

Postpartum emergency

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

After birth, the thick muscle wall of the uterus (called the myometrium) squeezes down hard. That squeezing compresses the spiral arteries (small blood vessels that fed the placenta during pregnancy) at the spot where the placenta was attached. The placenta separates and is pushed out. Then blood clotting proteins form a fibrin clot (like a scab) that seals the raw uterine lining (called the endometrium). Over the next 6 to 12 weeks, blood volume and blood pressure return to normal levels. This coordinated process—stopping bleeding (hemostasis) and shrinking the uterus back to pre-pregnancy size (involution)—is the normal postpartum state.

BODY'S JOB

If you do not know the normal job first, the abnormal findings—fast heart rate (over 100 bpm), low blood pressure (top number under 90), fever ($\geq 100.4^{\circ}\text{F}$ or 38.0°C), seizure, trouble breathing—seem like random facts instead of a chain reaction pointing back to one broken step. The normal job is: uterus squeezes, vessels clamp shut, placenta comes out, clot seals the wound, bleeding stops. Each downstream alarm (fast heart, low pressure, fever, low oxygen) tells you which upstream step broke.

1. Birth happens and the placenta separates from the uterine wall, leaving raw open blood vessels at the attachment site (like peeling off a sticker and seeing the sticky spot underneath).
2. The uterus muscle squeezes down tight like a fist, compressing those spiral arteries so blood cannot pour out (picture wrapping your hand around a garden hose and squeezing to stop the flow).
3. Clotting proteins in the blood form a fibrin clot over the raw surface, sealing the wound (like a scab forming over a scrape).
4. Over days, the uterus continues to shrink (involution), the clot organizes into scar tissue, and the endometrial lining heals.
5. Blood volume drops slowly from pregnancy levels (about 5 to 6 liters) back to normal (about 4 to 5 liters), and blood pressure returns to baseline over 6 to 12 weeks.
6. If every step works, bleeding stops within minutes, the uterus feels firm on exam, vital signs stay stable, and the mother goes home healthy.

What breaks

Usually one root cause explains all the abnormal findings together. The uterus does not squeeze (atony), or pieces of placenta stay stuck inside, or there is a tear in the birth canal, or the blood cannot clot, or infection spreads from the uterus into the bloodstream, or blood pressure stays dangerously high and damages organs, or a clot floats to the lungs and blocks oxygen. That upstream break triggers a predictable cascade you see at bedside.

CLINICAL TERM

Uterine atony (the uterus stays soft and does not contract, accounting for 70 to 80 percent of postpartum bleeding (hemorrhage)). Retained placental tissue blocking contraction or serving as a breeding ground for infection. Genital tract laceration (tear in cervix, vagina, or uterus). Impaired blood clotting (Coagulopathy) (blood clotting disorder from preeclampsia, placental abruption, amniotic fluid a traveling clot lodging in a vessel (embolism), disseminated intravascular coagulation, or inherited disorder). Endometritis or sepsis (infection in the uterus spreading to bloodstream and causing systemic inflammatory response). Preeclampsia with severe features or eclampsia (sustained blood pressure $\geq 160/110$ mmHg with end-organ damage: seizure, stroke, liver rupture, pulmonary edema (fluid flooding the lungs) meaning fluid in the lungs, acute kidney injury). Pulmonary embolism (a clot lodging in a lung artery) (blood clot blocking pulmonary artery, causing sudden low blood oxygen and obstructive shock). That upstream break triggers a predictable cascade you see at bedside: bleeding causes fast heart rate first, then low blood pressure late when young compensatory reserve is exhausted. Read all abnormal vitals and labs backward—they all point to one of these upstream failures.

