

Hemoptysis

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THE STORY IN ONE SENTENCE

Blood is coming from the lower airway — the breathing tubes and lungs — not the nose, mouth, or throat.

“Coughing up blood (Hemoptysis) means blood from the lungs, and how much blood tells you how fast to act.”

Normal anatomy — the body's job

The lungs have two separate blood supplies doing two different jobs, and understanding both is the key to knowing why big bleeds happen.

BODY'S JOB

The pulmonary arteries carry dark, oxygen-poor blood from the right side of the heart into the lungs at low pressure (around 15-25 mmHg), so the blood can pick up oxygen in the alveoli (the tiny air sacs) and return bright red to the left heart. The bronchial arteries branch off the aorta and deliver oxygen-rich blood at high pressure (the same pressure as the rest of your body, around 120/80 mmHg) to feed the walls of the airways, the pleura (lung lining), and the lymph nodes. Normally, the bronchial arteries are small — less than 2 mm wide — and quiet. The airways are lined with cilia (tiny waving hairs) and mucus that trap dust and germs, and you cough or swallow small amounts of mucus every day without noticing. The alveoli are so thin that oxygen and carbon dioxide can jump across in a split second, and the lungs are always slightly moist inside to let that gas swap happen smoothly.

1. Right heart pumps dark blood → pulmonary arteries (low pressure, ~15-25 mmHg) → capillaries wrap around alveoli → oxygen jumps in, carbon dioxide jumps out → blood turns bright red → pulmonary veins → left heart → body.
2. Aorta sends a small branch → bronchial arteries (high pressure, ~120/80 mmHg) → feed airway walls, pleura, lymph nodes → drain into pulmonary veins or azygos vein → back to heart.

3. Airways (trachea → bronchi → bronchioles → alveoli) stay open and clear by constant mucus flow and cilia sweeping upward, plus the cough reflex clears anything extra.
4. Alveoli (300 million tiny air sacs, each wrapped in capillaries) stay dry and thin so oxygen and CO₂ can cross in milliseconds — any extra fluid is pulled away by lymph vessels running through the lung.

What breaks

A blood vessel in or around the lung cracks open and pours blood into the breathing tubes, blocking air from moving and threatening the airway.

CLINICAL TERM

Coughing up blood (Hemoptysis) arises when a vessel — most often a bronchial artery (high-pressure, systemic circulation) or less commonly a pulmonary artery or capillary (low-pressure, pulmonary circulation) — erodes or ruptures and bleeds into the tracheobronchial tree or alveolar space. The bleeding source and volume determine urgency: massive coughing up blood (≥ 100 – 200 mL in 24 hours by most definitions, though some use ≥ 500 mL or any amount threatening the airway) carries high mortality from asphyxiation, not exsanguination. The downstream effect you see at the bedside is blood filling airways, triggering cough, impairing gas exchange, and risking airway blockage (occlusion). Read every sign — blood-tinged coughed-up mucus (sputum), frothy pink secretions, low oxygen in the blood (hypoxemia), unilateral crackles, fast breathing (tachypnea) — backwards from that one upstream break.

How the body compensates

The body works hard to keep oxygen delivery going and airways open, so vital signs can look deceptively stable even while the patient is sliding toward airway collapse and respiratory failure. Your job is to see the crash coming before it arrives.

1. {'what': 'The body raises heart rate and squeezes blood vessels (vasoconstriction).', 'why': 'To push whatever oxygen-carrying blood is left to the brain and heart, buying time while the lungs struggle. A patient coughing up blood may have a heart rate of 110–120 and high blood pressure at first — that is compensation, not stability.'}
2. {'what': 'Breathing rate climbs (tachypnea) and the patient sits upright, leaning forward.', 'why': 'To pull in more air and try to clear the airway by coughing. The upright position uses gravity to drain blood away from the main airways. Lying flat can flood both lungs and cause sudden asphyxiation.'}

