Obstetric Disorders in the ICU

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Abstract

Pregnant and postpartum patients present a challenge to critical care physicians, as two patients in one have to be cared for and because specific obstetric disorders, not universally covered in formal critical care training, need to be managed. Pregnancy also alters physiologic norms, so that the critical care physician may either fail to recognize a value as abnormal in pregnancy or mistakenly identify as abnormal a value within the normal range for a pregnant woman. In this article, we will review the most frequent obstetric causes of admission of pregnant/postpartum patients to the intensive care unit (hypertensive disease of pregnancy, obstetric hemorrhage, and obstetric sepsis) along with their diagnostic criteria, clinical presentation, and recommended treatment. We will also cover some specific, although less frequent, obstetric disorders, such as acute fatty liver of pregnancy, peripartum cardiomyopathy, and amniotic fluid embolism. Our primary aim is to improve quality of care for these types of patients.

Keywords ► obstetrics ► critical care ► preeclampsia ► eclampsia ► postpartum hemorrhage ► septic abortion ► puerperal infection

Pregnant and postpartum patients represent a particular category of patients entering the intensive care unit (ICU). They account for a variable proportion of all ICU admissions, with lower numbers in developed countries (~2%) and higher numbers in developing countries (up to 16%).1,2

Most patients are admitted to the ICU postpartum. The most frequent causes of admission are due to obstetric diseases—those only occurring in pregnant/postpartum patients—as opposed to nonobstetric disorders, which can affect women who are not pregnant.1,3 Critical care physicians are faced with the challenge of treating two patients in one and of managing specific obstetric disorders, not universally covered in formal critical care training. In fact, substandard care is one of the risk factors associated with maternal mortality.3 We will review the main obstetric causes of admission of pregnant/postpartum patients to the ICU, focusing on prompt recognition of illness and correct management, with the aim of improving quality of care for these types of patients.

Hypertensive Disease of Pregnancy

Diagnosis, Pathophysiology, and Classification

Preeclampsia is one of several hypertensive disorders in pregnancy (HDP) and, unlike chronic hypertension, is pregnancy specific. It is classically defined by the development of hypertension plus proteinuria, after 20 weeks’ gestation, but may refer to hypertension without proteinuria if any of the following end-organ effects are present instead:

- thrombocytopenia (platelet count < 100,000/µL)
- abnormal liver function (transaminases elevated to at least twice the upper limit of normal)
- new-onset renal insufficiency (in pregnancy, bearing in mind that the increased glomerular filtration rate is typically associated with a much lower normal range for serum creatinine, renal insufficiency is defined as a serum creatinine > 1.1 mg/dL)
- pulmonary edema

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cerebral or visual disturbances of fresh onset: this pathognomonic manifestation would be eclampsia (generalized tonic-clonic seizure), but the category also includes altered mental status, stroke, severe headache, blindness, scotomata, or hyperreflexia with clonus.

In some classification schemes, the presence of uteroplacental insufficiency, usually manifested as fetal growth restriction, would also serve as a marker of end-organ dysfunction. However, this is unlikely to be relevant to the critical care specialist.

Hypertension is defined here as a systolic blood pressure (BP) ≥ 140 mm Hg and/or diastolic BP ≥ 90 mm Hg. Proteinuria is defined as ≥ 300 mg of protein excreted in 24 hours; a protein-to-creatinine ratio ≥ 0.3 (both measured in mg/dL) in a single voided specimen would also qualify. The use of a urine dipstick to establish or rule out proteinuria is discouraged.

Although the physiology of normal pregnancy manifests as a blunted response to endogenous or exogenous pressors, this adaptation fails in pregnancies which will go on to develop preeclampsia. Clinical manifestations are delayed until after mid-pregnancy, but the retained responsiveness to angiotensin and other pressor agents can be observed much earlier if specific testing is done. The mechanism underpinning preeclampsia is not fully understood, and because it is a disease both specific to pregnancy and peculiar to human pregnancy, it is difficult to study in animal models. It is believed to relate to the development and architecture of the placenta, specifically, to a failure of trophoblasts to adequately invade and remodel maternal spiral arterioles in the inner third of the myometrium. Additional theories have been advanced, including an imbalance of angiogenic and antiangiogenic factors, oxidative stress, endothelial injury, placental ischemia, and immune factors. Preventive strategies are not available, with the possible exception of low-dose aspirin beginning after 12 weeks' gestation for high-risk women and calcium supplementation in areas with low calcium intake. High-risk factors for preeclampsia include previous preeclampsia, preexisting hypertension, gestational diabetes, multiple pregnancy (twins, triplets, and above), renal disease, and autoimmune disorders such as systemic lupus erythematosus. Aspirin, at doses between 60 and 150 mg/day, confers a modest benefit in reducing the risk of developing preeclampsia in these groups. Additional clinical factors (nulliparity, obesity, family history of preeclampsia, black/African ancestry, age ≥ 35 years) identify women at increased, though moderate, risk for whom aspirin prophylaxis may or may not be helpful. Beginning aspirin after the second trimester is not effective, nor is aspirin useful as a treatment for preeclampsia. The only treatment for preeclampsia is delivery.

After onset of preeclampsia, a subset of women is at increased risk for development of adverse maternal outcomes (central nervous system, cardiorespiratory, hepatic, renal or hematological morbidity, or death). Factors predisposing to worse maternal outcomes are chest pain, dyspnea, hypoxemia, thrombocytopenia, or an increase in serum creatinine or transaminases. Earlier gestational age at onset is also associated with worse outcomes, which may relate to an intrinsically more severe process or to an expectant management strategy (rather than delivery) when preeclampsia is diagnosed earlier. An online calculator for predicting the probability of adverse maternal outcomes is available at https://pre-empt.cfri.ca/monitoring/fullpiers. The value of isolated clinical signs, such as headache or abdominal pain for predicting adverse maternal outcomes, is low to moderate.

Preeclampsia complicates between 3 and 8% of pregnancies, though most cases are mild and occur late in pregnancy. A category which is called “severe preeclampsia” or “preeclampsia with severe features” is more worrisome. This group has either end-organ dysfunction or higher BP (systolic BP ≥ 160 mm Hg or diastolic ≥ 110 mm Hg).

