Guide for the Care of the Most Relevant Obstetric Emergencies

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**Latin American Federation of Societies of Obstetrics and Gynecology

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Introduction

The document herein intends to be an accessible and user-friendly tool for the approach of the most frequent obstetric emergencies and urgencies. It addresses the main causes of maternal mortality, in the understanding that their correct diagnosis and management may prevent the death of pregnant women.

The maternal mortality rate is an indicator of a population’s quality of life and health care, with most maternal deaths occurring in developing countries (99%). Poor schooling, inadequate nutrition, social support and health care are strongly associated with maternal mortality, which is considered an indicator of inequality between the rich and the poor. Moreover, maternal mortality is an evidence of gender inequalities, both in terms of access to education, nutrition and health care.1-4

This situation reflects the preventable nature of maternal death and the importance of establishing extra-sectoral actions to improve quality of life, and intra-sectoral measures to guarantee all the people’s quality and access to health care. A mother’s death is a human tragedy, a social injustice and a violation to the right to live.

In many countries, maternal mortality is under-estimated as a result of a number of reasons that range from the practice of home births lacking an adequate health care, clandestine graveyards, and the paucity of skilled health staff capable of detecting and dealing with those cases.

In addition, it is important to highlight that maternal death also entails severe consequences for the family and the community. It is associated with child neglect, malnutrition, violence and poor social development of families and communities, leading to high health care and social costs, in addition to being a serious threat to the development of communities and countries.
When seeking to prevent maternal deaths, there are several actions of key importance, including the list below:

- Prevention of unwanted pregnancy. This includes access to fertility regulation methods and health actions aimed at preventing unsafe abortions.

- Accessible and quality antenatal care. Enhancing access to control services and improving the quality of that control are high impact actions for the abatement of maternal mortality.

- Adequate and humane care of deliveries and obstetric life-threatening events, including skilled monitoring of labor and delivery, as well as a timely diagnosis and proper management of the obstetric complications.

- Adequate postpartum monitoring in search of complications, and with the purpose of establishing actions that promote health-related education.

Most deaths are due to antepartum and postpartum bleeding, complications of abortion, pregnancy-related hypertensive disorders, sepsis, protracted or obstructed labor, uterus rupture and ectopic pregnancy. Some of these complications cannot be prevented; they are of acute onset, and may occur even in patients free of any risk factors. Hence the importance of recognizing these complications in a timely manner, to enable an immediate and adequate approach to prevent the woman’s status from worsening and to avoid her subsequent death. The maternal mortality rate may be strongly reduced through the implementation of proper evidence-based actions, well enforced through management protocols, and carried out by skilled health professionals.

The analysis of the impact that issues like family planning, antenatal controls, delivery care provided by skilled staff, and management at obstetric emergency services have on MM reveals that access to obstetric emergency services and safe abortion services are significantly associated with a lower maternal mortality, and their impact is even greater than the availability of antenatal control and good delivery care.5, 6

Modern concepts on the safety and quality of care of high-risk pregnant
women, the use of standardized processes, check lists, team work training and obstetric sham scenarios are some of the strategies potentially available for the improvement of obstetric emergencies, because they provide the opportunity to identify areas that need further development, and allow for high quality continuing medical education under catastrophic situations that are rare in delivery rooms.7, 8

Doctors, midwives or nurses must be adequately trained on the care of uneventful deliveries or pregnancies, as well as on the identification, management and referral of the cases with maternal complications.

The aim of this manual is to enable the health care professionals at various levels to proceed to a timely diagnosis and implementation of proper actions, whenever they face a pregnant woman with potentially life-threatening complications.

The latter include serious obstetric infections, pregnancy-, delivery- and post-partum related hemorrhages, severe complications of pregnancy-related hypertensive conditions, and a chapter referred to the care of pregnant women in cardiorespiratory arrest. The adequate management of the above may imply the difference between life and death, both for the mother and the child, and its impact is even more significant in the countries with a high maternal morbi-mortality. Hence, the programmes aimed at reducing MM in countries with limited resources must focus on the adequate management of obstetric complications.
REFERENCES


I. Serious hypertensive conditions in pregnancy

Classification of hypertensive disorders in pregnancy

- **Chronic pre-gestational hypertension**: This condition is defined by the elevation of the pressure figures \( \geq 140 \) mmHg for the systolic pressure and/or \( \geq 90 \) mmHg for the diastolic pressure, in two isolated measurements; observed before pregnancy or before the 20\(^{th}\) week of pregnancy.

- **Pre-eclampsia**: This is the presence of a diastolic blood pressure \( \geq 90 \) mmHg, or a systolic pressure \( \geq 140 \) (in two measurements) and the presence of proteinuria (defined as the evidence of proteins in urine over 300 mg in 24 hours).

- **Gestational hypertension**: It is the presence of systolic and diastolic blood pressures respectively greater or equal to 140/90, with no proteinuria, detected after the 20\(^{th}\) week of pregnancy, and that disappears within the first three months following childbirth.

- **Overlapping Pre-eclampsia**: Chronic hypertension with the addition of preeclampsia.\(^1,2\)

1. **Pre-eclampsia**

This condition is defined as the presence of pregnancy-induced hypertension with a diastolic arterial pressure (DAP) \( \geq 90 \) mmHg and/or a systolic arterial pressure (SAP) \( \geq 140 \) mmHg; in other cases it will be defined as an SAP increased by 30 mmHg or more or an increase of 15 mmHg or more in the usual DAP, all this associated with proteinuria, and at times with oedema or target organ damage.

The diastolic arterial pressure measurement is the best predictor of perinatal maternal outcomes; however, stroke correlates well with SAPs over 160 mmHg.
Pressure measurements need to be taken after the patient has rested for 20 minutes; the patient needs to be sitting, her arm rising at a 45 degree angle, at heart level. The systolic arterial pressure corresponds to the first beat heard, while the diastolic pressure is the pressure at which beats get muffled or cease to be auscultated.\(^3\)

Pre-eclampsia is classified as **severe** or **not severe**. It is said to be severe when it meets any of the criteria below:\(^4^7\)

- DAP $\geq$ 110 mm Hg, or SAP $\geq$ 160 mm Hg.
- Proteinuria levels\(^3\) 5 g in 24 hours (qualitative test: 3+).

**Table 1. Other severity criteria in pre-eclampsia**

<table>
<thead>
<tr>
<th>System</th>
<th>Severity criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Kidney</strong></td>
<td>Oliguria under 0.3 cc/kg/hour in 6 hours (less than 500 cc/day). Increased serum creatinine levels.</td>
</tr>
<tr>
<td><strong>Nervous System</strong></td>
<td>Symptoms of impending eclampsia: phosphenes, headache, epigastralgia, blurry vision or other brain or visual changes. Eclampsia</td>
</tr>
<tr>
<td><strong>Respiratory</strong></td>
<td>Pulmonary oedema and cyanosis</td>
</tr>
</tbody>
</table>
| **Blood**      | Thrombocytopenia (lower than 150,000 per cc). Increased haemoglobin levels during haemo-concentration or reduced during haemolysis (HELLP). \(^8\)  
                  | Partial Thromboplastin Time (PTT), Increased International Normalized Ratio (INR)     |
| **Liver**      | Aspartate Amino Transferase (AST) $> 40$ IU/L, Alanine Amino Transferase (ALT) $> 40$ IU/L, Lactic Dehydrogenase (LDH) $> 600$ IU/L and high serum bilirubin levels. Painful right upper quadrant |

**Diagnostic criteria for the HELLP syndrome**

- Platelets $< 100,000/mm3$
- AST and/or ALT $> 70$ IU/L
- LDH $> 600$ IU/L
Differential diagnoses

- Gestational fatty liver
- Thrombotic microangiopathies (Thrombotic thrombocytopaenic purpura and uremic-hemolytic syndrome).
- Systemic Lupus Erythematosus.
- Catastrophic Antiphospholipid Antibody Syndrome. 9

Therapy

Severe pre-eclampsia is a hypertensive emergency, particularly when associated with a hypertensive crisis with SAP > 160 and/or DAP > 110 mmHg, added to manifestations of hypertensive encephalopathy or target organ involvement. This consideration has a significant impact on the therapeutic approach, since the management of blood pressure becomes more relevant, together with the prevention of seizures and termination of pregnancy10.
### Table 2. Managing the hypertensive crises in pregnancy

<table>
<thead>
<tr>
<th>Therapy</th>
<th>Description</th>
</tr>
</thead>
</table>
| **Anti-hypertensive management** | Hypertensive urgency: BP elevation > 160/110 with no target organ damage  
- Reduction of blood pressure in 24 to 48 hours.  
- Oral antihypertensive agents.  
- Intermediate Care Unit with continuous non invasive monitoring of blood pressure.  
Hypertensive emergency: BP elevation > 160/110 with target organ damage.  
- Immediate reduction of blood pressure.  
- Intravenous vasodilators and oral anti-hypertensive agents  
- Goal of therapy in pregnancy: SAP from 140 to 150 mmHg and DAP from 90 to 100 mmHg. Reducing DAP under 90 mmHg is associated with an increased risk of uteroplacental failure.  
- Goal of therapy in postpartum: BP lower than 140/90 mmHg in a < 24-hour period\(^{10-16}\)  
- Intensive Care Unit with continuous invasive monitoring of blood pressure. (Table 3) |
| **Prevention of eclampsia**     | Magnesium sulfate (intravenous)  
Impregnation dosage: 4 to 6 grams.  
Maintenance dosage up to 24 hours postpartum: 1 to 2 grams per hour. (Table 4) |
| **Obstetric management**        | Watchful management in pregnancies under 32 weeks after maternal stabilization, seeking to reach lung maturation as long as both the maternal and fetal status allow it; only recommended at high complexity units, with maternal intensive care and high technology for fetal and neonatal health monitoring. Termination of pregnancy in gestations over 32 weeks or gestations under 32 weeks not amenable to expectant management. At high complexity units and after maternal stabilization. Vaginal delivery must be monitored continuously and there are no contraindications for the use of obstetric analgesia or regional anaesthesia if the platelet count is over 75,000/cc 6 hours before delivery |
| **Others**                      | Intravenous fluids at 1 cc/Kg/hour (including the magnesium sulfate drip).  
Thromboprophylaxis in patients with low risk factors (age >35 years old, BMI>30, venous insufficiency, multiparity, etc.) or with one of the high risk factors (thrombophilias, earlier thrombotic events, autoimmune diseases, chronic hypertension, diabetes, etc.). |
Anti-hypertensive management in hypertensive emergencies

The treatment of hypertension may prevent the potential cerebrovascular events in pre-eclampsia, but it will not alter the natural course of the disease. Below is a table with the drugs available that could be used (Table 3).