3. {'what': 'The patient coughs repeatedly, often bringing up bright red or dark clotted blood mixed with frothy pink coughed-up mucus (sputum).', 'why': 'Coughing is the body's only way to clear blood from the airways. Frothy pink coughed-up mucus (sputum) means blood is mixing with air and mucus — a sign it is coming from deep in the lungs (alveoli or small airways), not the throat.'}
4. {'what': 'Oxygen saturation may stay near 90–94% for a while, even as bleeding continues.', 'why': 'Unaffected lung regions compensate by pulling in more oxygen, and the body shunts blood away from flooded areas (hypoxic vasoconstriction). But this is the dangerous middle — it hides how close the patient is to full airway blockage (occlusion). Once the good lung is overwhelmed or a large clot blocks the mainstem bronchus, saturation drops fast and does not come back up with simple oxygen.'}
5. {'what': 'Skin may stay warm and pink initially, then turn pale, cool, and sweaty as compensation fails.', 'why': 'Early, the body keeps blood pressure and blood flow (perfusion) stable. As blood loss continues or the airway clogs, cardiac output falls, oxygen delivery drops, and the sympathetic nervous system (fight-or-flight) floods the body with adrenaline — causing cold sweat, pale skin, and anxiety.'}
6. {'what': 'The crash point to watch for is: cannot protect the airway, oxygen saturation <90% despite high-flow oxygen, altered mental status, or hemodynamic instability (systolic BP <90, heart rate >120).', 'why': 'These signs mean compensation has failed. The patient is drowning in blood, the brain is not getting enough oxygen, or the heart cannot keep up. This is the point of no return without immediate intubation, airway suctioning, and intervention (bronchoscopy, interventional radiology, or surgery).'}- 7. {'what': 'Massive coughing up blood (hemoptysis) (≥ 100 –200 mL in 24 hours, or any amount threatening the airway) is a true emergency — mortality reaches 50–80% if the airway is not secured and the bleeding source not controlled within hours.', 'why': 'Death comes from asphyxiation (drowning in blood), not from bleeding out. The body has 5 liters of blood and can survive losing 1–2 liters, but it cannot survive even 200 mL of blood filling the airways and blocking oxygen.'}
- 8. {'what': 'Watch the trajectory, not just the snapshot.', 'why': 'A patient who coughed up 50 mL an hour ago and is now coughing up 150 mL is on a steep downhill slope, even if vital signs are still borderline normal. A stable patient on a worsening trajectory is not truly stable — you are watching compensation run out in real time.'}
- 9. {'what': 'If the bleeding side is known (from imaging or history), position the patient with the bleeding side down.', 'why': 'Gravity keeps blood from spilling into the good lung, protecting at least one side for gas exchange. Lying bleeding-side up can flood the healthy lung and cause sudden respiratory arrest.'}

Domino effect

1. A blood vessel wall in or near the lung weakens and cracks open. — Infection (TB, pneumonia), chronic inflammation (bronchiectasis), tumor invasion, autoimmune attack (GPA, Goodpasture), or a congenital weak spot (AVM) erodes the vessel wall. Bronchial arteries — fed by the aorta at high pressure (120/80 mmHg) — are the most common source of massive bleeding because even a small tear releases a lot of blood fast.
2. Blood pours into the airways (trachea, bronchi, bronchioles) or alveoli (air sacs). — The cracked vessel empties directly into the breathing space. If the bleed is from a bronchial artery or large pulmonary vessel, blood fills the airways like water filling a straw. If it is from alveolar capillaries (diffuse alveolar bleeding (hemorrhage)), blood floods the air sacs where oxygen is supposed to cross into the bloodstream.
3. Blood blocks air from moving down the airways, and oxygen cannot reach the alveoli. — Blood clots fast when it hits air, forming plugs in the bronchi and bronchioles. Even liquid blood coats the airway walls and pools in low spots, creating a physical barrier. The alveoli downstream of a blocked airway collapse (atelectasis), and any alveoli filled with blood cannot exchange oxygen or carbon dioxide — they become dead space.
4. Oxygen levels in the blood drop (hypoxemia), and the brain senses danger. — Chemoreceptors in the carotid arteries and brainstem detect falling O₂ and rising CO₂. The brainstem (medulla oblongata) immediately signals the body to breathe faster and the heart to beat faster, trying to push whatever oxygen is left to the brain, heart, and kidneys.
5. The patient coughs hard and repeatedly, bringing up bright red or dark blood mixed with frothy coughed-up mucus (sputum), and breathes fast (tachypnea) and shallow. — Coughing is the only reflex the body has to clear the airway. Irritant receptors in the bronchi fire, triggering the cough center in the medulla. Fast, shallow breathing (instead of slow, deep breaths) happens because the patient is trying to avoid triggering more coughing and because the work of breathing through narrowed, blood-filled airways is exhausting.
6. The danger is not bleeding to death — the danger is drowning in your own blood before your body runs out of volume. — A person has about 5 liters of blood and can survive losing 1–2 liters over time (the body compensates by squeezing vessels and making the heart beat faster). But if even 200–300 mL of blood fills the airways faster than it can be coughed out, it blocks the windpipe (trachea) or mainstem bronchi, cutting off all airflow. The patient cannot breathe in or out, oxygen saturation drops to zero in seconds, the heart stops from lack of oxygen (hypoxic cardiac arrest), and death follows in minutes — long before the body runs out of blood.