How the body compensates

Vital signs can look reassuring while the body is compensating hard. A young, healthy pregnant woman has huge reserves—her blood volume increased 40 to 50 percent during pregnancy, her heart can pump faster and harder, and her blood vessels can squeeze tight to shunt blood away from skin and gut to protect the brain and heart. That means she can lose 1000 to 1500 mL of blood (20 to 30 percent of blood volume) and still have a normal blood pressure and only a mildly elevated heart rate. Your job is to see decompensation (the crash point when the body's backup systems fail) coming before it arrives. Watch the trend lines—serial hemoglobin dropping, heart rate climbing, blood pressure falling, urine output dropping—because once the tipping point hits, the window to reverse shock closes fast.

1. {'what': 'The body keeps heart rate, blood pressure, and urine output steady for a while by increasing how hard and fast the heart pumps (cardiac output) and squeezing peripheral blood vessels (vasoconstriction) to shunt blood away from skin, muscles, and gut toward the brain and heart.', 'why': 'Pressure sensors in the aorta and carotid arteries (baroreceptors) detect dropping blood volume and signal the brainstem to release adrenaline (epinephrine) and noradrenaline (norepinephrine). These hormones make the heart beat faster and stronger and squeeze arteries in the skin, gut, and kidneys. Blood pressure stays normal even though blood volume is falling. This is called compensated shock.'}

2. {'what': 'That compensated shock phase is the dangerous middle, because normal vital signs let the team miss how much blood or function has already been lost.', 'why': 'A patient can lose 1000 to 1500 mL of blood and still have a systolic blood pressure of 100 to 110 mmHg and a heart rate of 100 to 120 bpm. The textbook says 'low blood pressure (hypotension) is a late sign'—it means the body's reserves are nearly exhausted. By the time blood pressure drops, you are already behind. Do not wait for low blood pressure to act.'}
3. {'what': 'The decompensation point to watch for is: massive bleeding-driven (hemorrhagic) shock (>1500 mL blood loss, systolic BP <90, confusion or unresponsiveness), septic shock (hypotension despite IV fluid, lactate >4 mmol/L showing poor oxygen delivery to tissues), eclamptic seizure with brain bleeding (intracerebral bleeding (hemorrhage)), or cardiac arrest from massive pulmonary embolism (a clot lodging in a lung artery) (pulseless electrical activity on monitor).', 'why': 'Once the body's compensatory mechanisms are exhausted, vital signs crash suddenly. A fast heart rate (Tachycardia) jumps to >120 bpm, systolic pressure drops below 90 mmHg, the patient becomes confused or unconscious (altered mental status from poor brain blood flow (perfusion)), urine output stops (kidneys shutting down), and skin becomes cold and pale (vasoconstriction maxed out). This is decompensated shock—multi-organ failure is starting.'}
4. {'what': 'Each root cause needs protocol-driven resuscitation within minutes: massive transfusion protocol (packed red blood cells, fresh frozen plasma, platelets in 1:1:1 ratio) plus uterotonic drugs (oxytocin 10 units IM or 40 units in 1 L IV, misoprostol 800 to 1000 mcg rectally, carboprost 250 mcg IM, tranexamic acid 1 g IV within 3 hours) or surgical intervention (uterine artery embolization, B-Lynch suture, hysterectomy) for bleeding (hemorrhage); broad-spectrum IV antibiotics (clindamycin 900 mg plus gentamicin 5 mg/kg, or ampicillin-sulbactam 3 g, or piperacillin-tazobactam 3.375 g) plus vasopressors (norepinephrine to raise blood pressure) and source control (removal of infected tissue or drainage) for sepsis; magnesium sulfate (4 to 6 g IV load, then 1 to 2 g/hour infusion to prevent seizures) and antihypertensives (IV labetalol 20 mg or hydralazine 5 to 10 mg to lower blood pressure below 160/110) for eclampsia; and anticoagulation (heparin or low-molecular-weight heparin) or thrombolysis (tissue plasminogen activator, tPA, to dissolve the clot) or surgical embolectomy (clot removal) for massive pulmonary embolism (a clot lodging in a lung artery).', 'why': 'Minutes matter. Bleeding-driven (Hemorrhagic) shock kills by taking away oxygen-carrying red blood cells; give blood products and stop the bleeding. Septic shock kills by widespread blood vessel leakage and toxin release; give antibiotics within 1 hour and squeeze vessels with vasopressors. Eclampsia kills by seizure and stroke; give magnesium to stop seizures and lower blood pressure fast. Pulmonary embolism (a clot lodging in a lung artery) kills by blocking oxygen pickup and right heart failure; give anticoagulation or break up the clot. Each has a different fix—identify which domino fell first.'}