**Table 3. Drugs for hypertensive emergencies**

<table>
<thead>
<tr>
<th>Name</th>
<th>Dose</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydralazine</td>
<td>20 mg/4 cc vials; bolus of 5 -10 mg every 15 – 20 minutes, not to exceed 30 mg, or a 0.5-10 mg/hour drip</td>
<td>Adverse effects: reflex tachycardia, palpitations, hypotension (especially if there is volume depletion), headaches, anxiety, tremor, vomiting, epigastralgia and fluid retention. No teratogenic effects or severe neonatal complications have been identified.</td>
</tr>
<tr>
<td>Labetalol</td>
<td>100 mg/20cc vials: Start with 10 mg (2 cc) or 20 mg (4 cc) intravenous; if there is no response, increase to 40 mg (8 cc) and then to 80 mg (16 cc) every 10 to 15 minutes, to reach an accumulated dose of 300 mg</td>
<td>Better outcomes with the bolus administration than in continuous infusion. Adverse effects: nausea, headache and fatigue. Hypotension, hypoglycemia, hypothermia and bradycardia have been reported in neonates. Contraindicated in patients with asthma and decompensated heart failure.</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>Short action 10-miligram capsules or tablets per os every 20 minutes, up to 3 tablets and then 1 tablet every 6 hours.</td>
<td>Rapid action nifedipine: there are reports of difficult-to-manage hypertensive crises. No impact on the uteroplacental blood flow, and causes less reflex tachycardia than nifedipine.</td>
</tr>
<tr>
<td>Nicardipine</td>
<td>Infusion at 5 mg/h 2.5 mg/h increases every 5 minutes up to a maximum dosage of 10 mg/h.</td>
<td>No impact on the uteroplacental blood flow, and causes less reflex tachycardia than nifedipine.</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>Continuous infusion starting with 0.2 micrograms/kg/min, to be incremented every 5 minutes, up to a maximum of 4 micrograms/kg/min.</td>
<td>Indicated after failure of the first line regimen. Several adverse effects have been reported after 6 hours of therapy, including a high fetal death rate, headache, palpitations, sweating, ototoxicity, central nervous system dysfunction, haemodynamic instability and lactic acidosis.</td>
</tr>
</tbody>
</table>
Prevention of Eclampsia, Magnesium Sulfate

The administration of magnesium sulfate in women with severe pre-eclampsia is associated with a 58% risk of eclampsia (95% CI 40-71%). In the presence of seizures, magnesium sulfate continues to be the therapy of choice.20, 21

Therapy is started with a loading dose of 4 to 6 grams IV, followed by a 1 to 2 gram infusion for up to 24 hours after childbirth or after the last seizure. In case of recurrence of seizures, a second bolus of magnesium sulfate may be administered, increasing the infusion up to 2 grams/hour.

Table 4. Regimen for the use of magnesium sulfate

<table>
<thead>
<tr>
<th>Presentations</th>
</tr>
</thead>
<tbody>
<tr>
<td>10-ml vials containing 5g at 50%, 2 g at 20% or 1g at 10%.</td>
</tr>
</tbody>
</table>

**Loading plan**: Intravenous use of 4g of 20% magnesium sulfate in 20 minutes in 150 cc isotonic saline solution.

**Maintenance plan**: Intravenous use of 10 vials at 10% in 400 cc 5% isotonic saline solution (2 to 3 grams /hour) in micro drips at 15 micro drips/minute or intramuscular, deep injection of 5 grams at 50% every 4 hours.

**Controls**: urinary output, mother’s osteotendinous reflexes and respiratory rate.

**Magnesium sulfate poisoning**: manifested through the progressive loss of the patellar reflex, respiratory rate < 12 rpm and/or urinary output < 30 ml/hour.

**Management of poisoning:**

*In non ventilated patients:* Discontinue magnesium sulfate.

Manual or mechanical ventilation.

Slow intravenous administration of 1 gram 10% Calcium gluconate.

*In patients under assisted ventilation:* Discontinuation of therapy or administration of calcium gluconate is not required.
Table 5. Regimen for the management of severe pre-eclampsia in pregnancy or within 4 weeks of childbirth

1. Ensure insertion of two venous lines (16 or 18F), for the infusion of a total volume of fluids at a 1 cc/Kg/h rate (including the drip of magnesium sulfate).

2. Start combination of intravenous vasodilator (labetalol or hydralazine), plus an oral anti-hypertensive agent (nifedipine) down to a 90 mmHg DAP if pregnancy continues (Table 3).

   - Take testing samples based on laboratory availability: blood count including platelet count, liver function tests (LDH, transaminases, AST and ALT) and serum creatinine levels.

4. Refer to high complexity center in ambulance, accompanied by doctor, nurse or medic to conduct the following:
   - Measurement of blood pressure every 5 to 10 minutes during transfer.
   - New dose of nifedipine or labetalol or hydralazine if SAP is ≥ 160 mmHg and/or DAP ≥ 110 mmHg.
   - 2 g bolus of 20% magnesium sulfate in 20 minutes in 150 cc isotonic saline solution, if there is a new seizure.

2. Eclampsia

Eclampsia is the presence of seizures and/or comma in a woman suffering from pre-eclampsia. Symptoms may start before, during or after childbirth, until the 4th week postpartum.22-24

Premonitory symptoms that anticipate eclampsia include occipital or frontal headache, blurry vision, photophobia, epigastric pain or pain in the right upper abdominal quadrant, and changes in the consciousness status.

Therapy

Therapy is based on resuscitation measures, termination of pregnancy, management of seizures and management of hypertension (Table 6).
## Table 6. Therapy of eclampsia

<table>
<thead>
<tr>
<th>Therapy</th>
<th>Description</th>
</tr>
</thead>
</table>
| **Basic and advanced resuscitation** | **Cardio-respiratory support** is the priority  
Insert a Mayo tube or the like, to prevent tongue bite and to keep a free airway and ensure ventilation.  
**Oxygen therapy** (5 Liters/minute) to maintain saturation over 95%, even after the patient has been stabilized.  
**Referral** to high complexity care center to allow for continuous monitoring of blood pressure, oxygen saturation, urinary output and fluid hourly balance.  
After childbirth, patients should be monitored for at least 48 hours; fluid redistribution during postpartum increases the risk for pulmonary oedema and high blood pressure. |
| **Obstetric management**         | **Termination of pregnancy** based on obstetric, maternal and fetal conditions. Patients should not be taken to emergency cesarean section if the mother's status is unstable; the first goal is to seek to recuperate the pregnant woman. If bradycardia persists 10 minutes after the seizure despite resuscitation efforts, pregnancy must be terminated. There are no contraindications for obstetric analgesia during delivery (epidural or combined technique) except for the presence of severe thrombocytopenia or coagulopathy with less than 75 thousand platelets. |
| **Prevention of seizures**       | **Intravenous magnesium sulfate.** (Table 4)  
- Being there no set cut-points to define the therapeutic range and its correlation with the clinical outcomes, there is no point for monitoring magnesium levels.  
- If seizures recur after receiving magnesium sulfate, administer a new 2-gram bolus in 3 to 5 minutes. Magnesium sulfate should be administered at least 48 hours after delivery and/or the last seizure. |
| **Anti-hypertensive management** | **Continuous invasive BP monitoring.** The objective for treating hypertension is to monitor the loss of cerebral auto-regulation without causing hypoperfusion of the uteroplacental system.  
Intravenous vasodilators and oral pressure lowering drugs:  
- Goal of therapy during pregnancy: SAP from 140 to 150 mmHg and DAP from 90 to 100 mmHg.  
- Goal of therapy in post-partum: lower than 140/90 mmHg (Table 3). |
Table 7. Regimen for the management of eclampsia during pregnancy or within the first 4 weeks after childbirth

1. Ensure the airway.

2. Start oxygen support at 5 L/min, aiming at an oxygen saturation over 95%.

3. Ensure 2 venous lines with 16 or 18 F catheters.

4. Start crystalloids at 1 cc/k/h (as the total volume administered, including the magnesium sulfate drip).

5. Place urinary bladder tube.

6. Start magnesium sulfate (Table 4).

7. Start anti-hypertensive therapy with a combination of intravenous vasodilator plus and oral antihypertensive drug, targeting a diastolic arterial pressure (DAP) of 90 mmHg before the end of pregnancy (Table 3).