7. The body tries to survive by raising heart rate, squeezing blood vessels, and shunting blood away from bleeding areas, but these are compensations — not the sickness itself. — A fast heart rate (Tachycardia), high blood pressure, and pale, sweaty skin are the sympathetic nervous system (fight-or-flight) flooding the body with adrenaline (epinephrine) and noradrenaline (norepinephrine) to keep the brain and heart alive. Oxygen-starved (Hypoxic) vasoconstriction (blood vessels in low-oxygen lung areas squeeze shut) redirects blood to healthier parts of the lung. These responses buy time, but they are not fixing the real problem — the bleeding vessel and the blocked airway.
8. Every clinical sign — cough, frothy pink or bright red blood, low oxygen, unilateral crackles or wheezing, anxious appearance, sitting upright and leaning forward — traces back to the same root cause: a vessel leaking into the lung, which blocks oxygen from moving through the airway. — The history and physical exam always point upstream to the break. Bright red, frothy blood = bronchial artery or large airway source; pink, frothy coughed-up mucus (sputum) that looks like foam = alveolar bleeding (hemorrhage) (blood mixed with surfactant and air in the air sacs); unilateral crackles or decreased breath sounds = blood or clot blocking one side; fever and night sweats = TB or infection; weight loss and smoking history = cancer; recent PE or leg swelling = pulmonary embolism (a clot lodging in a lung artery) with tissue death from blocked blood flow (infarction); sinusitis and kidney failure = GPA or Goodpasture. Every clue is a breadcrumb leading back to which vessel broke and why.

Symptoms · what the patient feels, and why

- Coughing up blood that is bright red, frothy, and mixed with air bubbles: Blood comes from the lungs or airways (the breathing tubes). When you cough it up, air mixes with it and makes it frothy and bright red, not dark like vomit. There is no food in it because it never went through the stomach.
- Shortness of breath or feeling like you cannot get enough air: Blood in the airways blocks air from flowing smoothly into the lung tissue. Also, if a lot of blood fills the air sacs (the tiny balloons where oxygen moves into your blood), less oxygen can get through.
- Cough that keeps happening or gets worse: Your airway is irritated by the blood itself, or by whatever is causing the bleeding (like infection, inflammation, or a growth). The cough reflex tries to clear the blood out.
- Chest pain on one side, especially when you breathe in deeply: The lining around the lung (the pleura) or the lung tissue itself may be inflamed or damaged. Breathing stretches that sore area and causes sharp pain.
- Fever and chills: If an infection (like pneumonia or tuberculosis) is the cause, your immune system raises your body temperature to fight the germs. Chills happen as your body resets its thermostat higher.

- Weight loss you did not try for, or feeling very tired all the time: Chronic diseases (like tuberculosis or lung cancer) drain your energy and make your body burn through its own stores. This leads to weight dropping and constant fatigue.
- Fast heart rate or feeling your heart pounding: When you lose blood or your oxygen level drops, your heart pumps faster to try to deliver enough oxygen to your brain and other vital organs.
- Wheezing or a whistling sound when you breathe: Blood, mucus, or swelling narrows the airways. Air squeezing through the narrow space makes a high-pitched whistle, just like blowing through a partly blocked straw.

