

Infective Endocarditis

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THE STORY IN ONE SENTENCE

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.

“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.”

Normal anatomy — the body's job

The heart has four valves—mitral, tricuspid, aortic, and pulmonic—that open and close in rhythm to keep blood flowing forward and prevent backflow. Each valve is covered by a thin, smooth lining called the endocardium (the inside skin of the heart). This lining is slippery and intact, so bacteria and platelets in the blood slide right past without sticking.

BODY'S JOB

The endocardium's smooth surface repels bacteria and keeps blood from clotting on the valve. The valve leaflets (thin flaps) meet tightly when closed, sealing perfectly so no blood leaks backward. This tight seal and smooth lining together keep the valve sterile and working quietly for a lifetime.

1. Blood flows into the heart chamber.
2. The valve opens and blood rushes forward into the next chamber or artery.
3. The valve snaps shut, and its smooth leaflets press together to seal the opening.
4. Because the endocardium is slippery and undamaged, bacteria and platelets in the blood cannot grip the surface and are swept downstream.
5. The cycle repeats every heartbeat, keeping blood moving one way and the valve sterile.

What breaks

A valve that is already damaged (from past illness, wear and tear, or birth defect) or artificial (mechanical or tissue replacement) loses its smooth surface and becomes rough. At the same time, bacteria enter the bloodstream from a source—dental work, skin infection, injecting drugs with dirty needles, or a medical procedure. Instead of sliding past, the bacteria land on the rough valve and stick. They multiply there, and platelets and fibrin (clotting threads) pile on top, forming a lumpy, fragile clump called a vegetation that damages the valve further and breaks off pieces into the blood.

CLINICAL TERM

Infective endocarditis occurs when bacteremia (bacteria in the bloodstream) meets an abnormal valve surface. Risk factors include prior rheumatic heart disease, degenerative valve disease (calcific aortic narrowing (stenosis), mitral valve prolapse), congenital heart defects (bicuspid aortic valve, ventricular septal defect), prosthetic valves, and intravenous drug use. Common bacterial causes are *Staphylococcus aureus* (most common overall and in IVDU and healthcare-associated cases), viridans group streptococci (after dental procedures or in native valve endocarditis), *Enterococcus* species (after GU or GI procedures, especially in older men), and HACEK organisms (*Haemophilus*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella*, *Kingella*—slow-growing, culture-negative cases). The vegetation is composed of platelets, fibrin, bacteria, and inflammatory cells. It is friable and sheds septic traveling clots (emboli).

How the body compensates

The body tries to fight the infection with fever and inflammation, and the heart tries to make up for the leaky valve by pumping faster and harder. These efforts can keep blood pressure and oxygen levels near normal for days, masking how sick the patient really is—until the valve tears apart, a big clot flies to the brain, or the heart muscle gives out and everything crashes suddenly.

1. {'step': 'The immune system ramps up fever and inflammation', 'how': 'White blood cells and cytokines (chemical alarms like interleukin-1 and tumor tissue death (necrosis) factor) flood the bloodstream to fight the bacteria. Fever raises body temperature to slow bacterial growth, and inflammation tries to wall off the vegetation.', 'cost': 'Fever and cytokines cause fatigue, muscle aches, loss of appetite, and weight loss. The inflammation itself damages surrounding heart tissue and can trigger more clotting.'}