5. {'what': 'Watch trend lines—serial hemoglobin (oxygen-carrying protein in red blood cells), heart rate rising, systolic pressure falling, urine output dropping—because once the tipping point hits, the window to reverse shock closes fast.', 'why': 'A single hemoglobin of 10 g/dL might be okay if it is stable, but if it was 12 g/dL an hour ago and 11 g/dL 30 minutes ago, the trend is dropping 2 g/dL per hour—that means active bleeding. A heart rate of 110 bpm might be normal postpartum, but if it was 90 bpm an hour ago and 100 bpm 30 minutes ago, the trend is climbing—that means the body is compensating for falling blood volume or rising infection. Plot the numbers over time, not just one snapshot.'}
6. {'what': 'Look at trajectory (where the patient is heading), not just a single snapshot.', 'why': 'A patient with stable vital signs right now but hemoglobin dropping 2 g/dL per hour, heart rate climbing 10 bpm every 15 minutes, and urine output falling from 30 mL/hour to 10 mL/hour is not stable—she is decompensating. Intervene early while compensatory mechanisms are still working, not after they fail.'}
7. {'what': 'A stable patient on a downhill slope—hemoglobin dropping 2 g/dL per hour, heart rate climbing 10 bpm every 15 minutes—is not really stable.', 'why': 'Stability is not one good number; it is a flat trend line. A downhill slope means the underlying problem is not fixed. Call for help, activate massive transfusion protocol or sepsis protocol, and prepare for the crash before it happens.'}

Domino effect

1. Uterine atony or retained placenta or genital tract laceration allows continuous bleeding from open spiral arteries at the placental implantation site. — 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. — 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. — 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/ μL), pulmonary edema (fluid flooding the lungs) (fluid in the lungs), and acute kidney injury. — 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/ μL). (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. — 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 PaO₂ (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. — 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. — 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.

Symptoms · what the patient feels, 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.
- Sudden trouble breathing, sharp chest pain that gets worse when you take a deep breath, fast breathing (over 20 breaths per minute), and low oxygen (less than 94% on room air): A blood clot travels to the lung and blocks a branch of the pulmonary artery (pulmonary a traveling clot lodging in a vessel (embolism)). Air still reaches the tiny air sacs, but no blood flows past them to pick up oxygen—like blowing into a balloon that is not connected to anything. This causes low oxygen, and the inflamed lung lining triggers sharp pain with each breath.
- Very little urine—less than 30 milliliters per hour or half a milliliter per kilogram of body weight per hour—and rising creatinine (a waste product measured in blood): If blood loss or infection drops blood pressure, the kidneys do not get enough pressure to filter blood properly (prerenal acute kidney injury—kidney damage from low blood flow). In severe preeclampsia, high blood pressure and other factors directly injure the tiny kidney filters (glomeruli). Either way, the kidneys make less urine and waste products build up.

Focused exam

- Vaginal bleeding soaking through one pad in 15 minutes or less, or measured blood loss above 500 mL after vaginal delivery or 1000 mL after cesarean: After the baby is born, the uterus (womb) must squeeze tight to close the blood vessels where the placenta (the organ that fed the baby during pregnancy) was attached. When the uterus stays soft and does not squeeze—a problem called uterine atony (uterus failing to contract)—blood pours out fast. Uterine atony causes 80% of all postpartum bleeding (hemorrhage) (dangerous bleeding after delivery that can kill if not stopped quickly).

