatures may be placed around the vessels after dissection before hysterectomy so that vessels may be tied off if needed. Blood loss in complication caesarean section is reduced if the placenta is allowed to separate and, deliver spontaneously rather than be removed manually by the obstetrician. Allowing spontaneous placental separation provides maximal decrease in implantation bed surface area and spinal artery perfusion pressure. If there is abnormal adherence, alternative strategies might be considered without necessarily initiating placental separation and major haemorrhage. One example is proceeding directly to hysterectomy with placenta in situ.

If the coagulation profile is normal and the woman is haemodynamically stable epidural analgesia can be used to control labour pain. Caesarean section is reserved for situations such as foetal distress with heavy maternal bleeding at time because further placental separation may cause foetal death. General anaesthesia is indicated for prolonged foetal bradycardia or a haemodynamically unstable or coagulopathic parturient. Regional anaesthesia may be used in less dramatic situations, but hypotension must be treated promptly and appropriately prior to neuraxial block. During surgery, coagulopathy and uterus, may occur. Couvelaire uterus is the widespread extravasation of blood into the contraction. Hysterectomy may be required in such cases. In cases of massive abruption, intensive resuscitation of the new born is essential and , If the infant is hypovolemic, blood transfusion may be lifesaving.

C. Anaesthetic Consideration in Retained Placenta

Retained placental fragments are a leading cause of early and delayed post partum haemorrhage. Treatment is manual removal and choice of anaesthesia has been discussed previously. General anaesthesia with any volatile agent (1.5-2 minimum alveolar concentration – MAC) may be necessary for uterine relaxation. Nitroglycerin, 50 to 250 microgram intravenously or two puffs (800 microgram) inhaled, is clinically an effective uterine relaxant and is worth a try case of a contracted uterus. Nitroglycerin neither promotes separation of an adherent placenta nor provides analgesia. On rare occasions, a retained placenta is an undiagnosed placenta accreta and massive bleeding may occur during attempted manual removal.

D. Anaesthetic Consideration in Placenta previa.

An elective caesarean section is planned as soon as the foetus reaches maturity with massive blood loss and a haemodynamically unstable mother, caesarean section under general anaesthesia is the best way to deliver the infant and stabilize. If bleeding has stopped and the mother is haemodynamically stable, regional anaesthesia can be used after careful assessment. If one strongly suspects a placenta accreta, it may be safer to use general anaesthesia. Postpartum haemorrhage may occur when the surgical incision goes through an anterior placenta, there is poor contractile capacity of the implantation site (lower uterine segment being less muscular) or there is placenta accreta. The hypovolemic newborn may need resuscitation. Placenta previa grade III/IV requires delivery by caesarean section, whether the foetus is alive or dead with minor grades (I/II anterior) of placenta previa and in the absence of malpresentation, vaginal delivery can be allowed and labour augmentation with artificial rupture of membranes and oxytocin. Decision may be difficult in patients presenting with acute bleeding episodes prior to 34 week gestation. Aggressive expectant management has been advocated even in those with heavy vaginal bleeding to the point of hypovolemia. Patient is transfused to maintain a hematocrit of $\geq 30\%$ to optimize maternal foetal oxygen consumption and to provide a reserve in the event of future

heavy bleeding. Pregnancies have been prolonged significantly with the use of intravenous magnesium sulphate. β - mimetics may result in severe hypotension hence should be avoided. Corticosteroids can be administered safely for foetal lung maturity if pregnancy needs to be terminated prematurely. At time of caesarean section for placenta previa, prolonged manipulation of partially separated placenta may lead to excessive haemorrhage and foetal anoxia. Severe bleeding may also occur from the placental implantation site following removal of the placenta because of poorly contracting lower segment or due to the adherent placenta/ increta/ percreta, particularly in association with previous caesarean section. Risk of accretism with anterior placenta previa can be as high as 40% with antecedent caesarean compared to 9% in those without previous scar. The surgeon should be experienced in locating and delivering the foetal head, whether transplacentally or avoiding the placenta. Bleeding from placental site can be controlled with over sewing sutures, warm pressure packs and intra – myometrial administration of prostaglandins or oxytocin. Local vasopressin has also been used to save the uterus in intractable bleeding due to placenta accreta. Further, conservative measures like ligation of uterine arteries, ovarian or hypogastric vessels may be tried to obtain haemostasis, particularly in patients where preservation of fertility is desired. External aortic compression can be used as a resuscitative measure while conservative measures are being tried. Hysterectomy should be resorted to if conservative measures fail to control bleeding. Blood loss, infection rates and overall morbidity and overall morbidity and mortality are higher when conservative methods are tried without success. Hysterectomy if needed for placenta previa should be total hysterectomy as bleeding may continue from cervical stump.