8. Tests: blood count including platelet count, liver tests (LDH, Trasaminases, AST and ALT) and Creatinine.

9. Transfer to high complexity center in ambulance, accompanied by doctor, nurse or medic to conduct the following:
   1. Measurement of blood pressure every 5 to 10 minutes during transfer.
   2. New dose of nifedipine or labetalol or hydralazine if SAP is ≥ 160 mmHg and/or DAP ≥ 110 mmHg.
   3. 2 g bolus of 20% magnesium sulfate in 20 minutes in 150 cc isotonic saline solution, if there is a new seizure.
REFERENCES


II. Antepartum hemorrhage

1. Ectopic pregnancy

Ectopic pregnancy is defined as the implantation of pregnancy outside the endometrial cavity; it occurs in approximately 1% of pregnancies, and it increases maternal mortality by 10% to 15% in developed countries, when associated with fallopian tube rupture.1-3

Although there are risk factors that make a woman prone to ectopic pregnancy, over half the women presenting with this condition have no known risk factors (Table 1).4-6

Table 1. Risk factors for ectopic pregnancy 6,7,8

<table>
<thead>
<tr>
<th>Factor</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of tubal surgery</td>
<td>21 (9.3-47)</td>
</tr>
<tr>
<td>History of ectopic pregnancy</td>
<td>8.3 (6-11.5)</td>
</tr>
<tr>
<td>In-utero exposure to di-ethylestilbestrol</td>
<td>5.6 (2.4-13)</td>
</tr>
<tr>
<td>History of inflammatory pelvic disease</td>
<td>2.5 (2.1-3)</td>
</tr>
<tr>
<td>History of infertility</td>
<td>5.0 (1.1-28)</td>
</tr>
<tr>
<td>Fallopian tube ligation</td>
<td>9.3 (4.9-18)</td>
</tr>
<tr>
<td>Current use of IUCD</td>
<td>5 (1.1-28)</td>
</tr>
</tbody>
</table>

Clinical and laboratory diagnosis

Ectopic pregnancy always needs to be considered in women presenting with abdominal pain and positive pregnancy tests, regardless the presence of vaginal hemorrhage. The classical triad of amenorrhea, bleeding and abdominal pain actually occurs in fewer than 50% of the cases. Any patient with the confirmed or suspected diagnosis of ectopic pregnancy must be transferred to a center capable of providing comprehensive obstetric emergency care.
Tests that help elucidate the diagnosis include a quantitative pregnancy test, the beta fraction assay of the Human Chorionic Gonadotrophin (B-hCG), ultrasound, culdocentesis and laparoscopy.\textsuperscript{7-8}

Patients with ruptured ectopic pregnancy typically present with signs suggestive of shock, including hypotension, tachycardia and signs of peritoneal irritation. However, most patients present before the rupture has occurred, with non-specific manifestations; usual signs and symptoms include vaginal bleeding (typically intermittent and rarely exceeding the volume of normal menses), and colicky pelvic or abdominal pain.

**Beta Human Chorionic Gonadotrophin (B-hCG) serum levels**

The women that present with an ectopic pregnancy tend to show lower B-hCG levels than those with intra-uterine pregnancy.

In a normal pregnancy, B-hCG levels soar in the first trimester, duplicating approximately every 2 days. Measuring serum B-hCG levels serially every 48 hours may contribute to the diagnosis of ectopic pregnancy; serum B-hCG concentrations lower than 66\% are highly suggestive of this diagnosis.\textsuperscript{11-13}

**Transvaginal sonography**

Transvaginal ultrasound may identify a non cystic adnexal mass.\textsuperscript{8} A positive B-hCG and lack of intrauterine gestational sac should always lead to rule out ectopic pregnancy, even when 35\% of ectopic pregnancies may present without any adnexal abnormalities (Table 2).\textsuperscript{14,15}
Table 2. Ultrasound findings of ectopic pregnancy by location

<table>
<thead>
<tr>
<th>Type of pregnancy</th>
<th>Ultrasound finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tubal pregnancy</td>
<td>Adnexal mass, tubal ring sign, fire ring sign, pelvic hemorrhage.</td>
</tr>
<tr>
<td>Interstitial pregnancy</td>
<td>Eccentric gestational sac, gestational sac surrounded by thin myometrium (&lt; 5 mm), interstitial line sign.</td>
</tr>
<tr>
<td>Ovarian pregnancy</td>
<td>Serum B-hCG level &gt; 1000 mIU/1, normal fallopian tubes; gestational sac, chorionic villi or atypical cyst inside the ovary; normal levels of B-hCG after therapy.</td>
</tr>
<tr>
<td>Cervical pregnancy</td>
<td>Trophoblastic flow around the gestational sac inside the cervix, normal endometrial line, gestational sac with cardiac activity inside the cervix, “hourglass” shaped uterus, cardiac activity below the internal os of cervix.</td>
</tr>
<tr>
<td>Abdominal pregnancy</td>
<td>Absence of the intrauterine gestational sac, gestational sac located within the peritoneal cavity, abdominal or pelvic hemorrhage.</td>
</tr>
</tbody>
</table>

Therapy

The treatment of ectopic pregnancy may be watch & wait, surgical (laparotomy, laparoscopy) or medical.9, 10, 19

Expectant therapy

Applied only if:

- There is no evidence of tube rupture.
- There is minimum pain or bleeding.
- The patient is hospitalized.
- Serum B-hCG levels lower than 1000 mIU/ml.
- Adnexal mass smaller than 3 cm or undetectable.
- No heart beats.
**Surgical treatment**

Patients presenting with hemodynamic instability or signs of haemoperitoneum, bulky adnexal mass and/or high serum B-hCG levels, or embryo’s cardiac activity must be managed surgically at centers with the capacity required to solve hypovolemic shock.

In case of an abdominal ectopic pregnancy, there is indication of a surgical approach through laparotomy to remove the fetus. A few considerations need to be born in mind concerning the placenta: if it is imbedded in a non vital organ (omentum), the organ needs to be excised with the placenta in situ, for any attempt to extricate the placenta from the organ in question may lead to severe bleeding; when the placenta is implanted in the small bowel, the large bowel or another vital organ, the umbilical cord needs to be severed proximally to the placenta, leaving the latter behind in the organ, and administering methotrexate subsequently.

Interstitial pregnancy requires a surgical wedge excision of part of the myometrium, ipsilateral salpingectomy and sparing of the ovaries.

The ectopic pregnancy nested in an ovary requires surgery consisting of partial or total resection of the ovary involved.

**Medical Therapy**

The aim of the medical therapy with methotrexate (folic acid antagonist) is to preserve a functional tube and prevent the risks and costs of surgical procedures. It may be done with a single dose, variable doses or through direct injection on the implantation site.

**Treating ectopic pregnancy with methotrexate**

Indications:

- Adnexal mass < 3 cm.

- Wish to preserve future fertility.
• Stable or steadily increasing B-hCG levels after curettage, with a maximum peak under 15,000 mIU/mL.

• No active hemorrhage (hemodynamic stability).

• Complete laparoscopic visualization of the ectopic pregnancy.

• Normal liver enzymes and blood count.

• Selected cases of cervical and cornual pregnancies.

Contraindications:

• Liver dysfunction, SGOT twice the normal value.

• Renal disease, serum creatinin levels > 1.5 mg/dL.

• Active peptic ulcer.

• Blood dyscrasia, white blood cells <3,000, platelets <100,000.

• The patient may be easily lost from follow-up

**Protocol for methotrexate therapy in case of ectopic pregnancy**

**Table 3. Methotrexate therapy of ectopic pregnancy**

<table>
<thead>
<tr>
<th>Laboratory testing before therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Complete blood count.</td>
</tr>
<tr>
<td>• Blood typing.</td>
</tr>
<tr>
<td>• Liver and renal function tests.</td>
</tr>
<tr>
<td>• Measurement of serum B-hCG levels.</td>
</tr>
<tr>
<td>• Transvaginal ultrasound.</td>
</tr>
</tbody>
</table>
**Therapy Day 0**
- Methotrexate 1 mg/kg i.m. (successful in 87% of the cases).
- Injection of Anti D Gamma Globulin in non immunized Rh negative patients (300 mcg) Discontinue folinic acid supplements.

**Day 7**
- Measurement of serum B-hCG concentration.
- Transvaginal ultrasound.
- Inject second dose of methotrexate if the level of B-hCG drops under 25%.

**Weekly**
- Measurement of serum B-hCG concentration under <15 IU/l.
- Transvaginal ultrasound.

**Any time**
- Laparoscopy if the patient presents with severe abdominal pain or acute abdominal condition, or if the ultrasound confirms the presence of more than 100 ml of blood in the abdomen

**Adverse effects of methotrexate**
- Usually mild and self-limited.
- Stomatitis and conjunctivitis are the most common.
- Rare occurrence of pleuritis, dermatitis, alopecia, gastritis, enteritis, high concentrations of liver enzymes and bone marrow suppression.
- Approximately 30% of the patients have adverse effects with one single dose, and 40% with multiple doses
The variable dose scheme consists of administering 1 mg/kg of methotrexate intramuscular in alternate days, interspersed with 0.1 mg/kg of bail out leucovorin intramuscularly, until a consistent response can be seen in the reduction of B-hCG (15%) in 48 hours, or until completion of four doses (Methotrexate on days 1, 3, 5 and 7, and leucovorin on days 2, 4, 6 and 8). Literature reports 93% success rates in the patients treated; tube patency rates and fertility rates are similar to those observed with the conservative surgical therapy, and the subsequent ectopic pregnancy rate is low. Direct injection of high doses of methotrexate in the ectopic implantation site, either ultrasound-guided or via laparoscopy reduces the toxic effects but the success rates (76%) are lower than those achieved with the systemic administration of the drug.

REFERENCES


2. Abruptio Placentae

Abruptio placentae, placental abruption or premature detachment of the placenta is defined as the detachment or total or partial separation of a normally implanted placenta from its site of implantation before the birth of the fetus and after the 22th week of pregnancy. It may occur with bleeding between the membranes and the decidua through the cervix, or remain confined inside the uterus, with bleeding behind the placenta.\(^1\)

It occurs in approximately 1% of pregnancies, with a fetal mortality of 1 in 500 to 750 births and a perinatal mortality rate of 119 per 1000 births, especially because of prematurity.\(^2,3\)

It corresponds to 30% of the hemorrhages of the second half of gestation, associated with a 1% maternal mortality ratio. It is associated with, although not limited to, an increased incidence of disseminated intravascular coagulation, renal failure, need for transfusions and hysterectomy.\(^4\)

Predisposing factors

- History of abruptio placentae, with an 11% recurrence after one episode and over 20% after two episodes.\(^5\)

- Pregnancy-related hypertensive disorders, with an incidence ranging from 2.5% to 17.9%, greater in severe early pre-eclampsia and chronic hypertension.\(^6\)

- Older mothers.

- Multiparity.