- Heart rate staying above 110 beats per minute: When blood volume drops from heavy bleeding, the heart speeds up to keep oxygen flowing to the brain and vital organs. A fast heart rate—called a fast heart rate (tachycardia)—is often the first warning sign of bleeding-driven (hemorrhagic) shock (life-threatening state from blood loss), because blood pressure may still look normal early on. The body compensates by speeding the heart before blood pressure crashes.
- Blood pressure dropping below 90 mmHg systolic (top number) or falling with each measurement: Low blood pressure—called low blood pressure (hypotension)—is a late sign of shock. By the time systolic pressure (the top number) falls below 90 mmHg, the body has lost its ability to make up for blood loss. Shock is already present and organs may be starting to fail from lack of oxygen. This is decompensated shock (the body's defenses are overwhelmed).
- Fever above 38°C (100.4°F) with lower belly tenderness or foul-smelling lochia (vaginal discharge after birth): Fever with uterine tenderness or bad-smelling discharge suggests endometritis (infection of the uterine lining), often from retained placental fragments (pieces of placenta left inside the uterus) or bacteria entering through vaginal or cervical lacerations (tears in the birth canal). Infection can lead to septic shock (life-threatening drop in blood pressure from bacteria releasing toxins into the bloodstream).
- Blood pressure above 160/110 mmHg with severe headache, vision changes, right upper quadrant pain (pain under the ribs on the right side), or confusion: Very high blood pressure after delivery—called severe high blood pressure (hypertension)—can signal postpartum preeclampsia (high blood pressure with organ damage after delivery) or HELLP syndrome (a life-threatening variant where red blood cells break apart, liver enzymes rise, and platelet count drops). Without treatment, this can cause eclampsia (seizures from brain swelling), stroke (bleeding or blockage in the brain), or liver rupture.
- Sudden shortness of breath, oxygen saturation below 92% on room air, or sharp chest pain: These signs point to pulmonary embolism (a clot lodging in a lung artery) (blood clot blocking an artery in the lungs), which cuts off oxygen delivery and can cause the heart to fail rapidly. Pregnancy and the postpartum period increase clot risk because blood clots more easily to prevent bleeding (hemorrhage) during delivery. PE is a leading cause of maternal death in the postpartum period.
- Uterus feels soft and boggy like a sponge instead of firm, or very tender when pressed at the fundus (top of the uterus): A soft, boggy uterus means uterine atony—the uterus is not contracting properly after delivery. The uterus should feel firm like a grapefruit at or just below the level of the belly button right after delivery. If the uterus is firm but very tender or larger than expected, suspect retained placental tissue (pieces of placenta still inside preventing full contraction).

Diagnostics · every test answers a question

- Complete blood count (CBC) with hemoglobin and hematocrit → Is the blood count dropping from bleeding (hemorrhage)? How much blood has been lost?
- Coagulation panel: fibrinogen level, prothrombin time/INR, partial thromboplastin time (PTT) → Is the clotting system failing? Can the body form clots to stop bleeding?
- Type and crossmatch (blood typing and antibody screen) → Do we have matched blood ready to transfuse safely?
- Bedside ultrasound of the uterus (transabdominal or transvaginal) → Is there retained placental tissue or blood clot in the uterus causing continued bleeding?
- CT pulmonary angiography (CTPA) with IV contrast → Is there a pulmonary embolism (a clot lodging in a lung artery) blocking blood flow in the lungs?
- Liver enzymes (AST, ALT), platelet count, lactate dehydrogenase (LDH), and peripheral blood smear → Is there HELLP syndrome or severe preeclampsia causing organ damage?
- Arterial blood gas (ABG) and serum lactate → Is shock causing lactic acidosis (acid from oxygen-starved tissues) from poor oxygen delivery to tissues?
- Blood cultures (two sets from separate sites) before starting antibiotics → Is bacteria in the bloodstream causing sepsis?

Treatment logic

- 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.
- 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).

