

Postpartum emergency

Postpartum emergency means any time within 6 weeks after birth. The four big killers are bleeding (hemorrhage) (too much bleeding), infection (endometritis spreading to sepsis), high blood pressure crisis (eclampsia or stroke from pressure $\geq 160/110$), and pulmonary embolism (a clot lodging in a lung artery) (clot in the lungs). Identify which one fast, because each needs different urgent treatment.

THE ONE-LINE TRUTH

After a baby is born, four life-threatening emergencies can happen within 6 weeks: postpartum bleeding (hemorrhage) (bleeding >500 mL vaginal or >1000 mL cesarean), endometritis (infection of the uterine lining), preeclampsia with severe features or eclampsia (dangerously high blood pressure $\geq 160/110$ mmHg causing seizures and organ damage), and pulmonary embolism (a clot lodging in a lung artery) (blood clot blocking lung arteries).

The chain — how it unfolds

1. Uterine atony or retained placenta or genital tract laceration allows continuous bleeding from open spiral arteries at the placental implantation site. — why: When the uterus muscle (myometrium) stays relaxed instead of contracting down tight, it cannot compress the spiral arteries that supplied the placenta. Blood pours out like an open faucet—500 to 1500 mL or more in minutes. Picture a garden hose with no one squeezing it shut. If there is a tear in the cervix or vagina or uterus, blood leaks from the torn vessel even if the uterus is firm. If pieces of placenta are stuck inside, they physically block the uterus from squeezing all the way and serve as a breeding ground for bacteria.

2. Bleeding (Hemorrhage) triggers a fast heart rate (tachycardia) (fast heart rate >100 bpm) first, then low blood pressure (hypotension) (low blood pressure, systolic <90 mmHg) late when compensatory reserves are exhausted. — why: Baroreceptors (pressure sensors in the aorta and neck arteries) detect dropping blood volume and signal the brainstem to release adrenaline. Adrenaline makes the heart beat faster and harder to push the shrinking blood volume through the body and keep oxygen delivery to the brain and heart. Heart rate climbs first—100, then 110, then 120 bpm. Blood pressure stays normal for a while because peripheral arteries squeeze tight (vasoconstriction) to shunt blood away from skin, gut, and kidneys toward vital organs. But once 1500 mL or more is lost (20 to 30 percent of blood volume), compensatory mechanisms fail and blood pressure crashes. Low blood pressure (Hypotension) is a late sign—by the time it appears, the patient is in decompensated shock.
3. Endometritis or chorioamnionitis seeds bacteremia (bacteria in the bloodstream), triggering systemic inflammatory response syndrome (SIRS): fever, a fast heart rate (tachycardia), fast breathing (tachypnea) (fast breathing >20 breaths per minute), and eventually septic shock. — why: Dead tissue, blood clot, and retained membranes in the uterus are a perfect breeding ground for bacteria—commonly *Streptococcus* (round bacteria in chains), *Staphylococcus* (round bacteria in clusters), *Escherichia coli* (rod-shaped gut bacteria), or *Bacteroides* (rod-shaped bacteria that live without oxygen). These bacteria release toxins (endotoxins and exotoxins) into the bloodstream. The toxins cause blood vessels to open too wide (vasodilation), fluid to leak out of vessels into tissues (capillary leak), and the heart to pump faster to try to maintain blood pressure. The result is septic shock: low blood pressure despite normal or high cardiac output, warm flushed skin early (because vessels are dilated), then cold clammy skin late (when compensatory vasoconstriction kicks in), plus fever $\geq 100.4^{\circ}\text{F}$ (38.0°C), heart rate >100 bpm, respiratory rate >20 breaths/min, confusion, and eventually multi-organ failure (kidneys stop making urine, liver enzymes rise, platelets drop, lactate rises >4 mmol/L showing cells are not getting enough oxygen).