Focused exam

- Blood in coughed-up mucus (sputum) that is frothy, bright red, and has no food in it: This tells you the blood is coming from the airway itself, not the stomach. Coughing up blood (Hemoptysis) (coughing up blood) means the lung or airway is bleeding, not the gut.
- Listen to one side of the chest with a stethoscope — hear crackles or quiet breath sounds on the bleeding side: Blood blocks the airways and stops oxygen from moving through. Finding which side is bleeding tells you where to send oxygen and where to watch hardest.
- Fast breathing, blue lips or fingers, or gasping for air: Coughing up blood (Hemoptysis) is dangerous because people drown in their own blood before they run out of blood. The crash point is not enough air moving in.
- Fever, weight loss, or cough for weeks (tuberculosis), or shortness of breath after a flight or surgery (pulmonary a traveling clot lodging in a vessel (embolism)): These clues point to what broke upstream — tuberculosis, cancer, bronchiectasis (airways widened and scarred by old infections), or a blood clot damaging the lung. Trace backwards to find the real sickness.
- Low oxygen on pulse ox (less than 90%), or the patient sits up and leans forward to breathe easier: The body is trying to keep blood flow and oxygen going. This position helps gravity drain blood away from the good lung so it can work.
- Swollen, clubbed fingers or nail beds, or old scars on chest X-ray from past lung damage: These findings tell you this has been breaking for a long time — bronchiectasis or chronic lung disease is the upstream break causing coughing up blood (hemoptysis) now.

Diagnostics · every test answers a question

- Complete blood count (CBC) → How much blood has been lost, and is anemia (low red cells) making oxygen delivery worse?

- Prothrombin time (PT), international normalized ratio (INR), and partial thromboplastin time (PTT) → Is the blood clotting normally, or is a medicine or bleeding disorder keeping the vessel from sealing?
- Type and crossmatch (blood bank) → What blood type does this person have, so we can give the right blood if they crash?
- Chest X-ray → Which lung is bleeding, and what upstream problem do I see — infection, tumor, fluid, or scarring?
- CT scan of the chest with contrast → Where exactly is the bleed, and what broke — a tumor eating a vessel, a dilated artery, or damaged airways?
- Flexible bronchoscopy → Can I see the bleeding site directly, and is it safe to use the scope to stop the bleed or take a sample?
- Arterial blood gas (ABG) → Is the person getting enough oxygen into their blood, or are they crashing from too much blood in the airways?

Treatment logic

- Position bleeding lung down (lateral decubitus with bad side down), give oxygen, and prepare for intubation with a double-lumen tube if massive bleed → Gravity keeps blood in the damaged lung instead of spilling into the good lung. Oxygen flows through the healthy side while you buy time to stop the bleed. A double-lumen tube isolates the lungs so the good one stays clear.
- Bronchial artery embolization (interventional radiology threads a catheter into the bronchial artery and blocks it with coils or particles) → Stops blood flow to the damaged vessel by closing the artery upstream. This is the main fix for massive bleeds (more than 200 mL in 24 hours or life-threatening) because most lung blood comes from the bronchial arteries, not the pulmonary arteries.
- Stop anticoagulants (warfarin, heparin, DOACs) and antiplatelet drugs (aspirin, clopidogrel); reverse warfarin with vitamin K and four-factor prothrombin complex concentrate (PCC), or reverse dabigatran with idarucizumab → Lets the body form a clot to seal the broken vessel instead of the drug keeping blood liquid and flowing.
- Rigid bronchoscopy with tamponade (packing with gauze or a balloon), topical epinephrine (squirted onto the bleeding site to shrink vessels), or cold saline lavage (washing with icy salt water to clot blood) → Directly packs or clots the bleeding point under direct vision so blood stops entering the airway. Rigid scope has a wide barrel so you can see better and pass tools in massive bleeds.

- Treat the underlying cause: antibiotics (amoxicillin-clavulanate or a fluoroquinolone) for bronchiectasis or bacterial bronchitis, four-drug anti-TB therapy (rifampin, isoniazid, pyrazinamide, ethambutol) for tuberculosis, antifungals (voriconazole or amphotericin) for aspergilloma (fungal ball), chemotherapy or radiation for lung cancer, immunosuppressants (cyclophosphamide and steroids) for granulomatosis with polyangiitis or Goodpasture syndrome → Fixes the broken step upstream — kills the infection, shrinks the tumor, or stops the immune attack — so the vessel stops being destroyed and can heal.
- Oxygen by nasal cannula or mask, IV fluids (normal saline or lactated Ringer), type and cross for packed red blood cells, transfuse if hemoglobin drops more than 2 g/dL or the person becomes dizzy, pale, or with a racing heart (tachycardic) → Keeps oxygen delivery running and replaces lost blood volume so the heart does not have to pump harder to feed the body.
- Tranexamic acid (antifibrinolytic that stops clot breakdown) 1 g IV over 10 minutes, then 1 g IV over 8 hours → Stabilizes the clot at the bleeding site so it does not dissolve too fast. Helpful when the bleed is moderate and you are waiting for embolization or when surgery is planned.