- 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.
- 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.
- 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.
- Broad-spectrum IV antibiotics started within 1 hour of sepsis recognition: gentamicin 5 mg/kg once daily plus clindamycin 900 mg every 8 hours, OR ampicillin-sulbactam 3 g every 6 hours, OR piperacillin-tazobactam 3.375 g every 6 hours (per ACOG endometritis guidelines and Surviving Sepsis Campaign) → Antibiotics kill aerobic and anaerobic bacteria in the uterus and bloodstream, stopping the release of endotoxins (bacterial toxins that cause blood vessels to open too wide and blood pressure to crash). Regimen must cover common postpartum pathogens: *E. coli*, Group A and B *Streptococcus*, *Enterococcus*, anaerobes like *Bacteroides*, and rarely *Clostridium* or *Staphylococcus aureus*. Early antibiotics reduce mortality in septic shock by stopping infection from progressing to multi-organ failure.

- Acute blood pressure control with IV labetalol 20 mg initial dose, then 20-80 mg every 10 minutes (max 300 mg total), OR IV hydralazine 5-10 mg every 20 minutes (max 30 mg), to keep systolic BP below 160 and diastolic below 110; PLUS IV magnesium sulfate 4-6 g loading dose over 15-20 minutes, then 1-2 g/hr continuous infusion → Labetalol (an alpha and beta blocker) and hydralazine (a direct vasodilator that relaxes arterial smooth muscle) lower blood pressure acutely to prevent stroke, which is the leading cause of death in severe high blood pressure (hypertension) during pregnancy and postpartum. ACOG 2019 high blood pressure in pregnancy guidelines require treatment within 30-60 minutes when blood pressure is 160/110 or higher. Magnesium sulfate raises the seizure threshold in the brain by blocking calcium channels in neurons, preventing eclamptic seizures (seizures from brain irritation caused by severe preeclampsia). Magnesium reduces seizure risk by 50% compared to no treatment.

Medications

Oxytocin · Uterotonic (makes the uterus squeeze)

Oxytocin is a copy of the body's natural hormone that tells the uterus muscle to squeeze tight. After delivery, the uterus must clamp down hard to pinch shut the open blood vessels where the placenta was attached (like closing a fist around garden hoses). If the muscle stays soft and floppy (called atony, meaning no tone), blood pours out. Oxytocin grabs special docking sites (receptors) on the muscle cells that line the uterus and flips the switch for the muscle to contract.

1. Oxytocin attaches to oxytocin receptors on the surface of uterine smooth muscle cells.
2. This binding triggers calcium ions to flood into the cell.
3. Calcium causes the muscle fibers inside to slide past each other and tighten (contract).
4. The whole uterus shrinks down, squeezing the open blood vessels shut and stopping the bleeding.

SAFETY WATCH

Giving too much too fast through the vein can briefly drop blood pressure and cause fast heart rate; run it slowly. · Prolonged high doses can cause the body to hold onto too much water (water intoxication) because oxytocin is chemically similar to a hormone that controls water balance. · Very rarely, can trigger abnormal heart rhythms if given as a rapid IV push.

Methylergonovine · Ergot alkaloid uterotonic

Methylergonovine makes uterine muscle squeeze by acting on different docking sites than oxytocin (serotonin and alpha receptors on the muscle cells). It causes a long, sustained

squeeze rather than rhythmic contractions. The muscle stays tight for a longer time, which helps keep blood vessels compressed. Because it also tightens blood vessels everywhere in the body, it can dangerously raise blood pressure.

1. Methylergonovine binds to serotonin and alpha-adrenergic receptors on uterine muscle cells.
2. This binding causes sustained (tonic) contraction of the muscle—the uterus clamps down and stays tight.
3. The prolonged squeeze keeps the blood vessels where the placenta was attached pinched shut.
4. At the same time, it also constricts blood vessels all over, including in the brain and heart.