- Premature delivery and premature rupture of membranes resulting from inflammation or sudden decompression of the uterus.\(^7\)
- Overdistension of the uterus due to multiple pregnancy or polyhydramnios.
- Vascular disease.
- Uterine anomalies or masses.
- In a dose-dependent relation, smoking is associated to decidual necrosis, chorionic villous hemorrhage and intervillous thrombosis.\(^8\)
- Alcohol use
- Use of cocaine and vasoconstricting drugs that impair the blood flow of the placenta and decidual integrity.
- Abdominal trauma or manipulation of uterus, such as external version of the head.\(^9\)
- Nutrition deficiencies (folate deficiency).\(^10\)
- Thrombophilias (hyperhomocysteinemia)\(^11\)
- Short umbilical cord.\(^12\)

**Classification**

Below is the classification of the grades of *abruptio placentae*:

- **Grade 0**: Asymptomatic. The diagnosis is retrospective and based on pathology.
- **Grade I**: Mild; it accounts for approximately 48% of the cases. The patient presents with mild or no vaginal bleeding (occult hemorrhage), mild tenderness of the uterus, normal heart rate and maternal blood pressure, not associated with coagulation disorders or changes in the fetal status.
- **Grade II**: Moderate; it accounts for 27% of all cases. Typically the onset is characterized by moderate or no vaginal bleeding
(occult hemorrhage), moderate to severe tenderness of the uterus, with potential tetanus contractions (hypertonic uterus), maternal tachycardia and orthostatic changes of blood pressure, fetal distress and hypofibrinogenemia (50 — 250 mg/ dl).

- Grade III: Intense; it accounts for 24% of all the cases. The patient presents with abundant or no vaginal bleeding (occult hemorrhage), very painful hypertonic uterus, maternal shock, hypofibrinogenemia (<150 mg/dl), coagulation disorder and fetal death.13, 14

**Clinical and laboratory diagnosis**

The clinical onset of *abruptio* varies broadly, ranging from an asymptomatic bleeding to severe life threatening bleeding that may result in maternal and perinatal death. The classical manifestation of the premature detachment of the placenta includes vaginal bleeding (usually dark), abdominal pain and uterus contractions.

The diagnosis of premature detachment of the placenta is clinical, and the symptoms include abdominal or pelvic pain (70%), vaginal bleeding (70%), contractions of uterus of unclear etiology (20%); signs include hypertony (35%), fetal distress (60%) and fetal death (15%).

In 20% of the cases, occult hemorrhage occurs inside the uterine cavity (retroplacental clot) with gradual (even complete) detachment of the placenta, frequently associated to severe complications. Approximately 10% of this form of *abruptio placentae* is associated with coagulopathy (disseminated intravascular coagulation). The external or visible hemorrhage is present in 80% of the cases. In these cases, bleeding occurs through the cervix; the detachment of the placenta may be complete or incomplete, and complications – although less frequent – tend to be serious.
The fetal heart rate patterns associated with *abruptio placentae* reveal life-threatening fetal distress.

Some acute cases may not show the typical ultrasound findings, and it is generally considered that in half the cases the ultrasound is not conclusive. However, when it suggests the presence of *abruptio*, the odds of confirming the diagnosis is extremely high. It is generally considered that its diagnostic sensitivity is 80%, with 92% specificity, 95% positive predictive value and a 69% negative predictive value.15 -18

**Treatment**

Management of *abruptio placentae* depends on the presentation of the clinical picture, gestational age and the degree of materno fetal impairment. The primary objective is to avoid maternal morbi-mortality (uterine atony, uterus of Couvelaire, hemorrhagic or hypovolemic shock, disseminated intravascular coagulation, tubular necrosis and Sheehan’s cortico renal syndrome) and fetal morbi-mortality (fetal hypoxia, anaemia, intrauterine growth restriction (IUGR), CNS disorders, fetal death), so it has to be tiered, and requires facilities where both blood and surgery are available.18

**Management regimen by levels of care**

Low-complexity centers should be ready to provide emergency care, including initial resuscitation of all the patients presenting with the suspicion of *abruptio placentae* until their timely transfer to a higher complexity center. That should include:

- Continuous haemodynamic monitoring of vital signs.
- Continuous supplementary high flow oxygen (pouchless mask at 12 - 15 l/min).
• Two good gauge 16 or 18 F venous lines to administer crystalloids (normal saline solution or Ringer lactate).
• Monitoring of the significance of vaginal bleeding.
• Monitoring of fetal heart rate.
• Treatment of hypovolemic shock, if necessary.

Hospital care depends on the gestational age and the severity of symptoms. This management includes:

• Continuous non-invasive hemodynamic monitoring.
• Supplemental oxygen.
• Immediate assessment of fetal wellness.
• Administration of intravenous fluids (crystalloids). Based on the patient's hemodynamic status or signs of hypovolemic shock, an aggressive resuscitation with crystalloids (1500-2000 cc bolus, followed by a 200-300 cc / h infusion) may be required to ensure adequate tissue perfusion.
• Stocks and availability of blood products (4 units of red blood cells, plasma, platelets and other blood products). Low complexity centers must be able to provide at least two volumes of packed red blood cells, preferably group O Rh negative.
• Immediate amniotomy to reduce the intrauterine pressure, in an attempt to avoid extravasation of blood into the myometrium, thus preventing thromboplastic substances from entering the circulation; these substances trigger the activation of the coagulation cascade, with the ensuing development of disseminated intravascular coagulation.
• Immediate caesarean section if the fetus is alive and delivery is not imminent, or if there are signs of maternal and / or fetal instability; consider starting an aggressive hemodynamic resuscitation and absolute availability of blood products.19,20
• Treatment of coagulopathy or disseminated intravascular coagulation, being the early diagnosis instrumental for the anticipated replacement of blood products. The treatment of the underlying cause is essential, terminating pregnancy and removing the fetus and the placenta. Preserving an effective circulation minimizes the negative effect of ischaemia.

• The use of heparin or anti-fibrinolitic agents is not indicated when there is disseminated intravascular coagulation induced by abruptio placentae. After childbirth, the process is usually solved rapidly, and it is rare for the overt coagulopathy to persist beyond the 12th hour. The platelet count usually goes back to normal levels by the second or third day post partum.21-23

REFERENCES


3. Placenta Previa

The term placenta previa refers to the placenta that either occludes or is close to the internal os of the cervix (IOC) and is implanted in the lower segment of the uterus after the 22nd week of pregnancy.

Placenta previa cases have traditionally been classified into 4 types:

- Complete placenta previa: the placenta fully covers the IOC.
- Partial placenta previa: the placenta covers the IOC only partially
- Marginal placenta previa: the placenta is close to the IOC, but without occluding it.
• Low insertion of the placenta: stretching along the uterus segment but not occluding the IOC.\textsuperscript{1-3}

Placenta previa gets complicated in approximately 0.3 to 0.5% of pregnancies, with 0.3% maternal mortality of the cases. In such cases, the perinatal mortality rate is increased 3 to 4 fold versus normal pregnancies.\textsuperscript{3,4}

The morbidity associated with placenta previa is described in Table 1.\textsuperscript{5,6}

Table 1. Maternal morbidity associated to placenta previa

<table>
<thead>
<tr>
<th>Condition</th>
<th>RR, 95 % CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antepartum hemorrhage</td>
<td>9.8 (8.9-10.8)</td>
</tr>
<tr>
<td>Need for hysterectomy</td>
<td>33.3 (18.2-60.9)</td>
</tr>
<tr>
<td>Intrapartum hemorrhage</td>
<td>2.5 (1.6-4)</td>
</tr>
<tr>
<td>Postpartum hemorrhage</td>
<td>1.9 (1.5—2.4)</td>
</tr>
<tr>
<td>Transfusion</td>
<td>10.1 (7.5-13.6)</td>
</tr>
<tr>
<td>Septicemia</td>
<td>5.5 (1.3-23.5)</td>
</tr>
<tr>
<td>Thrombophlebitis</td>
<td>4.9 (1.5-15.7)</td>
</tr>
</tbody>
</table>

Clinical and laboratory diagnosis

The classical clinical presentation of the placenta previa is painless bleeding occurring in the second half of pregnancy. Some patients may present painful bleeding, probably caused by the onset of uterine contractions.

Most patients with placenta previa are diagnosed with the ultrasound during the second trimester of pregnancy.
The transvaginal sonogram (TVS) is more precise than the transabdominal US, with 87.5% sensitivity, 98.8% specificity, 93.3% positive predictive value and a negative predictive value of 97.6%, making it the gold standard method for diagnosis.

Patients with the diagnosis of placenta previa at the end of pregnancy are more likely to present it still at the time of childbirth. Patients diagnosed at around the 20\textsuperscript{th} week of pregnancy should be reassessed to confirm the diagnosis.

**Expectant Therapy Management**

The high perinatal mortality rate in the cases of placenta previa related with preterm childbirth may be reduced through a watchful conservative management, and trying to push childbirth as close to the due date as possible. Watchful management is summarized in Table 2.

**Table 2. Watchful management of placenta previa**

<table>
<thead>
<tr>
<th>Management</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confirmation of diagnosis</td>
<td>Pelvic and/or transvaginal sonography plus Doppler in selected cases.</td>
</tr>
<tr>
<td>Uterine inhibition</td>
<td>Only when there are uterus contractions in preterm pregnancy</td>
</tr>
<tr>
<td>Pulmonary maturation inducing drugs</td>
<td>Gestational age between 24 and 34 weeks.\textsuperscript{8}</td>
</tr>
<tr>
<td>Termination of pregnancy</td>
<td>Elective cesarean section between weeks 36 and 37.</td>
</tr>
<tr>
<td></td>
<td>Cesarean section in totally or partially occlusive placenta previa.</td>
</tr>
</tbody>
</table>
Women that present with bleeding warrant a very careful exploration of the vagina.

As long as bleeding is moderate and there is no hemodynamic instability, the aim is to keep pregnancy going, especially with gestations under 32 weeks.