4. Severe preeclampsia or eclampsia causes seizure, stroke, HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets $<100,000/\mu\text{L}$), pulmonary edema (fluid flooding the lungs) (fluid in the lungs), and acute kidney injury. — why: The blood vessel lining (endothelium) is injured by circulating antiangiogenic factors (proteins like sFlt-1 and soluble endoglin that block normal blood vessel growth and repair). That injury causes blood vessels to squeeze tight (vasospasm), raising blood pressure to dangerous levels—top number ≥ 160 or bottom number ≥ 110 mmHg. The high pressure and injured vessels cause: (1) Seizures (eclampsia) when blood pressure spikes or small bleeds happen in the brain (intracerebral bleeding (hemorrhage) or posterior reversible encephalopathy syndrome, PRES). (2) HELLP syndrome: red blood cells get shredded in damaged small vessels (microangiopathic destroying red blood cells (hemolytic) anemia, seen as schistocytes on blood smear), liver swells and bleeds under its capsule (causing severe right upper quadrant or epigastric pain and elevated AST/ALT), and platelets clump and get used up (thrombocytopenia $<100,000/\mu\text{L}$). (3) Pulmonary edema (fluid flooding the lungs): injured capillaries in the lungs leak fluid into air sacs, causing shortness of breath, low oxygen saturation, and crackles (popping sounds) when you listen to the lungs. (4) Acute kidney injury: damaged glomeruli (filtering units) leak protein into urine (proteinuria ≥ 300 mg/24 hours or protein/creatinine ratio ≥ 0.3) and stop filtering waste (creatinine rises >1.1 mg/dL, urine output drops <500 mL/day).
5. Pulmonary embolism (a clot lodging in a lung artery) blocks pulmonary artery branches, causing sudden low oxygen in the blood (hypoxemia) (low blood oxygen, saturation $<94\%$ on room air), fast breathing (tachypnea) (fast breathing), pleuritic chest pain (sharp pain worse with deep breathing), and obstructive shock. — why: A blood clot (thrombus) forms in a deep leg vein (deep vein clot formation (thrombosis), DVT) or pelvic vein during pregnancy or postpartum because blood clots more easily (hypercoagulable state from high estrogen and clotting factors), blood flow slows in compressed leg veins (venous stasis), and vessel walls can be injured during delivery. The clot breaks loose, travels through the right atrium and right ventricle (right side chambers of the heart), and lodges in a pulmonary artery branch. That blocks blood flow to part of the lung—blood cannot pick up oxygen in the blocked segment, so PaO_2 (partial pressure of oxygen in arterial blood) drops and oxygen saturation falls. The patient suddenly feels short of breath, breathes fast to try to get more oxygen, and has sharp chest pain because the lung tissue downstream from the clot is not getting blood (ischemia). If the clot is massive, it blocks so much lung circulation that the right ventricle cannot pump blood forward—pressure backs up, neck veins bulge (jugular venous distension), blood pressure drops (obstructive shock), and the heart can stop (pulseless electrical activity arrest).

6. Each abnormal vital sign and lab result is the body's emergency compensation trying to restore oxygen delivery, not the disease itself. — why: A fast heart rate (Tachycardia) (fast heart) is the body trying to push shrinking blood volume harder. Fast breathing (Tachypnea) (fast breathing) is the body trying to blow off carbon dioxide and bring in more oxygen. Fever is the immune system raising body temperature to fight infection. Low platelets and rising liver enzymes in HELLP are the result of damaged blood vessels using up clotting cells and injuring the liver. Rising creatinine and falling urine output are the kidneys shutting down when blood pressure drops or vessels are damaged. Falling hemoglobin is red blood cells being lost to bleeding or broken apart in damaged vessels. Every lab and vital sign abnormality is the body's alarm system trying to fix blood flow (perfusion) (oxygen delivery), clotting, or blood pressure. Recognizing the pattern tells you which upstream break caused the cascade—bleeding (hemorrhage), infection, with high blood pressure (hypertensive) injury, or a traveling clot lodging in a vessel (embolism).
7. Trace it backward: normal uterus contracts to clamp spiral arteries and stop bleeding, then atony or trauma or impaired blood clotting (coagulopathy) breaks that step, and the body's compensatory response fires in predictable order—a fast heart rate (tachycardia) first, low blood pressure (hypotension) and end-organ injury last. — why: Understanding the normal physiology first—uterus squeezes down (contract), blood clots seal the wound (clot), uterus shrinks back to normal size (involute)—lets you recognize when one link fails. If the uterus does not contract (atony), bleeding continues. If there is a tear (laceration), bleeding bypasses the uterine squeeze. If clotting factors are used up (coagulopathy), the blood cannot seal the wound. If bacteria grow in trapped blood and tissue (endometritis), infection spreads to the bloodstream (sepsis). If blood pressure stays dangerously high (severe preeclampsia/eclampsia), it damages brain, liver, kidneys, and lungs. If a clot travels to the lungs (pulmonary a traveling clot lodging in a vessel (embolism)), oxygen delivery fails. Each upstream break triggers a predictable downstream cascade: bleeding (hemorrhage) → a fast heart rate (tachycardia) → low blood pressure (hypotension) → shock. Infection → fever, a fast heart rate → septic shock. High blood pressure (Hypertension) → seizure, stroke, liver/kidney injury. A traveling clot lodging in a vessel → low oxygen in the blood (hypoxemia) → right heart failure → arrest. Seeing the dominoes fall in order lets you intervene at the right step—stop the bleeding with uterotonics or surgery, kill the bacteria with antibiotics, lower the blood pressure with antihypertensives and prevent seizures with magnesium, or dissolve the clot with anticoagulation or thrombolysis—before the last domino (multi-organ failure, cardiac arrest, death) tips over.