E. Anaesthetic Consideration in Abruptio Placenta.

It is diagnosed by presence of severe pain, tense uterus, increasing abdominal girth (concealed haemorrhage). Consumptive coagulopathy may already be evident by the absence of clotting in vaginal blood, hematuria and blood loss from puncture sites. 50% patients may be in labour. Pregnancy should be terminated as soon as possible. Generally with severe abruption, the foetus is dead. Uterine contractions are stimulated with oxytocin or prostaglandins to allow for vaginal delivery except when caesarean section is indicated for obstetric reasons as transverse lie or rarely if uterus fails to respond to augmentation and bleeding is unrelenting. There could be underlying rupture of uterus occurring as a result of "Couvelaire uterus" in cases of severe abruption. With a live foetus, urgent caesarean section is performed if there is evidence of foetal distress or if the bleeding is heavy and the cervix is not favourable. Vaginal delivery may be allowed in haemodynamically stable patients, who are in active labour and have normal foetal heart pattern. Decision for vaginal delivery may also be taken if the foetus is extremely premature or is severely depressed, as a foetus with such severe hypoxia, that does not respond to maternal resuscitation, is unlikely to survive the neonatal period. Severe abruption cases have also to be watched for atonic postpartum haemorrhage and consumptive coagulopathy that must be corrected prior to any surgical intervention. Abrupt onset of bleeding that begins with rupture of membranes suggests vasa previa, especially if accompanied by decreased foetal movements and non-reassuring foetal heart. Immediate caesarean section has to be performed. Prenatal diagnosis can be made by transvaginal colour Doppler sonography to avert possible disaster.

F. Anaesthetic Consideration in PPH

Uterine massage, arterial embolization or laparotomy may be necessary to control haemorrhage from an atonic uterus. Uterine packing may be considered for stabilizing a bleeding patient or a patient with disseminated intravascular coagulopathy. Amniotic fluid embolism may present as uterine atony without pulmonary symptoms. In that situation disseminated intravascular coagulopathy is the predominant problem and treatment with blood products should be aggressive. Uterine exploration or laparotomy in a bleeding parturient may be necessary. Anaesthetic considerations are summarized below.

Anaesthetic Management in PPH

General anaesthesia (rapid sequence induction)

- Opioids (Fentanyl 1-2 micrograms/ kg)
- Ketamine 1 mg /kg
- Succinylcholine 1.5mg/ kg
- Volatile agents (Isoflurane) or total intravenous anaesthesia (Propofol, Ketamine)

Regional anaesthesia (in haemodynamically stable patient)

- T7-S4 block
 - Epidural (in situ) lignocaine 2% 10ml +/- fentanyl 25 microgram.
-

General anaesthesia is mandatory when the patient needs a laparotomy or is haemodynamically unstable. One should be prepared for massive bleeding because hysterectomy may be the final treatment. One may have to consider total intravenous anaesthesia during general anaesthesia because all volatile agents may worsen uterine atony. Regional anaesthesia may be an option in a stable patient who needs uterine or vaginal tract exploration but the anaesthesiologist need at least a T 7 block if manual uterine exploration is likely. Sedation is less than ideal because a thorough examination of the genital tract, including uterine exploration is a painful procedure. In a patient at risk for aspiration, sedation may cause the loss of protective airway reflexes in the immediate postpartum period leading to acid aspiration syndrome

CONCLUSION

Haemorrhage in obstetrics can occur with frightening speed and can lead to serious maternal and foetal outcome. The survival of patients depends significantly on the length of time patients have been in hypovolemic shock. The management of haemorrhage requires a team effort and both the obstetrician and the anaesthesiologists should strictly adhere to the principles of blood and volume replacement as outlined above along with prompt surgical decision making.