Among the strategies recommended for the management of placenta previa, there is no evidence to support cerclaje.⁹

The cesarean section must be elective; (whenever possible) it should be avoided before term, since in the emergency, this procedure entails a 50% increase in the risk of bleeding. An anterior placenta increases the risk of bleeding during the cesarean section because the incision has the potential of cutting through the placenta, posing a more challenging technical difficulty for the fetal extraction.

When the placenta is marginal, if the distance from the ICO to the placenta is greater than 2 cm as shown in the last ultrasound assessment conducted between weeks 35 and 36 of pregnancy, it is safe to allow for a vaginal delivery to take place.¹⁰

Patients with a massive bleeding or uterine activity need to be admitted and placed 2 16-18 venous accesses, with timely sampling of a complete blood count, blood typing and a stock of blood products.

**Management algorithm by levels of care**

Pre-hospital care attendants and low complexity centers should be ready to provide emergency care including initial resuscitation of all the patients with the suspicion of *abruptio placentae* until their timely transfer to a higher complexity center that needs to be able to offer:

- Continuous haemodynamic monitoring of vital signs.
- Continuous supplementary high flow oxygen (mask with breathing bag at 12 - 15 l/min).
- Two good gauge 16 or 18F venous lines to administer crystalloids (normal saline solution or Ringer lactate).
- Monitoring of the amount of vaginal bleeding.
- Monitoring of fetal heart rate.
- Treatment of hypovolemic shock, if necessary.

Hospital care depends on the gestational age and the severity of symptoms. This management includes:

- Continuous non-invasive hemodynamic monitoring.
- Administration of supplementary oxygen.
- Immediate assessment of fetal wellness.
- Administration of intravenous fluids (crystalloids). Based on the patient's hemodynamic status or signs of hypovolemic shock, an aggressive resuscitation with crystalloids (1500-2000 cc bolus, followed by a 200-300 cc / h infusion) may be required to ensure tissue perfusion.
- Stocks and availability of blood products (4 units of red blood cells, plasma, platelets and other blood products). Low complexity centers must be able to provide at least two volumes of packed red blood cells, preferably group O Rh negative.
- Determine whether the patient meets the clinical criteria for an expectant management in cases of placenta previa with no massive bleeding.
- Immediate cesarean section if there are signs of maternal and/or fetal instability; consider starting an aggressive hemodynamic resuscitation and ensure absolute availability of blood products.
- Treatment of consumption coagulopathy; an early diagnosis is very helpful for the anticipated replacement of blood products.
Algorithm for the management of antepartum hemorrhage by levels

**Antepartum hemorrhage occurring during the first and second trimester of pregnancy**

- Pregnancy less than 14 weeks with vaginal bleeding and/or pelvic pain
  - **Stable vital signs**
    - Refer for ultrasound, qualitative B-hCG
  - **Unstable vital signs**
    - Assessment of blood pressure, heart and respiratory rate, capillary filling, conscience status, fetal viability with fetal cardiac assessment. Evaluation of the uterus tone and uterine con-tractions (rule out uterine hypertony).
    - **Do not perform digital examination of the vagina**
    - Speculoscopy: evaluate source of bleeding — characteristics of bleeding (scarce, moderate or abundant) — color of bleeding (bright red or chocolate color).
- Pregnancy over 14 weeks with vaginal bleeding
  - **Abundant, life-threatening bleeding**
    - Refer to level IV in ambulance staffed with doctor, nurse or medic capable of the procedures below:
      1. Placing two venous lines with 16 or 18 gauge catheters to start the patient on crystalloids in bolus to maintain systolic arterial pressure $< 90$ mmHg; take blood sample for hemoglobin, hematocrite, platelet count, blood typing, coagulation time and serum creatinine levels.
      2. Early evaluation of the need for emergency transfusion 2 U of red blood cells O (-).
      3. Resuscitation with a massive transfusion pack in the presence of severe hypovolemic shock, with red blood cells: 6 units (including 2 units O-), plasma: 6 units, 1 platelets apheresis.
      4. Insert bladder catheter to evaluate the urinary output.
      5. Monitor maternal vital signs during transfer, and when indicated, check fetal heart rate every 15 minutes.
      6. Refer to a center with capability for laparotomy/ laparoscopy (first trimester), curettage (first trimester) and emergency cesarean section (second trimester).
REFERENCES


III. Postpartum hemorrhage

Postpartum hemorrhage (PPH) is defined as the loss of more than 500 cc of blood after vaginal delivery or greater than 1,000 cc following a cesarean section.

Massive PPH is defined as bleeding over 1,000 cc within the first 24 hours of puerperium. The definition of massive PPH was proposed in an attempt to establish more objective measures, such as a drop greater than or equal to 10% of the hematocrit, or the presence of bleeding resulting in hemodynamic instability.

The prevalence of 500-cc PPHs ranges from 6 to 10%, and for PPHs over 1,000 the prevalence ranges from 2 to 3%.

The main risk factors associated to PPH are summarized in Table 1.
### Table 1. Risk factors for PPH

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Adjusted OR (multivariate analysis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placenta previa</td>
<td>13.1</td>
</tr>
<tr>
<td>Abruptio placentae</td>
<td>12.6</td>
</tr>
<tr>
<td>Emergency cesarean section</td>
<td>3.6</td>
</tr>
<tr>
<td>Von Willebrand’s Disease</td>
<td>3.3</td>
</tr>
<tr>
<td>Chorioamnionitis</td>
<td>2.5</td>
</tr>
<tr>
<td>Elective cesarean section</td>
<td>2.5</td>
</tr>
<tr>
<td>Twin pregnancy</td>
<td>2.3</td>
</tr>
<tr>
<td>Fetal weight &gt; 4500 grams</td>
<td>1.9</td>
</tr>
<tr>
<td>Polyhydramnios</td>
<td>1.9</td>
</tr>
<tr>
<td>Multiparity</td>
<td>1.9</td>
</tr>
<tr>
<td>HELLP Syndrome</td>
<td>1.9</td>
</tr>
<tr>
<td>Instrument-assisted delivery</td>
<td>1.9</td>
</tr>
<tr>
<td>Induction of labor</td>
<td>1.6</td>
</tr>
<tr>
<td>Obesity</td>
<td>1.6</td>
</tr>
<tr>
<td>History of PPH</td>
<td>1.6</td>
</tr>
<tr>
<td>Previous cesarean section</td>
<td>1.5</td>
</tr>
<tr>
<td>Prolonged labor</td>
<td>1.1</td>
</tr>
<tr>
<td>Older than 40 y.o.</td>
<td>1.4</td>
</tr>
</tbody>
</table>
In 60% of the **PPH** cases, there are no identifiable risk factors.

PPH is the leading cause of direct maternal mortality worldwide, accounting for 25 to 30% of maternal deaths. The time from the onset of PPH to the time of death is typically short.\(^6,7\)

PPH accounts for 22 to 55% of the cases of extreme maternal morbidity, with increased rates of hysterectomy, renal failure, sepsis and admissions to the ICU.

The contraction of the myometrium on the coiled arteries, causing their lumen to obliterate is the final haemostatic effect after childbirth; this in turn triggers the coagulation process.\(^8\)

Uncontrolled active bleeding leads to secondary hypovolemic shock when the blood loss reaches 40%. When this happens, the tissue oxygen demands are not met by the oxygen supplied; as the depth and time of the hypovolemic shock worsen, they cause hypothermia, coagulopathy and metabolic acidosis, commonly referred to as the death triad.\(^9\)

**Classification**

Postpartum hemorrhages may be early or late. Early hemorrhage is defined as the hemorrhage that occurs during the first 24 hours of the postpartum period, generally within the first 2 hours, being the most frequent and severe; the causes are uterine atony, retention of placental debris, placental abnormalities and lacerations of the genital tract.

Late hemorrhage is that occurring from hour 24 to the 6th week after childbirth; its frequency ranges from 5 to 10% of childbirths; the most common causes being retention of placental debris, infections, lacerations and trophoblastic disease.

Table 2 categorizes the postpartum hemorrhages based on their etiology, applying the “4 t’s” mnemonics:
**Table 2. Classification of PPH by risk factors and etiology**

<table>
<thead>
<tr>
<th>Etiology and frequency</th>
<th>Etiology</th>
<th>Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tone 70%</strong></td>
<td>Uterine overdistension</td>
<td>Multiple pregnancy.</td>
</tr>
<tr>
<td></td>
<td>Protracted/ precipitate delivery (E.g.: induced).</td>
<td>Macrosomia.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Polyhydramnios.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Grand multiparaa.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe hydrocephalus.</td>
</tr>
<tr>
<td>Uterine Muscular Fatigue.</td>
<td></td>
<td>Protracted labor.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chorioamnionitis.</td>
</tr>
<tr>
<td><strong>Trauma 20%</strong></td>
<td>Vaginal / cervix/perineal tears.</td>
<td>Instrumental delivery.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Episiotomy.</td>
</tr>
<tr>
<td></td>
<td>Extension of the cesarean tear.</td>
<td>Fetal mal position.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Harsh manipulation during fetal extraction.</td>
</tr>
<tr>
<td>Uterine rupture.</td>
<td></td>
<td>Earlier uterine surgery.</td>
</tr>
<tr>
<td>Uterine reversion.</td>
<td></td>
<td>Excessive pulling of the cord.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Grand multipara.</td>
</tr>
<tr>
<td><strong>Tissues 9%</strong></td>
<td>Retention of debris.</td>
<td>Placenta or membranes.</td>
</tr>
<tr>
<td>Placental anomalies.</td>
<td></td>
<td>Location: Placenta previa.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Invasion: accreta, percreta, increta.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Congenital: Bicornuate uterus.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acquired: Previous surgery, leiomyoma.</td>
</tr>
<tr>
<td><strong>Thrombi 1%</strong></td>
<td>Congenital coagulopathies.</td>
<td>Haemophilia.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Von Willebrand's Disease.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypofibrinogenemia.</td>
</tr>
<tr>
<td>Acquired coagulopathies pregnancy.</td>
<td></td>
<td>Hypertension.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fetal death.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Liver disease.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HELLP syndrome.</td>
</tr>
<tr>
<td><strong>diopathic Thrombocytopaenic Purpura.</strong></td>
<td>Disseminated Intravascular Coagulopathy</td>
<td>Intrauterine fetal death.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Toxemia.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Abruptio placentae.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Amniotic embolism.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sepsis.</td>
</tr>
<tr>
<td>Dilutional coagulopathy.</td>
<td></td>
<td>Massive transfusions.</td>
</tr>
<tr>
<td>Anticoagulation.</td>
<td></td>
<td>History of DVT and PTE.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Use of Aspirin, Heparine.</td>
</tr>
</tbody>
</table>
Clinical and laboratory diagnosis

There is a trend to underestimate the blood loss in pregnant women. The signs, symptoms and their relation with the degree of blood loss and hypovolemic shock should be listed and exhibited at the facilities where childbirth care is provided, so as to guide the management strategies. Initial assessment in PPH, based on the losses estimated: (Table 3).