Potential complications of preeclampsia are wide ranging, including renal insufficiency or failure (2–8%), acute respiratory distress syndrome (1–9%), pulmonary edema (related to hypoalbunemia), often exacerbated by iatrogenic fluid overload (1–2%), stroke (1–2%), liver hematoa (1%), vaso- genic cerebral edema (posterior reversible leukoencephalopathy syndrome [PRES]) (Fig. 1), and seizures. Another possible complication is a constellation of laboratory abnormalities known as HELLP syndrome: hemolysis, elevated liver enzymes demonstrating hepatic dysfunction, and low platelet count.

**Clinical Presentation in the Intensive Care Unit**

In the ICU, only the most severe forms of HDP are encountered. Approximately, approximately 50% of patients present with severe systolic hypertension and 25% with severe diastolic hypertension; however, 11% of patients do not present with hypertension. As hypertensive disorders of pregnancy are the leading obstetric cause of admission to the ICU, physicians should still look for other signs and symptoms of severe preeclampsia even when hypertension is absent.

The most frequent complaints upon admission are neurological, abdominal, renal, and visual. Among neurological issues, headache is the most common, followed by seizures and hyperreflexia, and, less frequently, altered mental status. Renal dysfunction is generally characterized by oliguria and a rise in creatinine; a small proportion of patients present with acute renal failure, which is more frequent in the most severe forms of endothelial dysfunction, such as HELLP syndrome class 1. Critical care physicians should be aware that the normal upper limit of serum creatinine in pregnancy is between 0.6 and 0.8 depending on the trimester.

Abdominal symptoms are mainly epigastric or right upper quadrant pain and less frequently nausea and vomiting. Visual abnormalities occur in nearly 25% of patients with preeclampsia and between 19 and 45% of patients with eclampsia; they include blurred vision, presence of scotomata or photopsias, in severe cases cortical blindness associated with PRES, and infrequently retinal detachment.

Although stroke is rare among pregnant patients, the risk is higher among patients with HDP versus pregnant/
In patients with HDP, intracranial hemorrhage (ICH) is associated with worse outcomes compared with ischemic stroke; in fact, ICH is one of the leading causes of maternal mortality associated with preeclampsia. In parturient patients without HDP, HDP. In patients with HDP, intracranial hemorrhage (ICH) is associated with worse outcomes compared with ischemic stroke; in fact, ICH is one of the leading causes of maternal mortality associated with preeclampsia.

General Management in the Intensive Care Unit

Key components of preeclampsia management in the ICU are delivery, corticosteroids for fetal lung maturation if the pregnancy is preterm, magnesium sulfate (MgSO₄) for prevention of eclampsia, antihypertensive medications, and appropriate use of fluids.

Indications for Delivery and Fetal Lung Maturation

Preeclampsia can only be resolved by delivery, which is always appropriate for the mother’s health. However, since neonatal survival is tightly coupled with gestational age at delivery, cases of preeclampsia remote from term may be managed expectantly with stepped-up surveillance of mother and fetus. This often includes hospitalization, depending on degree of severity, and administration of antenatal corticosteroids to the mother so as to accelerate maturation of the fetal lungs and reduce neonatal mortality. Both betamethasone and dexamethasone cross the placenta; other steroids are ineffective. Betamethasone is administered as...
12 mg intramuscular (IM) q 24 hours, for a total of two doses. Dexamethasone is given as 6 mg IM q 12 hours, for a total of four doses. If expectant management is attempted, it should be ended if maternal or fetal status deteriorates or when gestational age between 32 and 34 weeks is attained. Contraindications to expectant management include eclampsia (seizure), abruptio placentae, stillbirth, coagulopathy, HELLP syndrome, or hypertension which is difficult to control with standard agents. Preeclampsia does not affect decision making as to mode of delivery; vaginal or cesarean delivery may be performed according to the usual obstetric considerations.

**Magnesium Sulfate**

Although at least 15 randomized trials support the efficacy of MgSO₄ to prevent eclampsia, the strongest evidence comes from a multicenter randomized placebo-controlled trial known as Magpie (magnesium trial for prevention of eclampsia). This study included 10,141 patients with pre eclampsia from 33 high- and low-income countries who were allocated MgSO₄ or placebo when there was uncertainty about using MgSO₄. Patients receiving MgSO₄ presented significantly lower risk of developing eclampsia (relative risk [RR]: 0.42; 99% confidence interval [CI]: 0.29–0.60) and abruptio placentae (RR: 0.67; 99% CI: 0.45–0.89) than patients allocated placebo, regardless of the severity of preeclampsia. Studies characterized as “real world use studies” by McDonald have confirmed these results.

MgSO₄ can be administered either via intramuscular injection or intravenous (IV) infusion, the latter being the most frequent, consisting of a loading dose of 4 g followed by a maintenance dose of 1 g/h for 24 hours. The appropriate use of MgSO₄ was not associated with serious maternal adverse effects. Some minor side effects were noted which included flushing, nausea or vomiting, muscle weakness, and itching or tingling. Other effects reported less...
frequently were hypotension, slurred speech, dizziness, drowsiness, confusion, headache, thirst, and a small increase (5%) in the risk of cesarean section.\textsuperscript{35} Although absent or reduced tendon reflexes and/or respiratory depression were uncommon (1 and 0.5% in MgSO\textsubscript{4} vs. placebo groups, respectively), the latter was significantly more frequent in patients using MgSO\textsubscript{4} (51 versus placebo group (26, number needed to harm = 206).\textsuperscript{32} Patients on the above-mentioned MgSO\textsubscript{4} regimen required only clinical monitoring: respiratory frequency and tendon reflexes every 30 minutes and urine output hourly, but not blood monitoring.\textsuperscript{33} Doses needed to be reduced by half if respiratory rate was reduced but oxygenation was preserved, tendon reflexes were slow, or urine output was less than 100 mL in 4 hours.\textsuperscript{33} In other studies, doses for MgSO\textsubscript{4} may be higher (e.g., loading dose up to 6 g and maintenance dose of 2 g/h, which is the common dose regimen in North America)\textsuperscript{32}; however, given the magnitude of the "Magpie" study, the above-mentioned doses are safe and were proven to be effective.