SAFETY WATCH

Never give if blood pressure is high ($\geq 140/90$) or if the woman has preeclampsia or heart disease—can cause stroke, seizure, or heart attack. · Can rarely cause coronary artery spasm (sudden closing of heart arteries), leading to chest pain or heart attack. · Nausea, vomiting, and headache are common. · Avoid in women with peripheral vascular disease (poor circulation in arms or legs).

Carboprost · Prostaglandin F₂-alpha analog uterotonic

Carboprost is a man-made copy of a natural body chemical (prostaglandin) that strongly squeezes smooth muscle. It grabs prostaglandin receptors on uterine muscle cells and tells them to contract hard. It also tightens smooth muscle in the lungs (which control airway size), so it can dangerously narrow airways in women with asthma.

1. Carboprost attaches to prostaglandin F receptors on uterine muscle cells.
2. This activates a chain reaction inside the cell that raises calcium levels.
3. Calcium makes the muscle fibers contract forcefully.
4. The uterus squeezes down, compressing open blood vessels and slowing the bleeding (hemorrhage).

SAFETY WATCH

Never give to a woman with asthma (active or history)—can trigger severe, life-threatening bronchospasm (airway narrowing). · Very common side effects: diarrhea, nausea, vomiting, fever, flushing. · Can rarely raise blood pressure or cause low oxygen if airways tighten. · Use cautiously in women with heart, liver, or kidney disease.

Misoprostol · Prostaglandin E1 analog uterotonic

Misoprostol is a prostaglandin pill (or tablet placed under the tongue or in the rectum) that tells the uterus muscle to contract. It is slower and less powerful than the injected drugs but is cheap, stable at room temperature, and easy to give. It binds prostaglandin E receptors on uterine smooth muscle and triggers contraction. Because it can be given by mouth or rectum, it is useful in low-resource settings or when IV access is hard.

1. Misoprostol is absorbed through the lining of the mouth, stomach, or rectum into the bloodstream.
2. It binds prostaglandin E receptors on uterine muscle cells.
3. This binding raises calcium inside the cells, causing muscle contraction.
4. The uterus tightens and blood flow from open vessels slows.

SAFETY WATCH

Very common: shivering (rigors), fever, diarrhea. · Usually well tolerated; no absolute contraindications in postpartum bleeding (hemorrhage) emergencies. · Fever can be confused with infection; reassure the patient it is a drug side effect. · Avoid in women with known allergy to prostaglandins (rare).

Tranexamic acid · Antifibrinolytic (stops clot breakdown)

When you bleed, your body first forms a clot (a plug made of platelets and fibrin protein threads). Then, to clean up later, your body makes an enzyme called plasmin that dissolves the clot. In postpartum bleeding (hemorrhage), too much plasmin can break down clots before bleeding stops, so blood keeps pouring out. Tranexamic acid blocks plasmin from attacking the clot, letting the plugs stay in place longer and giving the uterus time to squeeze shut and heal.

1. Tranexamic acid enters the bloodstream (given IV or by mouth).
2. It attaches to plasminogen (the inactive form of plasmin) and prevents it from turning into active plasmin.
3. It also binds to plasmin itself and blocks the enzyme from cutting fibrin threads in the clot.
4. Existing clots stay stable and intact, and bleeding slows or stops.

SAFETY WATCH

Must be given within 3 hours of delivery to save lives (the WOMAN trial proved this timing). · Rarely, can cause nausea or dizziness. · Theoretical risk of increasing dangerous clots (like pulmonary embolism (a clot lodging in a lung artery) or stroke), but large studies have NOT found this in postpartum women when given at the right dose. · Avoid in women with active clotting disorders (thrombophilia) or history of blood clots unless benefit outweighs risk.

Magnesium sulfate · Anticonvulsant and smooth muscle relaxant

Magnesium is a mineral that calms down overexcited nerve cells in the brain. In preeclampsia or eclampsia, the brain becomes irritable (like a circuit overloaded with electricity) and can fire off a seizure. Magnesium blocks calcium from entering nerve cells; without calcium, the nerve cannot send the seizure signal. It also relaxes blood vessel walls slightly, which can help lower very high blood pressure. It does NOT treat bleeding (hemorrhage) itself but prevents life-threatening seizures in women with severe preeclampsia or eclampsia during or after delivery.