2. {'step': 'The heart increases stroke volume and rate', 'how': 'The left ventricle (the heart's main pumping chamber) stretches to hold more blood and squeezes harder. The sympathetic nervous system (the body's gas pedal) releases norepinephrine (a stimulating hormone) to speed the heart rate and tighten blood vessels.', 'cost': 'The heart muscle works overtime, burning energy and oxygen faster. Over days, the muscle fibers stretch too far and weaken, losing their squeeze. The patient feels the fast heartbeat (palpitations) and gets short of breath with mild effort.'}
3. {'step': 'The kidneys hold on to salt and water', 'how': 'The kidneys sense lower forward flow and release renin, which triggers aldosterone (a hormone that tells the kidneys to save salt and water) to boost blood volume and pressure.', 'cost': 'Extra fluid raises pressure in the lungs and legs, causing shortness of breath, swollen ankles, and weight gain. The kidneys themselves may be hit by septic traveling clots (emboli), causing blood and protein to leak into the urine.'}
4. {'step': 'Watch for the crash point', 'how': 'Compensation hides the severity until one of four catastrophes breaks through: (1) a large vegetation breaks off and blocks a brain artery, causing sudden stroke with paralysis or speech loss; (2) the valve tears completely, flooding the lungs with fluid and dropping blood pressure (cardiogenic shock); (3) infection tunnels into the heart muscle, forming an abscess and interrupting the electrical pathway, causing complete heart block and a dangerously slow pulse; or (4) bacteria seed distant sites, forming abscesses in the spine, spleen, or brain.', 'cost': 'Any of these crashes can be fatal within hours, and the window to reverse damage closes quickly. Once shock or stroke occurs, even emergency surgery may not save the patient.'}

Domino effect

1. Bacteria stick to a damaged or artificial valve and multiply, forming a clump (vegetation) of germs, platelets, and fibrin. — 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. — 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. — 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). — 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. — 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. — 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. — 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. — 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.

Symptoms · what the patient feels, 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.
- Shortness of breath and swollen ankles (heart failure): The infected valve leaks so badly or gets so stiff that the heart can't pump blood forward efficiently—fluid backs up into the lungs and legs, just like a dam overflow.
- Night sweats and weight loss you didn't plan: The long-running infection revs up metabolism and triggers cytokines (alarm chemicals) that make you sweat at night and burn calories faster than you eat—your body is spending all its energy fighting the germs.
- Joint pain or back pain: The immune system attacks bacteria that have seeded into a joint or a spine bone (vertebra), causing swelling and pain—or tiny infected clots lodge in vessels near the joint.

- Blood in the urine or sudden flank pain: Infected clots fly to the kidneys and clog small vessels (causing tiny infarcts—dead patches) or trigger an immune reaction (glomerulonephritis) that inflames the kidney's filters and lets red cells leak into the urine.

Focused exam

- New heart murmur or change in an old murmur: The valve leaflets are damaged or covered with clumps of bacteria, platelets, and fibrin (the body's glue for clotting), so blood leaks backward or flows noisily forward through the broken valve. A new murmur or a murmur that sounds different from before is direct proof that bacteria are living on the valve itself — not just floating in the bloodstream.
- Fever that stays high or keeps coming back, often with chills and night sweats: Fever is the immune system's alarm that bacteria are multiplying on the valve and releasing toxins into the bloodstream. Infective endocarditis does not cause a quick fever that goes away in a day or two — the fever hangs on because the infection is deep inside the heart and hard for the body to reach.
- Janeway lesions (flat, painless red spots on the palms and soles) or Osler nodes (tender red bumps on the fingertip pads): Infected clumps of bacteria and platelets break off the valve, travel through the arteries, and lodge in tiny blood vessels under the skin. Janeway lesions are caused by bacteria seeding small vessels and causing tiny bleeds. Osler nodes are caused by immune complexes or infected clots triggering inflammation in the fingertip vessels. Both are proof that septic traveling clots (emboli) (infected clots) are traveling from the heart.
- Roth spots (round white-centered spots with red edges on the retina, seen during eye exam): Tiny blood vessels in the back of the eye burst from infected clots or immune complex damage. Roth spots are rare but are a classic sign of infective endocarditis and mean the infection is severe enough to send clots to the smallest vessels in the body.
- Splinter hemorrhages (thin red or brown lines under the fingernails or toenails): Infected clots hit the tiny blood vessels in the nail bed and broke them, causing a line of blood under the nail. This is not from trauma or fungus — it is proof that septic traveling clots (emboli) from the heart valve are traveling through the bloodstream and damaging small vessels.
- Signs of heart failure: swollen neck veins, swollen legs, shortness of breath, crackles in the lungs, or a weak and fast heartbeat: The valve is now so damaged — either leaking backward or too stiff to open — that the heart cannot pump blood forward properly. Blood backs up into the lungs and body. Heart failure from infective endocarditis means the valve damage is severe and antibiotics alone may not be enough; the valve may need to be replaced urgently.