To reduce monetary costs, diminish adverse effects, or mitigate trained staff shortages, some studies have evaluated alternative strategies for MgSO\textsubscript{4} administration, such as shortening the time of infusion to 12 hours instead of 24 hours, lowering overall doses, and only selecting patients who would receive the maximum benefit. The former two arguments did not reduce positive effects of MgSO\textsubscript{4}; however, the studies were too small to be conclusive.\textsuperscript{37,38} The latter argument referred to the number needed to treat (NNT) to prevent one case of eclampsia, which numbered 50 in cases of severe preeclampsia versus 100 in patients with nonsevere preeclampsia.\textsuperscript{32} The NNT is also affected by a country’s economic condition (324 high- vs. 43 low-income countries) and the associated higher and lower costs to prevent a single case.\textsuperscript{39}

In summary, to prevent eclampsia, it is good practice to administer MgSO\textsubscript{4} to any patient with severe preeclampsia, irrespective of the country of origin.\textsuperscript{40} The uncertainty regarding the use of MgSO\textsubscript{4} for nonsevere preeclampsia in high-income countries is due primarily to overall costs; however, considering the negligible costs and obvious benefits, preventive use of MgSO\textsubscript{4} in low- and middle-income countries is recommended by the International Society for the Study of Hypertension in Pregnancy guidelines, regardless of the severity of preeclampsia.\textsuperscript{5}

Treatment of Severe Hypertension

Antihypertensive drugs should be started in patients presenting with severe hypertension (BP $\geq$ 160/110 mm Hg) with the aim of preventing maternal complications, such as ICH.\textsuperscript{3,6,30,41} In one small case series study, 96% of patients with preeclampsia–eclampsia with ICH developed systolic BP $> 160$ mm Hg immediately before stroke; during the 6 hours prior to developing stroke, less than 10% of patients had received antihypertensive medication and 54% died.\textsuperscript{42}

A target BP in the range of 140 to 150/90 to 100 mm Hg is considered safe for the mother and fetus as it preserves maternal cerebral autoregulation.\textsuperscript{41} BP should not lower than 130/80 mm Hg, to ensure uteroplacental perfusion. Fetal heart rate tracing is considered a good marker for uteroplacental perfusion.\textsuperscript{40} It should be noted that the lower threshold recommendation is still being studied.

Although the existing evidence for treatment of severe hypertension during pregnancy cannot favor one antihypertensive drug over another,\textsuperscript{43} the most commonly used drugs are IV labetalol and hydralazine, and oral nifedipine.\textsuperscript{43–45} Drug preference will depend on setting, the physician, and fetal–maternal adverse effects (see Table 1).\textsuperscript{41,44,46–49} Labetalol and nifedipine have shown similar efficacy and safety in this context.\textsuperscript{50} Both short-acting (capsules) and intermediate-released (tablets) nifedipine were effective for controlling severe hypertension compared with IV hydralazine, IV labetalol, and other drugs.\textsuperscript{51} There was some concern about maternal hypotension with short-acting versus intermediate-released nifedipine based on results from one small trial; however, the incidence of hypotension with short-acting nifedipine was similar to the incidence with other drugs (< 2%) in six other trials.\textsuperscript{51} Nifedipine could also be used safely with MgSO\textsubscript{4}, as it did not increase neuromuscular blockade or hypotension.\textsuperscript{52} We do not recommend using extended-release nifedipine for treating severe hypertension in pregnancy given that there is no way to reverse effects rapidly.\textsuperscript{51} Labetalol should be avoided in patients with asthma, congestive heart failure, and heart disease. Continuous infusion of nicardipine is used in some countries where other drugs have failed.\textsuperscript{41,53} Sodium nitroprusside, for short-term use, may be required for uncontrollable hypertension.\textsuperscript{47} Antihypertensive drugs contraindicated during pregnancy are atenolol, for its association with fetal growth restriction, and angiotensin-converting enzyme (ACE) inhibitors, for their association to oligohydramnios, fetal renal failure, and stillbirth.\textsuperscript{47}

Treatment for postpartum patients with severe hypertension can be initiated with any of the previously mentioned drugs with the exception of nitroprusside due to the potential of thiocyanate toxicity for breastfeeding infants. Enalapril can be used safely during the postpartum period.\textsuperscript{54}

Fluid Management in the Intensive Care Unit

Fluid management of severe preeclampsia in the ICU is based mainly on theoretical concerns about pulmonary edema and less on evidence-based recommendations. Although most patients with preeclampsia have diminished intravascular volume compared with nonhypertensive pregnant patients, there is no evidence to support volume expansion for improving maternal–neonatal outcomes.\textsuperscript{55,56} Guidelines recommend judicious use of volume, usually at a maintenance rate of 80 mL/h.\textsuperscript{45,57,58}

Oliguria represents a challenge in preeclampsia patients, as it has low predictive value for fluid responsiveness.\textsuperscript{39} In one small study, more than 50% of patients with severe preeclampsia presented with oliguria, of whom 50% responded to fluids. Variation in velocity time integral of the subaortic blood flow\textsuperscript{4} 12% during passive leg raising was a good predictor of fluid response in these patients.\textsuperscript{59} Lung ultrasound to detect pulmonary edema and new hemodynamic monitoring tools such as electrical cardiometry, LiDCOplus, PICCO, or Vigileo have been
| Drug                          | Mechanism of action     | Peak effect | Initial dosage                  | Maintenance dosage       | Maximum dosage | FDA class | Adverse effects                                                                 | Breast feeding |
|-------------------------------|-------------------------|-------------|---------------------------------|--------------------------|-----------------|-----------|--------------------------------------------------------------------------------|----------------|-------------------------------------------------|
| Labetalol (IV)                | a1-blocker and b-blocker| 10–15 min   | 20 mg bolus, if ineffective after 15 min give 40 mg more; if still ineffective give 80 mg more 10–15 min later, and repeat for up to two more boluses of 80 mg each | Bolus q.i.d. or infusion 1-2 mg/min | 220–300 mg | C         | Maternal flushing, palpitations, light headedness, and scalp tingling Neonatal hypertension (large doses) Fetal bradycardia | Yes            |
| Hydralazine (IV)              | Arterial vasodilator    | 30–120 min  | 5 mg, if no response after 15–20 min give 5–10 mg q 20 min | t.i.d. or q.i.d.         | 20–30 mg | C         | Maternal headache, nausea, flushing, palpitations Neonatal thrombocytopenia | Yes            |
| Nifedipine (PO)               | Calcium channel blocker | SA = 30 min | SA = 5–10 mg PO, give 10 mg if no response in 20–30 min IR = 10 mg every 45 min | SA = t.i.d. IR = b.i.d.  | SA = 120–180 mg | C         | Maternal tachycardia, flushing, palpitations, peripheral edema, headache, nausea, vomiting | Yes            |
| Nicardipine hydrochloride (IV)| Calcium channel blocker | 20 min      | 5 mg/h can be increased 2.5 mg/h every 5 min until 15% reduction in BP or maximum dosage | Continuous infusion       | 15 mg/h | C         | Headache                                                                 | Yes            |
| Sodium nitroprusside (IV)     | Arterial vasodilator    | 1–2 min     | 0.2 µg/kg/min, increase every 5 min | Continuous infusion       | 2–4 µg/kg/min | C         | Cyanide and thiocyanate toxicity if used > 4 h                                    | No             |

**Abbreviations:** FDA, Food and Drug Administration; ICU, intensive care unit; IR, intermediate release; IV, intravenous; SA, short acting.