Table 3. Diagnosis and classification of the degrees of hypovolemic shock

<table>
<thead>
<tr>
<th>Volume loss in % and ml (50-70 kg woman)</th>
<th>Sensorium</th>
<th>Perfusion</th>
<th>Pulse</th>
<th>Systolic blood pressure (mm/Hg)</th>
<th>Degree of shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>10-15%</td>
<td>Normal</td>
<td>Normal</td>
<td>60-90</td>
<td>&gt;90</td>
<td>Absent</td>
</tr>
<tr>
<td>500-1000 mL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16-25%</td>
<td>Normal and/or agitated</td>
<td>Paleness, coldness</td>
<td>91-100</td>
<td>80-90</td>
<td>Mild</td>
</tr>
<tr>
<td>1001-1500 mL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>26-35%</td>
<td>Agitated</td>
<td>Paleness, coldness, more sweating</td>
<td>101-120</td>
<td>70-79</td>
<td>Moderate</td>
</tr>
<tr>
<td>1501-2000 mL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;35%</td>
<td>Lethargic or unconscious</td>
<td>Paleness, coldness, more sweating and capillary filling &gt; 3 seconds</td>
<td>&gt;120</td>
<td>&lt;70</td>
<td>Severe</td>
</tr>
<tr>
<td>&gt;2000 mL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Therapy

Prevention of postpartum hemorrhage: active management of the third stage of delivery

Literature reviews have shown that the only effective maneuver for the prevention of postpartum hemorrhage is the active management of the third stage of labor, clearly supported under an A level of evidence. In PPH high-risk pregnant women, there is a 62% reduction of blood losses over 500 cc, and 67% of losses greater than 1000 cc; 66% reduction of the need for blood transfusion and 80% less need for administering oxytocin therapy.10-13

Oxytocin is considered the drug of choice in the active management of the third stage of labor. Although ergotamine formulations also reduce the risk of PPH, there is a significant increase of adverse effects; misoprostol also reduces the likelihood of PPH, and its use is ideal wherever oxytocin is not available. The World Health Organization (WHO) recommends that all the oral or injectable uterotonic drugs must be available for the prevention and management of PPH.13

The active management components of the third stage of labor have been slightly modified in the light of new evidence on their effects on newborns; to avoid any confusions we have redefined the term as perinatal management of the third stage of labor. (Table 4)
Table 4. Definition of the perinatal management of the third stage of labor

<table>
<thead>
<tr>
<th>Component</th>
<th>Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Administration of utero tonic agents</td>
<td>Administration of prophylactic oxytocics:</td>
</tr>
<tr>
<td>and cord clamping</td>
<td>10 U of oxytocin or 0.2 mg of ergometrine (when oxytocin is not available) IM; after the umbilical cord has ceased to beat (1 to 3 minutes).</td>
</tr>
<tr>
<td>Controlled pulling of the cord</td>
<td>There is no evidence that this procedure in itself reduces the risk of PPH; rather, its routine use poses a greater risk of neonatal anemia (if it is associated with early clamping - before one minute) and uterine inversion and/or rupture of the cord.</td>
</tr>
<tr>
<td>Uterine massage</td>
<td>Every 15 minutes during the first two hours of puerperium.</td>
</tr>
</tbody>
</table>

Algorithm for the initial approach of PPH

The priorities in the management of postpartum hemorrhage are the control of bleeding and the replacement of the circulatory volume to improve the oxygen delivery capacity, and to maintain an adequate perfusion. Below is the algorithm proposed for the approach of pregnant women with PPH (Table 5).14,15
### Table 5. Initial approach in PPH

<table>
<thead>
<tr>
<th>Actions</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Call</td>
<td>Obstetrics team, ICU, anaesthesiology and nurses.</td>
</tr>
<tr>
<td>Warn</td>
<td>Blood bank and operation room.</td>
</tr>
<tr>
<td>Transfer</td>
<td>If the patient is at a level of care that lacks the capacity to provide the care required for the case in question.</td>
</tr>
<tr>
<td>Venous lines</td>
<td>Insert 2 16-18 F peripheral venous lines percutaneously. The presence of hemodynamic instability demands for a rapid transfusion system with the capability to heat fluids, and availability of transfusion products; in refractory hypotension administer vasoactive agents to attain a mean blood pressure over 65 mmHg.</td>
</tr>
<tr>
<td>100% O₂</td>
<td>10-12 litres per minute via mask.</td>
</tr>
<tr>
<td>Trendelenburg</td>
<td>Raise the lower limbs at 30 degrees.</td>
</tr>
<tr>
<td>Samples</td>
<td>Blood typing, match testing, blood count, coagulation testing: PT, PTT, fibrinogen, serum creatinine and electrolytes.</td>
</tr>
<tr>
<td>Use of compressive suits</td>
<td>Especially indicated when transferring patients to higher complexity centers.</td>
</tr>
<tr>
<td>Monitoring:</td>
<td>Heart rate, blood pressure, pulse oxymetry and ECG.</td>
</tr>
<tr>
<td>Vesical catheter</td>
<td>Hourly monitoring of the urinary output.</td>
</tr>
<tr>
<td>Central venous access</td>
<td>Indication of CVP monitoring.</td>
</tr>
</tbody>
</table>
### Table 5. (Continued)

<table>
<thead>
<tr>
<th>Actions</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluids</td>
<td>Rapid infusions of crystalloids (Normal saline solution or Ringer lactate) 1 to 2 liters, followed by 300 to 500 cc boluses, based on the clinical response.</td>
</tr>
</tbody>
</table>
| Blood              | Packed red blood cells to restore the oxygen delivery capacity. The massive transfusion protocol includes 6 units of red blood cells (2 O negative units), 6 units of plasma and one platelet aphaeresis (5 to 8 units of platelets); all blood products should be readily available for usage in not more than 15 minutes after the onset of bleeding.  

In patients presenting with massive bleeding, hemodynamic instability and losses greater than 1,500 cc, start a transfusion with 2 O-negative red blood cells without prior matching tests, followed by a red blood cell transfusion with matching tests, plasma and platelets, based on the clinical presentation, and later based on the results of laboratory tests.  

In patients presenting with losses lower than 1,500 cc and that show no signs of hemodynamic instability, transfusion with typing tests to check blood type match; however, the treating team must be clearly aware that this decision should not take longer than 30 to 60 minutes.  

If necessary, transfuse plasma, platelets and/or specific coagulation factors to recover hemostasis. Initially, the transfusion of plasma depends on the clinical presence of coagulopathy, and later it is adjusted based on the results of the coagulation tests.  

Laboratory tests should guide transfusion replacement therapy during the maintenance phase of resuscitation. The base deficit measurement indicates the severity of the hypovolemic shock. An alkaline deficit greater than -6 reveals that the patient’s status is severely compromised; regular samplings are then required to establish the effectiveness of any intervention undertaken. Other perfusion parameters used to guide resuscitation are serum lactate levels and central venous saturation. |

(Continues)
### Table 5. (Continued)

<table>
<thead>
<tr>
<th>Actions</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evacuation of debris</td>
<td>Of the placenta and/or membranes.</td>
</tr>
<tr>
<td>Suture of tears</td>
<td>Of the vagina, cervix and/or perineal area, under analgesia, for the continuous stitch suture using resorbable materials. Check the integrity of the anal sphincter and the urethra. In case of vaginal bursting, with heavy and uncontrollable bleeding, vaginal packing with vaginal gauzes (soaked in Vaseline or on a latex glove) is indicated the first 24 to 48 hours, after which the gauzes may be removed.</td>
</tr>
</tbody>
</table>
| Management of uterine atony | **Massage of the uterine fundus**  
**Bimanual compression of the uterus:**  
Use sterile gloves, place the fist of one hand in the vagina, pressing the anterior aspect of the uterus, while the other hand compresses the posterior aspect through the abdomen. Evidence shows a reduced blood loss at 30 and 60 minutes.  
**Medications:**  
- Oxytocin 10 IU in bolus; continue with 20 units in 1000 cc of a saline solution, at a 60 drops/minute rate.  
- Methylergonovine: 1 0.2 mg vial IM or IV; the second dose 20 minutes later, and then 0.2 milligrams every 2 to 3 hours, to a total of 5 doses.  
- Misoprostol tablets x 200 mcg 4 intra rectal tablets.  
**Uterine Tamponage:** either through packing with gauze soaked in Vaseline or using the Bakri SOS catheter specifically designed for the therapy of PPH. If that kind of catheter is not available, others may be used, such as the Sengstaken Blakemore esophageal tube, the Foley tube with 60-80 ml or the urological hydrostatic tube; the results of the latter are not as successful as the former. They are primarily used for transfers or as a diagnostic test, but they can be left up to 48 hours, together with the infusion of oxytocics and antimicrobial therapy. |

(Continues)
Table 5. (Continued)