- History of IV drug use, recent dental or surgical procedure, prosthetic heart valve, or previous endocarditis: These are the open doors that let bacteria enter the bloodstream and stick to the heart valve. IV drug use introduces skin bacteria directly into veins. Dental work or surgery releases mouth or gut bacteria into the bloodstream. Prosthetic valves and damaged native valves are rough surfaces where bacteria can easily attach and multiply. Without both a damaged valve and bacteria in the blood at the same time, endocarditis cannot start.

Diagnostics · every test answers a question

- Blood cultures (three sets from different sites, drawn 30 minutes apart before antibiotics) → What bacteria are growing on the valve, and are they actually in the bloodstream all the time?
- Transthoracic echocardiogram (TTE) or transesophageal echocardiogram (TEE, the preferred test) → Is there a clump of bacteria, platelets, and fibrin (called a vegetation) stuck to the valve, and how badly is the valve damaged?
- Complete blood count (CBC) with differential → Is the immune system fighting an infection, and is the bone marrow keeping up?
- Red blood cell (Erythrocyte) sedimentation rate (ESR) and C-reactive protein (CRP) → How much inflammation is in the body right now?
- Basic metabolic panel (BMP) and creatinine → Are the kidneys holding up, or are septic traveling clots (emboli) or immune complexes damaging them?
- Urinalysis → Are red blood cells or protein spilling into the urine from kidney damage?
- Electrocardiogram (ECG) → Is the infection spreading into the heart's electrical wiring and causing dangerous heart blocks?
- Chest X-ray → Are septic traveling clots (emboli) seeding the lungs, or is heart failure causing fluid to back up into the lungs?

Treatment logic

- 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.

- 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.
- 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.
- 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.
- 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.
- Monitor closely for embolic complications (stroke, septic pulmonary traveling clots (emboli), splenic abscess, mycotic aneurysm) and manage supportively: IV fluids for low blood pressure, diuretics (furosemide 20–80 mg IV) for heart failure and fluid overload, inotropes (dobutamine) if the heart is too weak to pump → Catches life-threatening complications early so you can treat them before they become irreversible. Stroke happens in 15–30% of patients when clots break off and block brain arteries. Mycotic aneurysms (weakened artery walls from infection) can rupture. Supportive care keeps the heart, lungs, and kidneys working while antibiotics clear the infection.

- Antibiotic prevention (prophylaxis) before dental or surgical procedures for high-risk patients after recovery: those with prosthetic valves, previous endocarditis, or unrepaired turning blue (cyanotic) congenital heart disease (amoxicillin 2 g PO 30–60 minutes before procedure, or clindamycin 600 mg if penicillin-allergic) → Reduces the chance of bacteria from the mouth or gut seeding the heart valve during procedures that cause transient bacteremia. High-risk patients have rough or abnormal valves where bacteria stick easily.

Medications

Vancomycin · Glycopeptide antibiotic

Vancomycin punches holes in the outer wall that bacteria build to protect themselves. Without that wall, the bacteria burst open and die. It works especially well against Gram-positive bacteria like MRSA (staph that resists many other antibiotics) and some strep species.

1. Vancomycin binds to the building blocks (called peptidoglycan precursors) that bacteria use to make their cell wall.
2. This stops the bacteria from finishing the wall-building process.
3. The weak, incomplete wall cannot hold the bacterium's internal pressure.
4. The bacterium swells and bursts, killing the infection.

SAFETY WATCH

Can harm the kidneys (nephrotoxicity), especially if blood levels get too high or if combined with other kidney-toxic drugs like gentamicin—check creatinine and vancomycin trough levels regularly. · Can damage hearing and balance (ototoxicity) if levels stay high for too long. · Red-man syndrome: flushing, itching, and rash if infused too fast; slow the infusion to prevent this. · Requires dose adjustment in kidney disease because vancomycin is cleared by the kidneys.