*a* Administer appropriate bolus dosage to control hypertension q.i.d. (e.g., 40 mg every 6 hours).

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**Ostetric Hemorrhage**

Ostetric hemorrhage is the first cause of maternal death globally, and the second cause of admission to the ICU for obstetric hemorrhage. Obstetric hemorrhage can be categorized as antepartum or postpartum hemorrhage (PPH). PPH may occur during the last 24 hours of pregnancy (early PPH) or between 24 hours and 6 weeks after delivery (late PPH). The majority of hemorrhage is due to obstetric hemorrhage, with postpartum hemorrhage accounting for the second most common cause of maternal death.

**Definition and Causes of Obstetric Hemorrhage**

Obstetric hemorrhage is the first cause of maternal death globally, and the second cause of admission to the ICU for obstetric hemorrhage. Obstetric hemorrhage can be categorized as antepartum or postpartum hemorrhage. Incorrect diagnosis, substandard or improper management, lack of monitoring, and insufficient resuscitation are directly linked to maternal death or near miss due to obstetric hemorrhage.

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**General Management of Specific Conditions in the Intensive Care Unit**

General management of eclampsia consists of hemodynamic and respiratory support, delivery, and MgSO₄. MgSO₄ is also the drug of choice for treating eclampsia with the same intravenous regimen and monitoring protocol as for preeclampsia. It is superior to diazepam, phenytoin, or a lytic cocktail (chlorpromazine, promethazine, and pethidine) for preventing recurrent convulsions, and it has been associated with fewer maternal deaths than diazepam or lytic cocktail at 4–6 g if seizure recur extra than 2 h of MgSO₄, given this criteria, the majority of clinical guidelines do not recommend using corticosteroids for accelerating postpartum recovery (Clinical Trials Register under the number NCT00711841).

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**Eclampsia**

Eclampsia is a severe manifestation of preeclampsia defined by new onset of convulsions or headache without focal neurological symptoms and increased blood pressure. It is a medical emergency, and treatment should be initiated immediately. The first step in management is the administration of magnesium sulfate, usually in doses of 4 g intravenously over 5 minutes, followed by maintenance doses of 6 to 8 g/h. Other therapies may include anticonvulsants (diazepam, phenytoin), corticosteroids, and renal replacement therapy. The goal of treatment is to prevent recurrent convulsions and protect the mother and fetus.
and 42 days postpartum (late PPH). We will primarily refer to early PPH in this section as it is more common.

The traditional definition of PPH is considered to be the loss of ≥ 500 mL of blood loss after vaginal delivery or ≥ 1,000 mL after cesarean section; however, these volumes are near normal losses in these settings. Presently, there is no universal definition for PPH and, as we have indicated earlier, can affect accurate diagnosis, resulting in underestimation of blood loss and treatment delay. A reasonable approach would include properly estimating blood losses, via training on accurate visual estimation or the use of calibrated drapes, and close clinical and laboratory monitoring, including coagulation parameters and arterial blood gases.

Definitions of severe PPH are also inconsistent, depending on the study, for example, transfusion of ≥ 4 units of blood, a hemoglobin drop of ≥ 4 g/dL, hemostatic intervention requirement, sudden blood loss of > 1.5 L, requirement of ICU admission, or a combination of at least two. In the same way, massive hemorrhage is usually defined as loss of 150 mL/min, loss of one blood volume in 24 hours, or half blood volume in 3 hours. These definitions are of limited value for planning advanced patient support, for example, triggering massive transfusion protocols. Therefore, centers need to set their own guidelines for these thresholds.

The most frequent cause of PPH is uterine atony, followed by placental irregularities, including retained products of conception or abnormal placental implantation, genital tract trauma, and coagulopathy. These are commonly referred to as “the four Ts”: tone, tissue, trauma, and thrombin.

### Risk Factors for PPH, Severe PPH, and Requirement for Advanced Procedures

Some risk factors for PPH are placenta previa, abruptio placentae, multiple pregnancy, previous PPH, obesity, anemia, or delivery by cesarean section. However, most patients with PPH do not present with any risk factor whatsoever.

Patients with PPH requiring ICU admission will usually be those who have failed the first line of treatment, such as uterine massage and the utilization of oxytocic drugs. In this particular subset of patients, baseline fibrinogen levels of < 2 g/L were independently associated with the development of severe PPH. At the same time, fibrinogen < 2 g/L on admission, abnormalities of placental implantation, prothrombin time < 50%, heart rate > 115 bpm, and detectable troponin I were independent risk factors for requiring an advanced procedure, namely, uterine arterial embolization or hemostatic open surgery (arterial ligation, peritoneal packing, or hysterectomy). Assessing these variables on admission may help physicians promptly set the most appropriate treatment.

### Clinical Presentation

Women suffering from PPH are young and usually healthy; therefore, they may appear well until collapse is imminent. Tachycardia is an unspecified and late sign, not occurring until 1,000 mL of blood have been lost. Similarly, the diagnostic accuracy of systolic BP < 95 mm Hg is poor for detecting moderate and severe blood loss. Clinical signs or laboratory parameters suggestive of tissue perfusion impairment should be sought instead: oliguria, skin perfusion abnormalities, altered mental status, central venous saturation, arterial base deficit, and lactate.

### Medical Management

Severe PPH should be managed by a multidisciplinary team including nurses, midwives, obstetricians, anesthetists, critical care physicians, hematologists, blood banks, and laboratories. Fluid resuscitation should be performed by the first responder while simultaneously calling for help; monitoring should be continuous as rapid assessment of the cause of PPH is established. The bladder should be emptied through a Foley catheter that is left in place. Hypothermia, acidosis, and hypocalcemia should be corrected to improve coagulation.