1. Magnesium sulfate is given IV and floods the bloodstream.
2. It crosses into the brain and competes with calcium at nerve cell receptors (NMDA receptors).
3. By blocking calcium channels, magnesium keeps neurons from firing uncontrollably.
4. The seizure threshold rises—meaning it takes much more stimulation to trigger a seizure.
5. At the same time, magnesium relaxes blood vessel smooth muscle, which can lower blood pressure a little.

SAFETY WATCH

Too much magnesium (toxicity) makes muscles weak and can stop breathing—monitor reflexes (knee jerk) and breathing rate every hour. · Keep calcium gluconate at the bedside as the antidote if toxicity occurs. · Can cause flushing, warmth, nausea. · Reduce dose if kidney function is poor (kidneys clear magnesium). · High levels can rarely cause the heart to stop (cardiac arrest)—follow strict dosing protocols.

Labetalol · Combined alpha and beta blocker (blood pressure lowering)

Labetalol blocks two kinds of docking sites (receptors) on blood vessel walls and the heart. By blocking beta receptors, it slows the heart and makes it pump less forcefully. By blocking alpha receptors, it relaxes the smooth muscle in artery walls, letting the vessels widen. Together,

these actions lower blood pressure quickly. In postpartum women with severe high blood pressure (hypertension) ($\geq 160/110$), this prevents stroke, seizure, and heart failure.

1. Labetalol is given IV and spreads through the bloodstream.
2. It blocks alpha-1 receptors on artery walls, which stops the signal to squeeze tight; the vessels relax and widen.
3. It also blocks beta-1 receptors on heart muscle, which slows the heart rate and reduces how hard the heart pumps.
4. Blood pressure falls as the heart does less work and the pipes (arteries) open wider.

SAFETY WATCH

Do not give to women with asthma or severe chronic lung disease (beta blockers can narrow airways). · Can mask signs of low blood sugar in diabetic women (beta blockade hides the racing heart that warns of low blood sugar (hypoglycemia)). · Can cause dizziness, nausea, tingling scalp. · Rarely, severe drop in blood pressure or slow heart rate—monitor closely and give IV fluids if needed. · Avoid in women with heart block or severe heart failure.

Hydralazine · Direct-acting vasodilator (blood vessel relaxer)

Hydralazine works directly on the smooth muscle inside artery walls (not through receptors like most drugs). It opens potassium channels in the muscle cells, which causes calcium to leave and the muscle to relax. When arteries relax, they widen and blood pressure drops. Hydralazine is given IV or IM for severe postpartum high blood pressure (hypertension) ($\geq 160/110$) to prevent stroke and organ damage. It is especially useful if labetalol cannot be used (for example, in a woman with asthma).

1. Hydralazine enters the artery wall smooth muscle cells.
2. It opens potassium channels, letting potassium ions leak out of the cell.
3. This causes calcium channels to close and calcium to leave the cell.
4. Without calcium, the muscle fibers cannot stay tight—they relax.
5. The artery widens and blood pressure falls.

SAFETY WATCH

Can cause fast heart rate (tachycardia) and pounding headache as blood pressure drops. · May trigger chest pain (angina) in women with heart disease because the heart works harder when vessels suddenly dilate. · Nausea, vomiting, flushing are common. · Very rarely, can cause a lupus-like syndrome with long-term use (not usually a concern in acute postpartum use). · Blood pressure can drop too low—give slowly and check BP every 5–10 minutes.

Heparin (unfractionated) or enoxaparin (low-molecular-weight heparin) · Anticoagulant (prevents blood clots from growing)

After delivery, some women develop blood clots in the legs (deep vein clot formation (thrombosis)) or lungs (pulmonary a traveling clot lodging in a vessel (embolism)), especially if they had a cesarean section, are overweight, or stayed in bed a long time. Heparin is a sugar molecule that turbocharges a natural anticlotting protein in your blood called antithrombin. Antithrombin usually works slowly to shut down clotting enzymes; heparin makes it work thousands of times faster. This stops existing clots from getting bigger and prevents new clots from forming, giving your body time to dissolve the clot naturally. It does NOT melt the clot directly.

