

Infective Endocarditis

Infective endocarditis — a bacterial clump growing on a heart valve, and the germ type, the patient's risks, and the valve condition together drive everything that happens next.

THE ONE-LINE TRUTH

Bacteria land on a heart valve, stick there, and grow a clump of germs and clotted blood that damages the valve and scatters pieces into the bloodstream.

The chain — how it unfolds

1. Bacteria stick to a damaged or artificial valve and multiply, forming a clump (vegetation) of germs, platelets, and fibrin. — why: A rough or prosthetic valve surface loses its slippery coating, so when bacteria enter the blood—from dental work scraping gums, a skin cut, injecting drugs, or a catheter—they land and stick instead of washing away. Platelets and fibrin pile on, building a fragile, infected clump.
2. The immune system mounts inflammation and fever to fight the infection. — why: White blood cells recognize foreign bacteria and release cytokines (chemical alarms like interleukin-1 and tumor tissue death (necrosis) factor) that raise body temperature (fever) and call more immune cells to the site. This is why patients feel feverish, exhausted, lose their appetite, sweat at night, and drop weight.
3. Pieces of the vegetation break off and travel through the arteries as septic traveling clots (emboli), lodging in the brain, kidneys, skin, and fingers. — why: The vegetation is soft and crumbly. Turbulent blood flow knocks chunks loose, and these infected clumps (septic traveling clots (emboli)) flow downstream until they wedge in smaller arteries. In the brain they cause stroke (sudden weakness, slurred speech). In the skin they cause Janeway lesions (painless red spots on palms and soles from tiny infected clots), Osler nodes (painful red bumps on finger pads from immune-complex inflammation), splinter hemorrhages (red streaks under the nails from broken capillaries), and Roth spots (retinal hemorrhages with white centers seen on eye exam). In the kidneys they cause glomerulonephritis (immune attack on filters, leaking blood and protein into urine).

4. The valve leaflets are eroded and torn by bacterial enzymes and inflammation, creating a leak (regurgitation). — why: Bacteria release enzymes that digest tissue, and the immune attack damages the valve further. Holes appear in the leaflets, or the leaflets tear away from their anchors. Now blood leaks backward through the valve with every heartbeat, producing a new or changing heart murmur (the whooshing sound of turbulent backflow heard through a stethoscope).
5. The heart pumps harder and faster to make up for the leaky valve, but over days the muscle tires and heart failure develops. — why: A leaky valve means some blood sloshes backward instead of moving forward, so the left ventricle has to pump the same blood multiple times. The ventricle stretches and squeezes harder, and the sympathetic nervous system speeds the heart rate. This compensation keeps blood pressure near normal at first, but the muscle fibers stretch beyond their limit, weaken, and lose their squeeze. Fluid backs up into the lungs (causing shortness of breath, cough, and pink frothy coughed-up mucus (sputum)) and into the legs (causing swelling). The patient cannot lie flat without gasping for air.
6. Infection may tunnel from the valve into the surrounding heart muscle, forming an abscess or disrupting the electrical conduction system. — why: Bacteria burrow through the valve ring into the myocardium (heart muscle) and the atrioventricular node or bundle of His (the wiring that carries the heartbeat signal from atria to ventricles). The body walls off the bacteria with pus (an abscess), but this destroys normal tissue. If the electrical pathway is interrupted, the signal from atria to ventricles is blocked (heart block), the ventricles beat very slowly or pause, and the patient faints or goes into cardiac arrest.
7. Without treatment, one of four crashes occurs: massive stroke, acute valve rupture with shock, complete heart block, or uncontrolled sepsis with multi-organ failure. — why: The body's compensation—fever, faster heart rate, fluid retention—buys time but cannot cure the infection. Eventually a large a traveling clot (embolus) hits the brain, the valve tears completely and floods the lungs, the conduction system fails, or bacteria overwhelm the bloodstream. At that point, even emergency antibiotics and surgery may not reverse the damage, and death is common.
8. Tracing backward: the whole chain started when bacteria met a damaged valve. Cure requires killing the bacteria (prolonged IV antibiotics, often 4–6 weeks) and, in many cases, surgically removing the destroyed valve and replacing it. — why: Antibiotics alone cannot always sterilize a large vegetation or repair a torn valve, so surgery to cut out infected tissue and implant a new valve is often necessary—especially if the valve is falling apart, heart failure is severe, abscesses have formed, or traveling clots (emboli) keep breaking off despite treatment. The key is recognizing endocarditis early (positive blood cultures, new murmur, fever, and embolic signs) and starting treatment before the crash point.