Most cases of early PPH are caused by uterine atony. Knowledge of how to manage uterine atony is crucial in preventing maternal deaths from PPH. If uterine atony is suspected, the first step is to assess uterine tone: within a few minutes after delivery of the placenta, the uterus should be palpably firm and at the level of the umbilicus. Transabdominal uterine massage or bimanual uterine massage (one hand in the vagina, and the other on the abdomen) can help restore tone and diminish bleeding while additional interventions are readied.

### Uterotonics

Pharmacological interventions require no specialized personnel and may be employed after vaginal or cesarean delivery. Commonly available drugs are oxytocin, methyl-ergonovine maleate, and the prostaglandin drugs dinoprostone, carboprost tromethamine, and misoprostol. Not all drugs are available in all facilities. Considerable variation exists in sequence of agents and protocol for use, but there is no evidence to recommend one particular sequence over another. Protocols should be derived at the local level. Some common dosage recommendations are given in Table 2.

### Fluids

Meta-analysis from Perel et al did not indicate differences in mortality among critically ill patients randomized to colloids versus crystalloids. However, resuscitation with hydroxyethyl starch (HES) was associated with a slightly higher risk of mortality than with crystalloids (RR: 1.10; 95% CI: 1.02–1.19). After Perel et al’s analysis, both the CHEST and CRISTAL trials did not isolate any differences in mortality between ICU patients randomized to 6% HES (130/40) versus normal saline (NS) in the former trial or any colloid versus NS in the latter. However, starches, gelatin, and dextran were associated with renal failure/requirement of renal-replacement therapy and bleeding. Considering safety profiles and cost, crystalloids are more favorable than colloids for resuscitation.

Normal saline (NS), lactated Ringer’s, and balanced salt solution are the most common crystalloids. In some studies, the use of large volumes of NS resulted in hyperchloremic
metabolic acidosis, which, in turn, was associated with renal dysfunction or immune response deficit.\textsuperscript{90,91} However, the evidence is still inconclusive.

**Blood Products and Hemostatic Agents**

New aspects of blood transfusion have illustrated that the use of higher fresh frozen plasma (FFP): RBC ratios for massive hemorrhage in trauma patients was associated with less coagulopathy and in some cases lower mortality than in standard regimens.\textsuperscript{75,90} In an observational study, Pasquier et al. demonstrated that the use of higher FFP:RBC ratios (1:1.2) in patients with PPH lower the odds for requiring advanced procedures (odds ratio: 1.25; 95% CI: 1.07–1.47), contrary to the use of lower ratios (1:1.6).\textsuperscript{72} Therefore, in obstetrics, some guidelines recommend the implementation of massive transfusion protocols.\textsuperscript{93} Guiding transfusion by near-patient tests (e.g., rotational elastometry/thromboelastography) for these patients is still confined to research, due to the lack of convincing evidence.\textsuperscript{75}

Other products have been proposed to improve hemostasis overall: fibrinogen (improves clot strength), tranexamic acid (TA) (reduces clot lysis), or recombinant factor VIIa (rFVIIa) (improves fibrin clot).\textsuperscript{94} The role of low fibrinogen levels as a contributor or a severity marker in PPH remains unclear; however, replacement of fibrinogen with cryoprecipitate, FFP, or fibrinogen concentrate\textsuperscript{75,94} is usually recommended when plasmatic levels fall below 1 to 2 g/L.\textsuperscript{75,76,94} To date, the only RCT assessing pre-emptive use of fibrinogen concentrate for patients with PPH, irrespective of baseline fibrinogen levels, failed to show any effect on primary (RBC transfusion) or secondary outcomes (blood loss, severe PPH, etc.).\textsuperscript{94}

TA reduced mortality in trauma patients and perioperative blood loss and/or transfusion requirement in some surgical settings.\textsuperscript{96} In obstetric populations, however, preventive use of TA is questionable due to the higher risk of thrombosis. Novikova et al’s meta-analysis was limited in scope and did not cover this issue.\textsuperscript{97} Evaluation of TA for PPH treatment by a small RCT indicated shorter bleeding time, less hemoglobin drop > 4 g/L, and clinically insignificant reduction in blood loss for TA group (51 mL); however, the study was not powered to assess significant outcomes, such as maternal mortality or safety issues.\textsuperscript{78} The massive international WOMAN trial (http://womantrial.lshtm.ac.uk/) should clarify the role of TA for treating PPH.

Effectiveness of rFVIIa for PPH was evaluated in one open RCT. Patients not responding to the first line of treatment were randomized to early use of a single dose of rFVIIa versus standard treatment. The intervention group was associated with significantly lower use of second-line treatments, such as uterine compression sutures, embolization, arterial ligation, or peripartum hysterectomy, compared with standard treatment (RR: 0.56 [0.42–0.76]).\textsuperscript{99} When subgroup analysis was performed, instance of embolization was lower in the intervention arm than in the standard arm (RR: 0.5 [0.29–0.86]), while requirement of hysterectomy was not significantly different between the groups (RR: 0.38 [0.11–1.32]). Furthermore, the sample size was insufficient to assess mortality and safety and the study was not designed to evaluate cost-effectiveness.

Although some guidelines consider the use of rFVIIa only when hysterectomy is the last option, when primary- and secondary-line treatments fail, and when fertility is strongly desired, they do highlight the low level of evidence supporting this recommendation.\textsuperscript{76,77,100}