1. Heparin (given IV or under the skin) binds to antithrombin in the blood.
2. This binding changes the shape of antithrombin, making it much more active.
3. Supercharged antithrombin grabs and shuts down thrombin (the enzyme that makes clots) and Factor Xa (an earlier step in the clotting chain).
4. New clot formation stops; existing clots do not grow bigger.
5. The body's natural clot-dissolving system (fibrinolysis) can now catch up and slowly break down the clot.

SAFETY WATCH

Major risk: bleeding—watch for heavy vaginal bleeding, blood in urine or stool, or bleeding at IV sites. · Rarely, can cause heparin-induced a low platelet count (thrombocytopenia) (HIT)—a dangerous drop in platelets that paradoxically causes clots; check platelet count if on heparin longer than 5 days. · Enoxaparin (low-molecular-weight heparin) is easier to dose and does not require as much blood monitoring. · Reverse with protamine sulfate if life-threatening bleeding occurs. · Safe during breastfeeding (does not pass into milk).

Red flags

- 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

Nursing priorities

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. — 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. — 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. — 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.

4. Measure vital signs every 5 to 15 minutes, watching for systolic blood pressure under 90, heart rate over 120, or any confusion or decreased responsiveness. — A fast heart rate (Tachycardia) (fast heart rate) is the body's first compensation for blood loss, followed by low blood pressure (hypotension) (low blood pressure) and altered mental status when shock develops, signaling the need for immediate blood transfusion and possible surgical intervention.
5. Quantify blood loss by weighing blood-soaked pads and linens, using the formula that 1 gram equals 1 milliliter of blood, and report cumulative totals to the provider every 15 minutes. — Visual estimation underestimates blood loss by 30 to 50 percent, so objective measurement triggers earlier intervention when loss reaches 500 mL for vaginal birth or 1,000 mL for cesarean birth, preventing progression to bleeding-driven (hemorrhagic) shock.
6. Give IV tranexamic acid 1 gram diluted in 10 mL normal saline or sterile water, infused over 10 minutes, within 3 hours of bleeding onset if bleeding (hemorrhage) continues despite oxytocin. — Tranexamic acid blocks plasmin (an enzyme that breaks down clots), stabilizing fibrin clots at the bleeding site and reducing mortality from postpartum bleeding (hemorrhage) by 19 percent when given early.
7. Prepare second-line uterotonic medications and have them at the bedside: methylergonovine 0.2 mg IM every 2 to 4 hours (check blood pressure first, avoid if with high blood pressure (hypertensive)), or carboprost 250 micrograms IM every 15 to 90 minutes up to 8 doses (avoid if patient has asthma). — When oxytocin fails to control bleeding, methylergonovine causes sustained vasoconstriction (narrowing of blood vessels) and uterine contraction, while carboprost triggers strong myometrial contractions through prostaglandin receptors, but each has contraindications (reasons not to use) that must be checked.
8. Insert a Foley catheter to empty the bladder and monitor urine output hourly, keeping output above 30 mL per hour. — A full bladder prevents the uterus from contracting effectively and worsens atony, while urine output below 30 mL per hour signals poor kidney blood flow (perfusion) from hypovolemia and guides fluid resuscitation needs.

Sources

- ACOG Practice Bulletin No. 183: Postpartum Hemorrhage (2017, reaffirmed 2023)
- ACOG Practice Bulletin No. 222: Gestational Hypertension and Preeclampsia (2020, reaffirmed 2022)
- American College of Chest Physicians Antithrombotic Therapy for VTE Disease: CHEST Guideline (2021)
- CDC Pregnancy Mortality Surveillance System (2023)

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