Medications

Tranexamic acid (TXA) · Antifibrinolytic (stops the body from dissolving clots too soon)

It blocks plasmin, the enzyme that breaks down blood clots. Think of it as putting a lock on the clot so it stays in place long enough to seal the bleeding vessel in the lung. This gives the body time to repair the damaged spot without the clot washing away too early.

1. You cough up blood because a vessel in your airway or lung tissue has torn or eroded
2. Your body forms a clot to plug the hole, like a cork in a leaky pipe
3. Normally, plasmin (a clot-dissolving enzyme) starts breaking down that clot within hours
4. Tranexamic acid locks onto plasminogen so it cannot turn into plasmin
5. The clot stays firm and keeps the vessel sealed while healing begins
6. Bleeding slows or stops because the plug holds

SAFETY WATCH

Can raise the risk of clots in the legs (deep vein clot formation (thrombosis)) or lungs (pulmonary a traveling clot lodging in a vessel (embolism)) if you are already at high risk—tell your doctor if you have a history of clots, recent surgery, or are on birth control pills · Not safe if you have blood in your urine from a kidney or bladder bleed (it can cause clots to block the urinary tract) · Rarely causes seizures, especially at high doses or in people with kidney disease · Should be dose-adjusted or avoided if your kidneys are not filtering well (creatinine clearance below 30 mL/min)

Antibiotics (specific choice depends on the infection) · Antimicrobial (kills or stops bacteria)

If the bleeding comes from infection—tuberculosis, pneumonia, lung abscess, or infected bronchiectasis (permanent widening of airways that traps mucus and bacteria)—antibiotics kill the germs that are eating away at blood vessels. Once the infection clears, inflammation drops and vessels heal, stopping the bleed.

1. Bacteria invade the lung or airway lining
2. They release toxins and trigger fierce inflammation
3. Inflammation weakens the walls of nearby blood vessels, making them fragile or eroding straight through
4. The antibiotic enters the infected area and either kills bacteria (bactericidal drugs like fluoroquinolones or beta-lactams) or stops them from multiplying (bacteriostatic drugs like macrolides)
5. With the germs gone, white blood cells calm down, swelling drops, and vessel walls begin to repair
6. Bleeding slows as the tissue heals

SAFETY WATCH

Fluoroquinolones (like levofloxacin) can damage tendons, especially the Achilles; avoid heavy exercise and stop immediately if you feel sudden heel or leg pain · Macrolides (like azithromycin) can disturb heart rhythm (QT prolongation), particularly risky if you are already on heart drugs or have low potassium or magnesium · Beta-lactams (like amoxicillin-clavulanate or ceftriaxone) can cause severe allergic reactions; tell your doctor about any past penicillin or cephalosporin allergy · Rifampin (for tuberculosis) turns urine, tears, and sweat orange and interacts with many drugs—birth control pills, blood thinners, and seizure medicines lose effectiveness

Antifungal agents (e.g., voriconazole, amphotericin B) · Antifungal (kills or stops fungi)

Fungi like *Aspergillus* can grow a ball (aspergilloma) inside old lung cavities—from tuberculosis scars or bronchiectasis—and the fungus ball rubs against blood vessels, causing bleeding. Antifungals punch holes in the fungal cell wall or stop fungi from making the fats they need to survive. As the fungus dies, the irritation drops and bleeding can lessen, though often surgery or embolization is still needed to remove the ball.

1. A fungus ball forms in a pre-existing lung cavity, sitting like a sponge soaked in blood vessels
2. The ball moves with breathing, scraping against fragile vessel walls
3. Vessels erode and bleed into the airway, sometimes in large amounts
4. Voriconazole blocks ergosterol production (the main fat in fungal membranes), making the fungus cell wall fall apart
5. Amphotericin B binds to ergosterol directly and tears holes in the membrane, killing the fungus quickly
6. As the fungus shrinks or dies, local inflammation drops and the cavity may stabilize, reducing bleeding risk

SAFETY WATCH

Voriconazole can cause blurry vision, bright flashes, or color changes, especially in the first weeks; usually improves but tell your doctor if it persists · Both drugs can harm the liver—regular blood tests (AST, ALT) are needed; watch for yellow skin, dark urine, or belly pain · Amphotericin B is hard on the kidneys and can drop potassium and magnesium dangerously low—you will need IV fluids and lab checks several times a week · Voriconazole interacts with many drugs (statins, blood thinners, seizure meds)—bring a full medication list to every visit