**Table 2** Oxytocic drugs for managing PPH and common dosage recommendations

| Drug                | Dosage                          | Comments                                              | Source   |
|---------------------|---------------------------------|                                                      |          |
| Oxytocin            | IV infusion (preferred): 10–40 units diluted in 1 L of crystalloid; alternatively, 10 units (1 mL) IM | Arrhythmias may occur                                 | US FDA   |
|                     |                                 | Hypotension if bolus injection                        |          |
|                     |                                 | Water intoxication with prolonged administration      |          |
| Methylergonovine    | 0.2 mg (1 mL) IM                 | Hypertension and vasospasm may occur; avoid if history of hypertension, angina, MI, stroke | US FDA   |
|                     | May repeat in 2–4 h              | Drug requires refrigeration                           |          |
|                     | Avoid IV administration          |                                                      |          |
|                     | PO formulation not helpful in acute PPH |                                                      |          |
| Dinoprostone        | Suppository, 20 mg, vaginal or rectal | Prostaglandin E2                                      | ACOG     |
|                     | May be repeated in 2 h           | Stored frozen; must be thawed before use              |          |
| Carboprost tromethamine (15-methyl prostaglandin F2) | 250 µg (1 mL) given IM; may repeat q15 min as needed, max. dose 2 mg (8 doses) | Analogue of prostaglandin F2α | US FDA   |
|                     |                                 | May trigger fever                                     |          |
|                     |                                 | Use with caution in asthmatic patients                |          |
| Misoprostol         | 800 µg, rectally; alternative, 800 µg sublingually | Form of prostaglandin E2 | ACOG; RCOG |
|                     |                                 | Note: FDA off-label for this use                      |          |

Abbreviations: ACOG, American College of Obstetricians and Gynecologists; FDA, Food and Drug Administration; IM, intramuscular; IV, intravenous; PPH, postpartum hemorrhage; RCOG, Royal College of Obstetricians and Gynaecologists.
Resuscitation Monitoring
Patients presenting with severe PPH should be monitored in a setting where vital signs and tissue perfusion parameters can be assessed frequently. Complete laboratory evaluation, including coagulation tests with fibrinogen, calcium, arterial blood gases, lactate, and mixed venous saturation, should be reviewed regularly. Monitoring parameters via some kind of structured chart, such as the modified early obstetric warning system triggering a protocolized action plan is recommended.

Nonmedical Treatment
If uterine atony is not corrected by first-line treatments, more invasive measures will be required. The intensive care specialist will probably not be undertaking them in person; however, they are discussed here merely to familiarize the reader with their management.

Insertion of an intravaginal balloon is useful to tamponade the uterus. A product designed for this specific purpose is the Bakri postpartum balloon manufactured by Cook Surgical, but the Sengstaken–Blakemore esophageal balloon, the Rusch urological balloon, a high-volume Foley catheter (single or multiple), and a condom catheter have all been used. These techniques replace the old concept of uterine packing. All these devices can be inserted vaginally, without need for anesthetics.

Laparotomy may be required for hemorrhage control. If this is undertaken, it requires appropriate surgical personnel and an operating room. Uterine compression sutures may be tried as a first line, either with or without a hysterotomy incision. Stepwise uterine devascularization, ligating one or both uterine arteries, one or both ovarian arteries, and/or the internal iliac (hypogastric) artery has been reported to be successful in avoiding hysterectomy in many cases. In some centers, embolization of the pelvic vessels may be available in the interventional radiology suite, as an alternative to hysterectomy. In all cases in which hemorrhage cannot be managed with conservative strategies, hysterectomy is required. This will usually be a first-line approach when hemorrhage is due to placenta accreta rather than to uterine atony.

Sepsis in Obstetric Patients
Epidemiology and Definition
Discussion of sepsis in pregnancy and the puerperium is complicated by shifting definitions and, until recently, a paucity of reliable data. The concept of sepsis itself was reframed in 2016 and should be understood in current practice as “life-threatening condition defined as organ dysfunction resulting from infection during pregnancy, childbirth, post-abortion, or the post-partum period.” At this point, however, more specific criteria have not been agreed upon.

The 2016 sepsis redefinition has yet to be deployed in studies of sepsis in obstetric populations. Published studies have utilized older case definitions, often relying on ICD-9 or ICD-10 coding for bacteremia instead of focusing on manifestations of organ failure. The relatively new availability of population-based studies is, of course, an improvement over older literature, which relied almost exclusively on single-center case series. An additional problem in calculating the incidence of sepsis in obstetrics is the question about what to use as the denominator: some use live births, although pregnancies may end in stillbirth, abortion, or miscarriage, while others use delivery hospitalization; still others use a calculated number of estimated total pregnancies. With these caveats in mind, it appears that in high-income countries, somewhere between 2 and 12 pregnancies per 10,000 maternities may be complicated by sepsis. In low- and middle-income countries, incidence is thought to be higher, though data collection are even more challenging. A review of “puerperal sepsis” commissioned by the World Health Organization estimated an incidence of 3 to 5 cases per 100 live births in the late 1990s—two orders of magnitude higher than figures above—though the case definition here included all cases of puerperal infection, regardless of whether we would consider them sepsis per se, and for the most part excluded nongenital causes. A multisite cross-sectional study is planned for late 2017 to get a worldwide snapshot of the extent of maternal sepsis, with a particular focus on low- and middle-income countries.

The diagnosis of septic shock may be complicated in pregnancy. Lactate levels do not change but, because of the higher cardiac output, increased plasma volume and vasopressors, despite adequate fluid resuscitation, plus serum lactate > 2 mmol/L. Simple clinical criteria that correlate well with sepsis are summed up in the quick Sequential Organ Failure Assessment SOFA (qSOFA) score. Any two of the following count as a positive screen for sepsis in a patient who is thought to have infection and who is not yet in an ICU: (1) systolic BP < 100 mm Hg; (2) respiratory rate > 22/min; and (3) altered mental status. In a patient believed to be infected who is already in the ICU, a two-point increase in the standard SOFA score should be taken to represent sepsis.

It remains unclear at this time how the qSOFA score will perform in obstetric patients. Although mental status does not change, normal pregnancy may increase respiratory rate by a small amount and definitely lowers BP. Many healthy pregnant women would be expected to have a systolic BP < 100 mm Hg. To limit the false-positive rate of the qSOFA score during pregnancy, it would make sense to use a different cutoff for systolic BP, probably around 90 rather than 100 mm Hg; studies to validate the best cutoff are urgently needed.

The World Health Organization has recently followed up the Sepsis-3 consensus conference with a proposal to redefine maternal sepsis as: “a life-threatening condition defined as organ dysfunction resulting from infection during pregnancy, childbirth, post-abortion, or the post-partum period.” At this point, however, more specific criteria have not been agreed upon.
decreased systemic vascular resistance, BP is normally lower in pregnancy than at other times. Thus, using a nonpregnant norm for mean arterial pressure will tend to overdiagnose septic shock in pregnancy. As earlier, before measuring BP perform left uterine displacement. An indicator of hypoperfusion not available outside of pregnancy is fetal heart rate tracing, which is sensitive to alterations in uteroplacental perfusion. This is feasible to obtain as early as 24 weeks into the pregnancy, depending on the patient’s body habitus but should be interpreted by a qualified individual.