Spot it — what you see, and why

- Heavy vaginal bleeding that soaks one or more pads in an hour, or passing clots bigger than a golf ball: After the placenta detaches, the uterus normally squeezes down (contracts) hard, clamping shut the spiral arteries that fed the placenta—like closing a fist around open hoses. If the uterus stays soft and boggy (uterine atony) or pieces of placenta are stuck inside, those arteries stay open and keep bleeding freely from the raw spot where the placenta was attached.
- Fast heart rate—over 100 to 110 beats per minute—while blood pressure is still normal or just a little low: Your body notices it is losing blood, so it speeds up the heart and squeezes blood vessels in your skin and gut to keep enough blood flowing to your brain and vital organs. This early reflex buys time before blood pressure drops.
- Low blood pressure—top number under 90—and confusion, agitation, or loss of consciousness: Once you have lost more than about 1,500 milliliters of blood (roughly three soda bottles), your body's backup systems run out. Blood pressure falls because there is not enough blood left to fill the vessels, and your brain does not get the oxygen it needs to think clearly or stay awake.
- Fever of 100.4°F (38°C) or higher, smelly vaginal discharge after birth, and tenderness low in the belly: Bacteria growing inside the uterus (endometritis—infection of the uterine lining) release molecules that signal your immune system to turn up your body's thermostat, causing fever. The infected, inflamed tissue inside hurts when pressed.
- Severe headache, vision problems (blurry, flashing lights, or blind spots), and sharp pain under the right ribs: Very high blood pressure—top number 160 or higher, bottom 110 or higher—forces fluid into brain tissue (swelling) and squeezes blood vessels in the eyes, causing headache and vision trouble. Blood can also pool under the liver's capsule (the tight wrapper around the liver), stretching it and causing intense pain under the right ribs in HELLP syndrome.
- A full-body seizure (convulsions with stiff muscles then jerking movements) in a woman with high blood pressure after delivery: Eclampsia happens when severe high blood pressure causes brain swelling and squeezing of brain blood vessels. This lowers the seizure threshold—the brain becomes electrically unstable—and triggers a generalized seizure.

Red flags — act now

- Top blood pressure under 90 or a drop of 40 points or more from your usual, heart rate over 120, or confusion and altered mental status—these mean shock (the body's circulation is failing) and you need emergency IV fluids, blood products, and immediate treatment to find and stop the cause

- Vaginal bleeding over 500 milliliters in the first 24 hours after vaginal birth (or over 1,000 milliliters after cesarean), especially with a soft, boggy uterus you cannot feel at the belly button—this is bleeding (hemorrhage) from uterine atony or hidden bleeding and requires urgent uterine massage, medications to make the uterus contract (oxytocin, misoprostol, carboprost), and possibly a balloon inside the uterus, artery embolization, or emergency hysterectomy
- Blood pressure 160 over 110 or higher on two checks 15 minutes apart, plus severe headache, vision changes, or pain under the right ribs—this is preeclampsia with severe features or HELLP syndrome; you need IV magnesium sulfate to prevent seizures and fast-acting blood pressure medicine (labetalol, hydralazine, or nifedipine) right away
- A new seizure after delivery in a woman with high blood pressure—assume eclampsia until proven otherwise; give IV magnesium sulfate (4 to 6 grams over 15 minutes, then 1 to 2 grams per hour), protect the airway, lower blood pressure, and check for brain bleeding with imaging
- Sudden severe shortness of breath, chest pain, oxygen saturation under 90%, fast heart rate, and low blood pressure—think pulmonary embolism (a clot lodging in a lung artery) (clot in the lung); get an urgent CT scan of the chest arteries (CTPA) or ventilation-blood flow (perfusion) scan, start blood thinners immediately if suspicion is high, and consider clot-busting drugs if the clot is massive and blood pressure is crashing