Immunosuppressants (e.g., cyclophosphamide, rituximab, corticosteroids) · Immunosuppressive (calms or blocks the immune system)

If bleeding comes from the immune system attacking blood vessels—vasculitis like granulomatosis with polyangiitis (GPA) or Goodpasture disease—these drugs turn down or redirect the immune assault. Corticosteroids (like prednisone) damp inflammation broadly. Cyclophosphamide wipes out fast-dividing immune cells. Rituximab kills B cells, the immune cells that make harmful antibodies. By stopping the attack, vessel walls stop breaking down and bleeding stops.

1. In vasculitis or autoimmune disease, your immune system mistakenly tags your own blood-vessel lining as an enemy

2. White blood cells swarm the vessels and release enzymes and inflammatory chemicals that chew through the walls
3. Vessels rupture and blood leaks into the airway or lung air sacs (alveolar bleeding (hemorrhage))
4. Corticosteroids block the genes that produce inflammatory signals, quieting the immune cells within hours to days
5. Cyclophosphamide attaches to DNA in dividing immune cells, killing them so fewer attackers remain
6. Rituximab binds to CD20 (a marker on B cells) and marks them for destruction, erasing the cells that make harmful antibodies
7. With the immune attack dialed down, inflammation fades, vessel walls repair, and bleeding stops

SAFETY WATCH

All three raise infection risk—watch for fever, cough, painful urination, or any new symptom and call your doctor immediately · Corticosteroids (prednisone, methylprednisolone) raise blood sugar, blood pressure, and can thin bones; you may need calcium, vitamin D, and osteoporosis screening · Cyclophosphamide can cause bladder bleeding (hemorrhagic cystitis)—drink plenty of water and tell your doctor if you see blood in your urine; it also raises cancer risk (bladder, blood) years later · Rituximab can cause infusion reactions (fever, chills, rash, low blood pressure) during or right after the IV; you will be watched closely and given premedication · Live vaccines (MMR, varicella, nasal flu) are unsafe while on these drugs—talk to your doctor before any immunization

Reversal agents for anticoagulants (e.g., vitamin K, prothrombin complex concentrate, idarucizumab, andexanet alfa) · Anticoagulant reversal agents (turn off blood-thinner effects)

If you are on a blood thinner (warfarin, dabigatran, rivaroxaban, apixaban) and start coughing up blood, the thinner keeps the blood from clotting properly and can turn a small bleed into a dangerous one. Reversal agents either replace the clotting factors the drug blocks (prothrombin complex concentrate) or directly bind and neutralize the drug (idarucizumab for dabigatran, andexanet alfa for Xa inhibitors). This restores normal clotting so your body can plug the leak.

1. Blood thinners work by blocking specific clotting factors or enzymes—warfarin blocks vitamin K-dependent factors (II, VII, IX, X), dabigatran blocks thrombin, and Xa inhibitors (rivaroxaban, apixaban) block factor Xa
2. When a vessel tears, the missing or blocked factors mean clots form slowly or not at all

3. Blood pours into the airway instead of clotting and sealing the hole
4. Vitamin K (for warfarin) helps the liver make new clotting factors over 6–24 hours, slowly restoring balance
5. Prothrombin complex concentrate delivers ready-made factors II, VII, IX, X immediately, bypassing the wait
6. Idarucizumab (for dabigatran) is an antibody fragment that grabs and locks onto dabigatran molecules, pulling them out of circulation within minutes
7. Andexanet alfa (for Xa inhibitors) is a decoy factor Xa that soaks up the drug, freeing the real factor Xa to do its job
8. With clotting restored, the body seals the bleeding vessel

SAFETY WATCH

Prothrombin complex concentrate and recombinant factor VIIa carry a risk of clots (stroke, heart attack, a clot in a deep leg vein (deep vein thrombosis), pulmonary embolism (a clot lodging in a lung artery))—use only for serious, life-threatening bleeds · Vitamin K can interfere with warfarin for days to weeks afterward, making it hard to restart safely—cardiologists adjust dosing carefully · Idarucizumab and andexanet alfa can cause allergic reactions or infusion-related fever and headache; you will be monitored during and after · Reversing a blood thinner means the original clot risk (stroke in atrial fibrillation (an irregular, quivering heartbeat), recurrent pulmonary embolism (a clot lodging in a lung artery)) comes roaring back—your team will restart a thinner or switch strategies as soon as bleeding is controlled