Spot it — what you see, and why

- Fever that won't quit: Bacteria are growing on the heart valve and releasing toxins into the blood, so the body raises its temperature to try to kill the germs—like turning up the heat to fight invaders.
- New heart murmur or a murmur that sounds different: The infection eats away at the valve or makes clumps of germs and clot stick to it, so blood leaks backward or the valve can't close right—the turbulent flow makes the whooshing sound the doctor hears.
- Tiny red or purple spots on the skin (petechiae) or painful red bumps on fingers and toes (Osler nodes): Small clumps of bacteria and clot break off the valve, travel through the bloodstream, and jam tiny blood vessels in the skin—blocking flow causes the spot or swelling.
- Painless red spots on the palms or soles (Janeway lesions): Bacteria or toxins lodge in small vessels under the skin and trigger tiny patches of bleeding without pain—like miniature bruises from the inside.
- Splinter hemorrhages—tiny red lines under the nails: Clumps of bacteria plug the tiny vessels that feed the nail bed, causing streaks of bleeding that look like wood splinters trapped under the nail.
- Sudden stroke or patches of vision loss: A chunk of infected clot (septic a traveling clot (embolus)) breaks off the heart valve and shoots up into the brain or eye arteries, cutting off blood and starving that tissue of oxygen.

Red flags — act now

- New or worsening heart murmur—means the valve is being destroyed fast
- Sudden weakness, slurred speech, or vision loss—a clump may have hit the brain
- Severe shortness of breath or coughing up pink foam—sign the heart is failing and fluid is flooding the lungs
- Confusion or severe headache—infection may have spread to the brain lining (meningitis) or caused a brain abscess
- Uncontrolled fever despite antibiotics—the infection may be walled off in an abscess or the germ is resistant to the drug

Treat it — in this order

1. Draw three sets of blood cultures from different sites before antibiotics, then start empiric IV antibiotics immediately (vancomycin 15–20 mg/kg every 8–12 hours + ceftriaxone 2 g daily or gentamicin 1 mg/kg every 8 hours) → Kills bacteria multiplying on the valve and stops them from breaking off and seeding more clots to the brain, kidneys, and lungs. Drawing cultures first gives you the chance to identify the exact bacteria and tailor antibiotics later. Vancomycin covers methicillin-resistant *Staphylococcus aureus* (MRSA) and coagulase-negative staph. Ceftriaxone or gentamicin covers *Streptococcus* and Gram-negative bacteria.
2. Transesophageal echocardiogram (TEE) within 24–48 hours to confirm vegetations and assess valve damage → Proves the diagnosis is infective endocarditis and not another cause of fever and murmur (like rheumatic fever or atrial myxoma). Shows the size of vegetations, how badly the valve is leaking or blocked, and whether there is an abscess or prosthetic valve dehiscence (loosening). This tells you whether antibiotics alone will work or surgery is needed urgently.
3. Switch to targeted antibiotics once blood culture results and sensitivities return: nafcillin or cefazolin 2 g IV every 4–6 hours for methicillin-sensitive *Staphylococcus aureus* (MSSA), vancomycin for MRSA, penicillin G 4 million units every 4 hours or ceftriaxone 2 g daily for *Streptococcus viridans*, ampicillin 2 g every 4 hours + gentamicin for *Enterococcus* → Targets the exact bacteria growing on the valve so you use the most effective antibiotic and avoid resistance. Beta-lactams like nafcillin and penicillin are bactericidal (kill bacteria outright) and penetrate vegetations better than vancomycin when the bacteria are sensitive.
4. Cardiac surgery for valve replacement or repair if: heart failure develops, infection cannot be controlled with antibiotics alone (persistent fever and positive blood cultures after 5–7 days), prosthetic valve is loose or infected, vegetation is very large (>10 mm) with high embolic risk, perivalvular abscess forms, or fungal endocarditis is diagnosed → Removes the destroyed or infected valve and replaces it with a mechanical or bioprosthetic valve, eliminating the bacteria's home and restoring normal blood flow. Surgery also drains abscesses and repairs holes in the heart wall. For fungal endocarditis, surgery is almost always needed because fungi do not respond well to antifungals alone.
5. Continue IV antibiotics for 4 to 6 weeks total (native valve endocarditis) or 6 to 8 weeks (prosthetic valve endocarditis), with repeat blood cultures to confirm clearance → Gives enough time to sterilize the vegetations on the valve and kill bacteria hiding in biofilm (a protective slime bacteria make). Stopping too early leads to relapse and resistant bacteria. Repeat blood cultures after 48–72 hours of antibiotics should be negative; if they stay positive, the infection is not controlled.

Your top priorities at the bedside

1. Obtain at least three sets of blood cultures from different sites before starting any antibiotics, spacing them 30 minutes apart — why: Bacteria grow continuously in endocarditis so blood cultures are positive 95 percent of the time if drawn before antibiotics; getting cultures from different puncture sites proves the bacteria are really in the blood and not just skin contamination, and three sets meet the Duke major criterion
2. Start IV antibiotics within one hour after blood cultures are drawn, using vancomycin plus ceftriaxone for empiric coverage — why: Early antibiotic therapy stops bacteria from making the vegetation bigger and reduces risk of traveling clots (emboli) (bacteria clumps breaking off); vancomycin covers MRSA and ceftriaxone covers streptococci and gram-negative bacteria until culture results identify the exact organism
3. Monitor temperature every 4 hours and immediately report new fever spikes or persistent fever after 72 hours of antibiotics — why: Fever should decrease within 3 to 7 days if antibiotics are working; persistent fever means the bacteria are resistant, an abscess formed in the heart muscle around the valve, or the patient needs surgery because antibiotics cannot sterilize the vegetation