Treat it — in this order

1. Bimanual uterine massage and IV oxytocin 10 units as slow IV push or IM injection, then continuous infusion of 10-40 units oxytocin in 1 liter normal saline or lactated Ringer's at 125-200 mL/hr → Oxytocin causes the smooth muscle of the uterus to contract powerfully, squeezing shut the spiral arteries (blood vessels that fed the placenta) at the placental implantation site and stopping bleeding (hemorrhage) at the source. Bimanual massage (one hand on the abdomen pressing the fundus, the other hand inside the vagina pushing the uterus upward) mechanically stimulates contraction. This is first-line therapy per ACOG 2017 guidelines for postpartum bleeding from uterine atony, the cause in 80% of cases.
2. IV tranexamic acid 1 gram infused over 10 minutes, given within 3 hours of bleeding onset → Tranexamic acid blocks plasmin (an enzyme that breaks down clots), so clots that form at bleeding sites stay intact and bleeding (hemorrhage) slows. The WOMAN trial (2017, published in Lancet) showed tranexamic acid reduces death from postpartum bleeding by one-third when given within 3 hours of bleeding onset. WHO and ACOG now recommend it for refractory bleeding (bleeding not controlled by uterotonic drugs like oxytocin).

3. Second-line uterotonic medications if oxytocin fails: methylergonovine 0.2 mg IM every 2-4 hours (avoid if with high blood pressure (hypertensive)), or misoprostol 800-1000 mcg rectally or sublingually, or carboprost 250 mcg IM every 15-90 minutes (max 8 doses, avoid if asthmatic) → Methylergonovine (an ergot alkaloid) causes sustained tonic uterine contraction by stimulating alpha-adrenergic and serotonin receptors on smooth muscle. Misoprostol (a prostaglandin E1 analog) causes strong uterine contractions and is absorbed well rectally even during shock. Carboprost (prostaglandin F2-alpha) is highly effective for atony but can cause bronchospasm in asthmatics and should be avoided if the patient has asthma. These drugs work through different pathways than oxytocin, so they can succeed when oxytocin alone does not stop bleeding.
4. Intrauterine balloon tamponade using a Bakri balloon or Foley catheter filled with 250-500 mL sterile saline → The inflated balloon presses against the entire inner surface of the uterus, creating direct pressure on bleeding vessels (like pressing a sponge against a bleeding wound) and buying time for uterotonics (medications that make the uterus contract) to work. Success rate is 80-90% for atony when medications alone fail. ACOG recommends balloon tamponade before moving to surgical interventions like uterine artery embolization or hysterectomy.
5. Massive transfusion protocol: packed red blood cells, fresh frozen plasma, and platelets in 1:1:1 ratio, guided by ongoing blood loss and coagulation labs; give crystalloid (lactated Ringer's or normal saline) IV to maintain blood pressure until blood products arrive → Packed red cells restore oxygen-carrying capacity by replacing lost hemoglobin. Fresh frozen plasma replaces clotting factors (proteins like fibrinogen, prothrombin, and factor V needed to form clots). Platelets provide the building blocks for clot formation. The 1:1:1 ratio prevents dilutional impaired blood clotting (coagulopathy) (clotting failure from replacing lost blood with fluids and red cells that lack clotting factors and platelets). Crystalloid restores intravascular volume (fluid inside blood vessels) to maintain cardiac output (amount of blood the heart pumps per minute) and prevent organs from failing due to low blood flow.

Your top priorities at the bedside

1. Establish two large-bore IV lines (18-gauge or larger) and draw blood for type and crossmatch, complete blood count, and coagulation studies if not already done. — why: Large IV catheters allow rapid infusion of crystalloid fluids (saline or lactated Ringer's), blood products, and medications to restore circulating volume quickly when bleeding (hemorrhage) causes hypovolemic shock (low blood volume), and typed blood must be ready for transfusion.

2. Perform fundal massage by placing one hand on the lower abdomen just above the pubic bone and the other hand cupping the uterine fundus, then massaging in a circular motion until the uterus feels firm. — why: Bimanual uterine massage stimulates the myometrium (uterine muscle) to contract and compress the spiral arteries (blood vessels inside the uterus) that are bleeding, which is the fastest non-medication way to reduce bleeding (hemorrhage) from uterine atony.
3. Administer IV oxytocin 10 units by slow IV push over 1 to 2 minutes or IM injection, then start a continuous infusion of 10 to 40 units oxytocin in 1 liter of normal saline or lactated Ringer's running at 125 to 200 mL per hour. — why: Oxytocin causes sustained uterine contraction by binding to receptors on smooth muscle cells in the uterus, reducing blood flow through the placental implantation site and stopping most cases of postpartum bleeding (hemorrhage) from atony.