The case fatality rate in maternal sepsis is also somewhat difficult to elucidate, again related to the variable definitions of sepsis. In the United Kingdom, it has been reported as 1.5% in the United States roughly 3 to 5%, and in the Netherlands 7.7%. In a study from Texas looking only at cases of pregnancy-associated severe sepsis in which organ failure or shock featured, maternal mortality was 9.7% Group A streptococcal sepsis, the classic agent of “childbed sepsis” or “puerperal fever,” remains especially deadly; the case fatality rate in the 20th century was 14%. Obstetric Causes of Sepsis

Sepsis among pregnant and postpartum patients may be related to the uterus and genital tract or to any other organ system. Onset may be antepartum, intrapartum, or postpartum. Major causes of antepartum maternal sepsis are pneumonia, urinary tract infection, and chorioamnionitis, while postpartum cases are most often attributed to the uterus, genital tract, wound (genital tract laceration or episiotomy, or cesarean incision), and urinary tract. As is true outside of pregnancy, a source is not always identified. A wide range of organisms have been implicated, from streptococci and staphylococci to Escherichia coli and other gram negatives. Anaerobic and fungal infections have been reported. Mixed infection is common, as is failure to identify the inciting organism (33–42% of cases).

Septic abortion is still a frequent cause of maternal mortality in low-income countries with restrictive abortion laws. It is usually an ascending polymicrobial infection caused by bacteria from vaginal flora but still can be related to Clostridium perfringens. Also, some cases of fatal Clostridium sordellii infections after medical abortion in previously healthy women were recently reported in high-income countries.

Patients with septic abortion usually present with fever, uterine tenderness, a foul smelling cervical discharge of blood and pus, nausea, and vomiting. In countries where abortion is restricted, women are usually reluctant to disclose they have had an abortion; thus, a pregnancy test should be ordered for any woman of childbearing age presenting with even mild signs of sepsis. Serum or urine human chorionic gonadotropin will be positive days to weeks after the end of pregnancy.

The classical “Mondor’s syndrome” consists of a fulminant form of septic abortion due to C. perfringens characterized by rapidly progressing shock and intravascular hemolysis, expressed by jaundice, mahogany-colored urine, and acute renal failure. Disseminated intravascular coagulation (DIC) with skin necrosis, muscular cramps, and particularly absence of fever are common. Uterine findings are generally necrotizing endomyometritis or gas gangrene.

Toxic shock syndrome is a particular clinical presentation of septic abortion due to toxin-producing bacteria, such as some strains of E. coli, Staphylococcus aureus, Group A streptococcal infection, C. sordellii, and the above-mentioned C. perfringens. The clinical picture is categorized by acute onset of abdominal cramping, vomiting or diarrhea, and hypotension, fever, skin rash, and myalgia are frequent signs of E. coli, S. aureus, and Group A streptococcal infections, while hemoconcentration, a leukemoid reaction, and the absence of fever are found in C. sordellii infections. Septic abortion can also present with pelvic or abdominal abscesses, septic thrombophlebitis, or uterine perforation with or without bowel injury (Fig. 2). Obstetric Disorders in the ICU

Fig. 2 (A, B) Abdomen computed tomographic scan demonstrating free intraperitoneal air (arrows) due to bowel perforation in a 26-year-old patient admitted to ICU for septic abortion, referring abdominal pain, vomiting, and diarrhea after 2 weeks of having undergone uterine curettage under unsanitary conditions. ICU, intensive care unit.
General Medical Management of Sepsis

Sepsis during pregnancy and the puerperium should be treated no differently than in a nonpregnant adult. Early recognition with appropriate cultures and expedient antibiotic therapy is essential. Because of the wide range of potential sources and possible organisms, broad-spectrum coverage must be initiated. Empiric antibiotic choices should be driven by suspected source and local antibiotic resistance patterns. Most antibiotics can be given in pregnancy, though generally it is best to avoid tetracyclines, chloramphenicol, and fluoroquinolones. Aminoglycosides are potentially ototoxic to fetuses as well as their mothers, but are commonly given when gram-negative infection is suspected. As is true when prescribing any drug during pregnancy, it is prudent to consult experts (maternal–fetal medicine specialists, infectious disease specialists, perinatal pharmacologists) or available databases to minimize risk insofar as is feasible. Fortunately, organogenesis is complete by the end of the first trimester; therefore, teratogenicity is seldom an issue when antibiotics are prescribed for maternal sepsis. In the event of septic shock during pregnancy, norepinephrine is the vasopressor of choice.136,137

Sepsis is commonly associated with preterm birth. This is true even when the source of infection is nonuterine, presumably related to the systemic effects of proinflammatory mediators. No attempt should be made to delay or postpone delivery if the mother is suspected to be septic. However, because delivery is likely and the newborn may be premature, septic, or sick, maternal sepsis must be handled in a hospital with obstetric facilities and neonatal intensive care services.

Fluid resuscitation as a component of sepsis management may be affected by pregnancy.137 Pregnant women have lower colloid oncotic pressure and are more predisposed to pulmonary edema with large volumes of isotonic crystalloid. If hypotension is a concern, the intensivist must bear in mind that after midpregnancy, compression of the inferior vena cava (IVC) by the gravid uterus limits cardiac output because of its effect on preload. Thus, the first step in addressing hypotension in a pregnant woman is not to administer intravenous fluids but to relieve IVC compression. Even a 10-cm elevation of the right hip off the bed is enough to effect left uterine displacement and resolve vena caval compression. Both passive leg raising and point-of-care ultrasound138,139 have been advocated as ways of assessing fluid responsiveness in the hypotensive septic patient and may be considered in this population as well, though no information is available about reliability of these tests in pregnancy.

Source Control

Many cases of sepsis in obstetric patients are localized to the uterus, which makes source control simple. Chorioamnionitis inevitably provokes labor, whether term or preterm, which serves to empty the uterus. Postpartum endometritis requires assessment for retained products of conception and may require curettage or other methods of uterine evacuation. Surgical site infection (episiotomy or cesarean incision) is treated in the standard way.