Red flags

- Coughing up more than a few teaspoons of blood at once, or blood that keeps coming in large amounts (this can block your airway or mean a major vessel is torn)
- Sudden trouble breathing, very fast breathing, or blue lips or fingertips (signs your oxygen is dangerously low)
- Feeling dizzy, confused, or about to pass out (your brain is not getting enough oxygen or you have lost a lot of blood)
- Chest pain that is severe, crushing, or spreads to your arm or jaw (could be a heart problem or a clot in the lung artery)
- History of recent surgery, long travel, or a known clotting disorder (raises the chance of a pulmonary embolism (a clot lodging in a lung artery), a clot blocking the lung)

Nursing priorities

1. Apply continuous pulse oximetry and place the patient on high-flow oxygen or non-rebreather mask to keep oxygen saturation above 90 percent — Blood fills alveoli (air sacs) and blocks gas exchange (oxygen moving into blood). Low oxygen in the blood (Hypoxemia) (low oxygen) causes organ damage within minutes. Oxygen buys time while we find and stop the bleeding source.
2. Position the patient sitting upright or with the suspected bleeding lung down if the side is known — Gravity keeps blood in the bad lung and prevents it from spilling into the good lung. If blood floods both lungs, the patient cannot oxygenate at all and will need emergency intubation (breathing tube placement).
3. Establish two large-bore IV lines (18-gauge or bigger) and send blood for type-and-cross, complete blood count, PT/INR, PTT, and basic metabolic panel — Massive coughing up blood (hemoptysis) causes hypovolemia (low blood volume) and bleeding-driven (hemorrhagic) shock (not enough blood to perfuse organs). Large IVs allow rapid fluid resuscitation (giving saline or blood fast). Labs show anemia (low red blood cells), coagulation problems (clotting disorders), and readiness for transfusion.
4. Hold all anticoagulants and antiplatelet drugs immediately and notify the provider for reversal agents if the patient is on warfarin, dabigatran, or heparin — Anticoagulants stop clot formation and make bleeding continue. Warfarin reverses with vitamin K and prothrombin complex concentrate (PCC). Dabigatran reverses with idarucizumab (a specific antidote). Heparin reverses with protamine sulfate. Stopping these drugs helps the body form clots at the bleeding site.
5. Keep the patient NPO (nothing by mouth) and prepare for emergent bronchoscopy or intubation with a double-lumen endotracheal tube — Aspiration (breathing in stomach contents) during a bleeding episode or procedure causes aspiration pneumonia (lung infection from stomach acid). A double-lumen tube isolates the good lung so the patient can breathe while providers work on the bleeding lung through bronchoscopy.
6. Monitor vital signs every 15 minutes, watching for a fast heart rate (tachycardia) (heart rate above 100), low blood pressure (hypotension) (systolic blood pressure below 90), fast breathing (tachypnea) (breathing faster than 20), and decreasing oxygen saturation — These signs show bleeding-driven (hemorrhagic) shock (not enough circulating blood) or worsening low oxygen in the blood (hypoxemia). A fast heart rate (Tachycardia) tries to compensate for low blood volume. Low blood pressure (Hypotension) means shock is decompensating (the body cannot keep up). This triggers immediate ICU transfer and massive transfusion protocol (giving packed red blood cells, plasma, and platelets rapidly).

7. Measure and document the amount of blood expectorated using a graduated container or absorbent pads, and note color and consistency — Quantifying blood loss guides treatment intensity. More than 100 to 600 milliliters in 24 hours is massive coughing up blood (hemoptysis) requiring bronchial artery embolization (interventional radiology blocking the artery) or surgery. Tracking trends shows if bleeding is slowing or accelerating.
8. Notify interventional radiology and the ICU team immediately if coughing up blood (hemoptysis) is massive or the patient shows signs of respiratory distress — Bronchial artery embolization (IR procedure to plug the bleeding artery) stops massive coughing up blood (hemoptysis) in 90 percent of cases and must happen within hours. ICU provides mechanical ventilation (breathing machine support), selective lung ventilation, and close hemodynamic monitoring (watching blood pressure and heart function) that floor nurses cannot provide.

Sources

- American Thoracic Society (ATS)
- European Respiratory Society (ERS)
- American College of Chest Physicians (ACCP)
- CDC (for tuberculosis and infectious causes)

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