<table>
<thead>
<tr>
<th>Actions</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Laparotomy</strong></td>
<td><strong>Suture technique</strong> designed for hemostasis of the uterus (B-Lynch suture): Indicated in patients with PPH secondary to atony of the uterus; intended to achieve a vertical compression on the uterus vascular system through ligation (resorbable polylactin or nr. 2 polyglycolic acid) on the anterior and posterior aspect of the uterus. Haemostatic sutures may be performed after hysterectomy, in an attempt to define whether the uterine cavity is clean, and to assure a more successful therapy. The first stitch should be 3 cm below the rim of the hysterotomy and 3 cm away from the lateral edge of the uterus, exiting 3 cm above the hysterotomy and 4 cm away from the edge of the uterus. The suture goes upward, with the posterior stitch penetrating the cavity at the same level as the hysterotomy, about 4 cm away from each side of the edge of the uterus, at the origin of the broad ligament. The suture must be at a certain tension, compressing the uterus; it should be left about 4 cm away from the horn, to prevent it from sliding laterally.</td>
</tr>
<tr>
<td><strong>Consider more advanced management</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Consider other medical options</strong></td>
<td>Tranexamic acid: not indicated routinely; the dosage used is 1 gram every 4 hours until reaching a cumulative 3-gram dosage. Activated Factor VII: adjuvant to medical and surgical treatment and to resuscitation. The suggested dose is 90 micrograms per kilo, and it may be repeated if there is no clinical response after 10 to 15 minutes; it is extremely costly.</td>
</tr>
<tr>
<td><strong>Embolization</strong></td>
<td>Arterial selective embolization of the hypogastric or perineal arteries (pudendal): this is a fertility-sparing procedure. The effectiveness rate reported is over 90%, achieving resumption of regular menstrual cycles in almost 100%. The presence of severe shock, coagulopathy and hypoperfusion preclude its use.</td>
</tr>
</tbody>
</table>

**Suture technique** designed for hemostasis of the uterus (B-Lynch suture): Indicated in patients with PPH secondary to atony of the uterus; intended to achieve a vertical compression on the uterus vascular system through ligation (resorbable polylactin or nr. 2 polyglycolic acid) on the anterior and posterior aspect of the uterus. Haemostatic sutures may be performed after hysterectomy, in an attempt to define whether the uterine cavity is clean, and to assure a more successful therapy. The first stitch should be 3 cm below the rim of the hysterotomy and 3 cm away from the lateral edge of the uterus, exiting 3 cm above the hysterotomy and 4 cm away from the edge of the uterus. The suture goes upward, with the posterior stitch penetrating the cavity at the same level as the hysterotomy, about 4 cm away from each side of the edge of the uterus, at the origin of the broad ligament. The suture must be at a certain tension, compressing the uterus; it should be left about 4 cm away from the horn, to prevent it from sliding laterally.
Figure 1. Management scheme that conjugates resuscitation and medical and surgical management of PPH in terms of average times.

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30. Burtelow M, Riley E, Druzin M, Fontaine M, Viele M, Goodnough LT. How we treat: management of life-threatening primary postpartum hemorrhage with a standardized massive transfusion protocol. Transfusion 2007; 47:1564-


IV. Severe obstetric infections

1. Septic Abortion

Abortion is defined as the expelling or extraction from the mother’s womb of a conception product weighing ≤ 500 grams, or whose gestation age is lower than 22 weeks.

Abortion is suspected when a woman in child-bearing age presents with symptoms such as: amenorrhea with metrorrhagia with clots and/or products of conception occurring in the first half of pregnancy, and possibly associated with colicky pain. Enlarged uterus, painful at palpation and potential cervix changes.

Septic abortion is the infection of the uterus and/or the adnexa that presents after a spontaneous or induced abortion. Most septic abortions derive from “unsafe abortion” practices; consequently, the World Health Organization defines it as “a procedure to terminate an unwanted pregnancy by people that lack the necessary skills, or in an environment that fails to meet the minimal standards, or both”. The term “unsafe” should not be construed as “illegal” or “clandestine”.

In septic abortions the infection is initially limited to the uterus; however, it progresses rapidly to more serious forms as it extends to neighboring organs or through the bloodstream.

Clinical features of the septic abortion

- Temperature over 37.5 °C; it may present with malaise, asthenia, adynamia, chills and myalgias.
- Foul-smelling metrorrhagia and/or foul-smelling products of conception (clinical expression of the endometritis).
- Tender and softened uterus at palpation (clinical expression of myometritis).
- Pain upon lateral mobilization of cervix (clinical expression of parametritis).
- The cervix is usually partially open and there may be evidence of lacerations, foreign bodies or products of conception. Neighboring organ (bladder and bowel) damage needs to be ruled out.

The dissemination of the infection leads to more severe cases that typically add:
- Pain on palpation of the adnexal regions (salpingitis).
- Para-uterine masses or collections (tubo-ovarian abscesses).
- Pain on palpation of the lower hemi-abdomen (peritonitis).

**Laboratory assessment**

- Complementary tests:
  - ABO and Rh blood group typing.
  - Blood count: assess the degree of anaemia. An increased white blood cell count (over 14,000) is to be expected. Leukopenia is a severity indicator in the framework of sepsis. Coagulations changes may lead to low platelet counts.
  - ESR and Reactive Protein C.
  - Azotemia, serum creatinine levels, uricemia, ionogram.
  - Liver function and enzymes tests.
Complete blood coagulation testing (Prothrombin Time, Partial Thromboplastin Time, Thrombin Time, Fibrinogen and Fibrinogen Degradation Products (FDPs)).

Blood sugar.

Blood cultures. For aerobic and anaerobic organisms.

Blood gases to assess saturation and changes in the acid-base balance.

Urine test and urinary culture.

**Therapy of septic abortion**

Rest in dorsal recumbent position, semisitting.

Discontinue the oral route, any persisting products of conception require curettage evacuation (aspiration or instrumental).

Vital parameters monitoring:

- Neurological status.
- Pulse.
- Blood pressure.
- Respiratory rate.
- Color of skin and mucosas.
- Peripheral perfusion (capillary filling, Central or peripheral cyanosis, peripheral coldness).
- Urine output. Insert bladder tube connected to collecting pouch for control.

Insert a large caliber peripheral venous line (catheter 16 to 18F). Administration of crystalloids: Ringer — Lactate or saline at 1 liter every 4 hours. Fluid replenishment will be assessed, trying to preserve blood pressure, peripheral perfusion and urinary output over 30 ml/h.\(^{3-9}\)
Contact the referral hospital immediately to coordinate the patient’s transfer and reception.

Transfer should be done in ambulances specialized in the transportation of critically ill patients, and staffed with doctor, obstetrician or nurses specifically trained in the management of these complications.

**Pharmacological Measures**

- **Analgesia**: Ketoprofene 100 mgr IV every 8 to 12 hrs. In case of intense pain: opiate derivatives (tramadol 50 to 100 mg IV every 4 to 6 hours, not exceeding 400 mg/day, demerol 50 to 150 mg IV every 3 to 4 hours).

- **Antimicrobial therapy.** Start right away. The use of two antibiotics in association, and a prolonged therapy, that should never be shorter than 7 to 10 days. The recommendation is to count with antibiotic coverage at least 1 hour before the curettage.
  - Ampicillin 1 gr i/v every 6 hrs or Cefazolin 1 gr i/v every 6 hrs, plus Metronidazole 500 mg i/v every 8 hrs.
  - Clindamycin 600 mg i/v every 6 hours or Metronidazole 500 mg i/v every 8 hours, plus Gentamycin 80 mg i/v every 8 hrs or Amikacin 1 g IV every day. Its use should be avoided in patients with renal impairment.
    - Ampicillin/sulbactam 1.5 a 3.0 g i/v every 6 hours, plus Clindamycin 600 mg i/v every 6 hours.
    - Ceftriaxone 1 gram i/v every 12 hours plus Metronidazole 500 mg i/v every 8 hours.

Intravenous antibiotic therapy should be maintained for 48 hours after the last spike of fever; therapy may be continued per os to complete 7 to 10 days.

- **Antipyretic drugs**: If temperature exceeds 37.5°C, administer: dipyrone 1 gr intravenously every 8 hours.
Obstetric management

- Eradicate the infectious focus, products of conception and/or potential foreign bodies.

  - **Infection confined to the uterus**: evacuation of uterus to remove products of conception. The recommendation is to start the patient on antibiotics and proceed to evacuation within 6 hours of admission. In abortions with a gestational age:

    - < 12 weeks may be handled with manual aspiration, instrumental vacuum aspiration or instrumental curettage.

    - 12 weeks require expelling the debris using prostaglandins or oxytocin, followed by instrumental curettage.

    The material obtained should be sent to pathology and culture. The use of uteroconstrictors after the curettage is indicated to contract the uterus and reduce bleeding to a minimum. Patients may benefit of the use of oxytocin, methylergonovine, carbetocyn or prostaglandins.

  - **Infection beyond the uterus**: exploratory laparotomy is required; a midline incision will enable the surgeon to choose the appropriate procedure based on the intraoperative findings. Therapy may include hysterectomy with or without adnexectomy; tubal-ovarian abscess should be removed and the patients should be drained surgically. If the course is protracted despite the hysterectomy, consider taking the patient back to the operation room in search of pelvic abscesses or thrombosis of the ovarian pedicle, which, if confirmed, should be extracted.
2. Obstetric sepsis and septic shock

The diagnosis of sepsis must be suspected in the presence of 2 or more of the signs below:

- Temperature over 38°C or under 36°C.
- Pulse > 90 beats/minute.
- Respiratory rate over 20/min or the existence of a PaCO2 lower than 32 mmHg.
- White blood cell count over 12,000/mm3 or lower than 4000/mm3 (or more than 10% immature forms of white blood cells).
- Distant organ failure depends on the organs involved; there may be abnormalities of coagulation or liver, renal, respiratory or neurology function.

The presence of blood hypotension (systolic pressure under 90 mmHg or a drop of 40 mmHg of its previous level), cyanosis, peripheral hypoperfusion, oliguria and consciousness status (agitation, obnubilation) should suggest the existence of high mortality septic shock.10,13

- Vital parameters controls:
  The same as those described for septic abortion.