Septic abortion requires urgent removal of infected products of conception,122,140 which can be safely performed through either sharp or suction curettage (electric or manual).135 In places where it is available, these procedures may be performed under ultrasound guidance. Laparotomy is reserved for patients not responding to antibiotics and uterine curettage, presenting with clostridial myonecrosis, uterine perforation with suspected bowel injury, and pelvic abscess needing open surgical drainage.140 Indications for hysterectomy include uterine gas gangrene (crepitation of the pelvic tissue or abdominal computed tomography scan showing air within the uterine wall) and a discolored woody appearance of the uterus.140

Acute Fatty Liver of Pregnancy

Acute fatty liver of pregnancy is a rare cause of hepatic failure related to pregnancy. Incidence is estimated at roughly 1:10,000 pregnancies and the underlying cause is still in doubt. The leading theory at this time is that it relates to a fetal defect in fatty acid oxidation, specifically a deficiency of long-chain 3-hydroxyacyl coenzyme A dehydrogenase. Fetally derived fatty acids accumulate in the maternal compartment and overwhelm hepatocytes, leading to hepatotoxicity.141 The standard approach to liver failure will be insufficient unless coupled with delivery.

Peripartum Cardiomyopathy

Peripartum cardiomyopathy (PPCM) is defined by the European Society of Cardiology as “an idiopathic cardiomyopathy presenting with heart failure secondary to left ventricular systolic dysfunction toward the end of pregnancy or in the months following delivery, where no other cause of heart failure is found.”142 This should mean that it is a diagnosis of exclusion. PPCM behaves like any other dilated cardiomyopathy. Incidence varies widely, though stringency of diagnosis is not consistent; in some areas of Africa and Haiti, the incidence has been reported as high as 1:100, while only 1:20,000 in Japan.142,143 Symptoms, which are no different from symptoms of any other heart failure, may be confused in early mild stages with common discomforts of pregnancy, such as edema, fatigue, and dyspnea. Most cases are diagnosed postpartum, which obviates any concerns about the fetus. There may be a causative or inciting factor in overproduction of a low-molecular-weight form of prolactin, and therefore, a possible therapeutic approach is bromocriptine; though there are no conclusive data to support its efficacy and safety are available to date.

Echocardiography is required for diagnosis. Left ventricular hypokinesia will be present, and ejection fraction is usually less than 45%. Troponins are rarely elevated in PPCM. Brain natriuretic peptide is elevated in PPCM, as in most other causes of heart failure.

Acute heart failure from PPCM is managed much the same as any other heart failure. Diuretics, vasodilators, and β-blockers may be used. ACE inhibitors and angiotensin-receptor blockers are not safe in pregnancy, being associated
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Amniotic Fluid Embolism

Amniotic fluid embolism (AFE) is an uncommon but severe and very often fatal condition. Case fatality rates associated with AFE decreased from 61% in previous reports to 11 to 35% in recent studies; probably related to early recognition and prompt treatment support.146,147

Although the exact pathogenesis of this syndrome is still unknown, it seems to be the result of an exaggerated maternal immune/inflammatory response to fetal antigens considered idiosyncratic by some authors.146,148,149 Risk factors associated with AFE vary according to different reports, such as multiple pregnancy, maternal age > 35 years, multiparity, cesarean or instrumental delivery, polyhydramnios, abruptio placenta or placenta previa, eclampsia, uterine rupture, cervical laceration.146,147,150

Some patients have premonitory signs or symptoms occurring before overt AFE, such as dyspnea, thoracic pain, panic, nausea, vomiting, or dizziness.146,147 AFE usually develops abruptly during or within 30 minutes of vaginal delivery or cesarean section, although some studies reported its occurrence within 48 hours peripartum/cesarean.151 The clinical presentation of AFE is usually protean, involving a combination of severe hypotension, arrhythmia or cardiac arrest, breathlessness, hypoxemia, cyanosis, respiratory distress, mental status alterations or seizures, and coagulation abnormalities, such as DIC.146,147,150 Diagnosis of AFE is based on the above clinical criteria and the exclusion of other likely causes.149,150,152

Although the detection of squamous cells in the maternal pulmonary artery was once considered pathognomonic of this syndrome, it should not be used as diagnostic criteria because similar cells have been found in pregnant patients without AFE and in nonpregnant patients.146,152,153 Some laboratory tests, including fetal antigens or markers of mast cell degranulation, among others, were proposed for diagnosing AFE, namely, sialyl Tn, zinc coproporphyrin 1, serum tryptase, C1 esterase inhibitor (C1INH1), complement C3 and C4, interleukin 6, among others.146,154–156 C1INH1 is a protein that also inhibits kallikrein and coagulation factors XIIa and IXa. During normal pregnancy, C1INH1 activity levels are decreased. All patients with AFE presented even lower C1INH1 activity than normal pregnant patients, and those with fatal AFE had the lowest levels recorded.157 Although these tests seem promising, more research is needed before extending their use.146,158

Initial evaluation, management, and monitoring should be simultaneous. Cardiovascular and respiratory support, including invasive mechanical ventililation, should be provided urgently. If maternal cardiac arrest occurs before delivery, perimortem cesarean should be performed.159 Massive transfusion protocols are frequently required to replace red blood cells and coagulation factors in case of bleeding and DIC.152 FFP is also a source of C1INH1, which is reduced in AFE patients,156,160 while cryoprecipitate contains fibrinogen which can improve cellular and amniotic fluid removal from maternal blood.146

Differential diagnosis such as thrombotic or air pulmonary embolism, peripartum cardiomyopathy, tocolytic-induced pulmonary edema, PPH, and septic or anaphylactic shock should be considered.149 One key criteria in discriminating AFE from the other diagnoses is the presence of DIC, a hallmark variable in AFE, which only presents in severe cases of obstetric hemorrhage and septic shock, both easily identifiable.

Conclusion

Most obstetric patients admitted to the ICU arrive postpartum, rather than pregnant, taking the fetus out of the equation. The mother may be admitted to the ICU for nonobstetric reasons, which we have not touched upon here, or for specific reasons stemming from the pregnancy or delivery. We have presented here a few of the more common obstetric causes of critical illness. It is important to emphasize that care of a critically ill obstetric patient is not only a multidisciplinary endeavor involving critical care physicians and nurses, obstetricians and maternal–fetal medicine specialists, obstetric nurses, anesthesiologists, neonatologists, among others but also poses a challenge to medical personnel not familiar with the changes and disorders typical of pregnancy.

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