Laboratory assessment

The same tests indicated in the case of septic abortion, plus:

- Electrocardiogram.
- Chest X-ray.
- Abdominal-pelvic ultrasound.
- Other imaging studies (Computerized Axial Tomography, Magnetic Resonance Imaging) or laboratory tests will be requested as needed, based on each case.
Management of sepsis

Admit the patient into an intensive care unit in a hospital with surgery facilities; the patient should be assessed by a multidisciplinary team that should identify and remove the septic source. This decision should not be delayed; delaying the hysterectomy in severely ill patients often ends up in an irreversible septic shock. Such cases require total hysterectomy, checking the ovarian pedicles to rule out the thrombosis of those vessels.

- Maintain an adequate oxygenation through an oxygen mask or nasal catheter. Some patients may require ventilatory assistance. Oxygen saturation levels must be monitored with pulse oxymetry whenever possible; O2 saturation should be kept over 92 - 94%. Oxygenation will depend on the patient’s ventilation, peripheral perfusion and degree of anaemia. In case of respiratory failure, oxygen with free flow mask or nasal tube at 6 lt/min. In cases of septic shock: orotracheal intubation and ventilatory assistance.

- Insert 2 large (16 or 18 F) peripheral venous lines to allow for administration of medication and fluids. If the patient’s condition worsens, a venous collapse might make the procedure more difficult, so having an adequate vascular access is paramount.

- Keep an adequate volemia replenishing with crystalloids: Ringer — Lactate or saline solutions, at a rate of 1 lt/ every 4 hours. Replenishment will be assessed trying to keep the systolic blood pressure over 90 mmHg and a normal peripheral perfusion, with urinary output higher than 30 ml/h. The need for colloids and administration of blood products will be determined subsequently. Any anaemia should be corrected as soon as possible, not hesitating to use blood transfusions.

- Central venous pressure measurement is indicated. In case of pulmonary oedema, the use of diuretics may be beneficial (Furosemide: 20 to 60 mg IV.).
Use Dopamine 3 - 10 µg/kg/min in continuous IV infusion as an inotropic agent to improve cardiac function; if the blood pressure fails to improve because of myocardial depression, administer Dobutamine at 2 - 20 µg/kg/min in continuous IV infusion; if the pressure still does not improve because of vasodilation, administer norepinephrine: 2 to 8 µg/kg/min in continuous IV infusion.3-9,14-15

Correct any abnormalities of the internal milieu, including, but not limited to acidosis, dysionia and hyperglycemia.

Start a combined, intense and broad-spectrum antibiotic plan based on the cause of sepsis; refer to pharmacological measures in septic abortion.

The use of anticoagulants should be considered in the case of coagulopathy. Start IV or SC high molecular weight heparin at 5000 IU, followed by dosages of 700 to 2000 IU per hour, or Enoxaparin 20 to 40 milligrams subcutaneously every 24 hours (in both cases the activated partial thromboplastin time (aPTT) will be monitored every 4 hours until its level reaches 1.5 to 2 fold its mean value; once that level is reached, the aPTT will be checked only once a day).

Replacement with blood, plasma, coagulation factors and platelets.

REFERENCES


V. Cardiorespiratory arrest in pregnancy

1. Introduction

The incidence of cardiac arrest during gestation is estimated in one case per 30,000 pregnancies.\(^1\) Mortality rates are higher than in non pregnant women.

2. Causes

Cardiorespiratory arrest in pregnancy may be due to direct obstetric causes (resulting from conditions typical of pregnancy), indirect obstetric causes or not obstetric (resulting from processes that occur in addition to pregnancy, but not related to it).

Chart 1 presents the key causes of cardiorespiratory arrest during pregnancy.

Chart 1

<table>
<thead>
<tr>
<th>Primary causes of cardiorespiratory arrest during pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obstetric hemorrhage.</td>
</tr>
<tr>
<td>Pre-eclampsia – eclampsia syndrome.</td>
</tr>
<tr>
<td>Amniotic fluid embolism syndrome.</td>
</tr>
<tr>
<td>Peripartum myocardial disease.</td>
</tr>
<tr>
<td>Hypermagnesemia.</td>
</tr>
<tr>
<td>Diabete.</td>
</tr>
<tr>
<td>Septic or anaphylactic shock.</td>
</tr>
<tr>
<td>Trauma.</td>
</tr>
<tr>
<td>Pulmonary embolism.</td>
</tr>
<tr>
<td>Anesthesia-related complications.</td>
</tr>
<tr>
<td>Heart diseases and vasculopathies.</td>
</tr>
<tr>
<td>Endocrinopathies.</td>
</tr>
<tr>
<td>Collagen diseases.</td>
</tr>
<tr>
<td>Malpractice (administration of drugs or surgical complications).</td>
</tr>
</tbody>
</table>
3. Management

Initial approach in case of occurrence of cardiorespiratory arrest in a pregnant woman.

Chart 2

<table>
<thead>
<tr>
<th>Management of cardiorespiratory arrest in pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Seek help</strong></td>
</tr>
<tr>
<td><strong>Position of patient</strong></td>
</tr>
<tr>
<td>• Place the patient bending laterally in a 15 to 30 degree angle, or in supine recumbent position on a rigid surface or on the floor.</td>
</tr>
<tr>
<td>• Shift the uterus laterally to release the pressure upon the inferior vena cava.</td>
</tr>
<tr>
<td><strong>Circulation</strong></td>
</tr>
<tr>
<td>• Initiate chest compressions with a standard ratio (15 compressions of the chest every 2 ventilations). Compress the chest 3 cm above the traditional sternal point (as a result of the changes in the position of the heart due to pregnancy).</td>
</tr>
<tr>
<td>• Insert 2 large venous lines. Do not use the veins of the lower limbs, since the venous return is affected by the compression exerted by the pregnant uterus.</td>
</tr>
<tr>
<td><strong>Airway</strong></td>
</tr>
<tr>
<td>• Hyper extended neck if there is no cervical trauma.</td>
</tr>
<tr>
<td>• Pull the jaw downward.</td>
</tr>
<tr>
<td>• Aspirate of secretions.</td>
</tr>
<tr>
<td>• Remove dentures and other elements from the mouth.</td>
</tr>
<tr>
<td>• Insert an oropharyngeal tube.</td>
</tr>
<tr>
<td><strong>Breathing</strong></td>
</tr>
<tr>
<td>• Start ventilating with intermittent positive pressure (mouth to mouth, mouth to nose or from mouth to the oropharyngeal tube).</td>
</tr>
<tr>
<td>• Auto inflatable balloon, when available, and a mask to administer 100% oxygen.</td>
</tr>
<tr>
<td>• Press the cricoid bone until the airway is protected with an adequate tracheal tube (reduces the risk of aspiration).</td>
</tr>
<tr>
<td>• Introduce an orotracheal tube; to do so, put a pillow under the patient’s neck to raise it, and to cause the neck to extend.</td>
</tr>
<tr>
<td>o Ensure an adequate pre-oxygenation (with mask, for 30-60 seconds, with 100 % oxygen at a 12 lt/min flow rate). This permits the filling of the alveoli with 100% oxygen and helps the patient tolerate the apnea longer while she undergoes the laryngoscopy.</td>
</tr>
<tr>
<td>o The maneuver must be conducted by the most skilful staff possible. Then provide ventilatory support to produce effective oxygenation and ventilation.</td>
</tr>
<tr>
<td>o Check the symmetric movement of the chest and the pulmonary ventilation.</td>
</tr>
<tr>
<td>o Attach the tube and provide 100 % O₂.</td>
</tr>
</tbody>
</table>

(Continues)
Chart 2 (Continued)

**Defibrillation**
- Place one of the pads on the sternum and the other one on the left lateral aspect of the chest (give a 200-Joules discharge) and resume chest compressions immediately (if necessary, continue with two additional discharges, one of 300 Joules and the other one of 360 Joules).
- Defibrillation shocks do not transfer significant currents to the fetus. Disconnect the mother’s and fetus’s monitors before starting.

If possible, conduct and electronic monitoring of the vital parameters:
Heart rate, respiratory rate, ECG, pulse oxymetry and CO₂.

Evaluate the potential cause and act accordingly:
- Hypovolemia: immediate replacement.
- Hypoxia: ventilation.
- Changes of the internal milieu: correction of acidosis, K or Ca levels.
- Hyperthermia.
- Pneumothorax.
- Coronary event.
- Pulmonary embolism (thrombotic or amniotic fluid).
- Poisoning caused by:
  - Magnesium sulfate: administration of calcium gluconate.
  - Bupivacaine; proceed to electric cardioversion, use of bretylium or lidocaine.
  - drug overdose or poisoning (treat according to the specific agent).

Remember

Vaso active drugs (epinephrine, norepinephrine, dopamine, dobutamine, etc.) may affect the circulation of the uterus and placenta, but their usage at the recommended doses is justified during the cardiopulmonary resuscitation when the mother’s hemodynamic condition needs to be improved, to preserve her life.

The best way the fetus can be resuscitated is through an effective resuscitation of its mother.

Make sure you keep biosafety in mind during resuscitation.
Use mask and gloves.
Handle any sharp objects with care.
Obstetric management

Management depends on gestational age; cesarean section is indicated whenever the fetus is live and viable.

The cesarean section may not be indicated when the arrest is the result of a rapidly reversible cause (drug reaction, arrest induced by anaesthesia, bronchospasm-induced hypoxia); conversely, if the cause is irreversible, an emergency cesarean section should be indicated.

Time from the onset of the mother’s arrest and the cesarean section is of great importance both for the mother’s and the child’s prognosis; the best outcomes are obtained when the fetus is extracted within the first 5 minutes.

The cesarean section in patients under cardiorespiratory arrest does not require anaesthesia, and should prioritize a rapid extraction of the fetus. This implies using a midline incision approach, a quick opening of the wall, vertical incision of the uterus and rapid extraction of the fetus, and completed with immediate clamping of the cord.

REFERENCES