Ceftriaxone · Third-generation cephalosporin (beta-lactam antibiotic)

Ceftriaxone stops bacteria from building their cell wall by blocking the enzymes (called penicillin-binding proteins) that glue the wall pieces together. Without a finished wall, the bacteria pop and die. It covers many strep species (especially viridans strep, a common cause of endocarditis after dental work) and some Gram-negative bacteria.

1. Ceftriaxone enters the bacterial cell and binds tightly to penicillin-binding proteins (PBPs), the enzymes that cross-link the cell wall.
2. With PBPs blocked, the bacteria cannot finish building a strong wall.
3. The weak wall breaks under pressure, and the bacterium dies.

4. Ceftriaxone is stable against many (but not all) bacterial enzymes (beta-lactamases) that try to destroy beta-lactam antibiotics.

SAFETY WATCH

Can cause allergic reactions, including rash or (rarely) anaphylaxis; ask about penicillin allergy history (about 10% cross-reactivity). · Can cause diarrhea or *C. difficile* colitis (severe gut infection) by killing normal gut bacteria. · May interact with calcium-containing IV fluids in neonates, forming precipitates; avoid co-administration in newborns. · Rarely causes gallbladder sludge or stones (biliary pseudolithiasis) because it is excreted in bile.

Gentamicin · Aminoglycoside antibiotic

Gentamicin sneaks into bacteria and jams their protein-making factory (the ribosome). Without working proteins, the bacteria cannot survive or multiply. It is often paired with a cell-wall antibiotic (like vancomycin or ampicillin) to create synergy—the cell-wall drug punches a hole, and gentamicin rushes in to finish the job. This combo is critical for enterococcus endocarditis.

1. Gentamicin crosses the bacterial cell membrane (easier once a cell-wall antibiotic has weakened the wall).
2. It binds to the 30S subunit of the bacterial ribosome.
3. This causes the ribosome to misread the genetic code (mRNA) and make faulty, nonfunctional proteins.
4. The bacterium's machinery breaks down, and it dies.

SAFETY WATCH

Can damage the kidneys (nephrotoxicity), especially with prolonged use or high trough levels; monitor creatinine and drug levels closely. · Can harm the inner ear, causing permanent hearing loss or balance problems (ototoxicity); risk goes up with longer treatment or high levels. · Toxic risk increases when combined with other nephrotoxic drugs like vancomycin; use the lowest effective dose for the shortest time. · Requires dose adjustment in kidney disease; once-daily dosing is often used to reduce toxicity while maintaining effect.

Nafcillin or Cefazolin (for methicillin-sensitive *Staphylococcus aureus*, MSSA) · Penicillinase-resistant penicillin (nafcillin) or first-generation cephalosporin (cefazolin), both beta-lactams

Both drugs block the same step as ceftriaxone—they stop bacteria from building their cell wall by jamming the enzymes (penicillin-binding proteins) that assemble it. MSSA is sensitive to

these drugs, so they kill it effectively. Nafcillin and cefazolin are preferred over vancomycin for MSSA because they work faster and more completely.

1. The drug binds to penicillin-binding proteins (PBPs) on the bacterial wall.
2. PBPs can no longer cross-link the wall's peptidoglycan strands.
3. The unfinished wall collapses, and the bacterium dies.
4. These drugs resist breakdown by penicillinase (an enzyme some staph make), so they work where regular penicillin would fail.

SAFETY WATCH

Nafcillin can cause phlebitis (vein inflammation) at the IV site; rotate sites or use a central line for long infusions. · Both can cause allergic reactions; serious penicillin allergy is a contraindication (cefazolin has ~10% cross-reactivity). · Nafcillin may cause liver enzyme elevation (monitor AST/ALT) and rarely interstitial nephritis (kidney inflammation). · Cefazolin is easier on veins and usually preferred if renal function is normal.

Penicillin G or Ceftriaxone (for viridans group streptococci and *Streptococcus gallolyticus*) · Natural penicillin (penicillin G) or third-generation cephalosporin (ceftriaxone), both beta-lactams

These drugs stop strep bacteria from finishing their cell wall by blocking the enzymes that link wall pieces together. Viridans strep (common mouth bacteria that enter the blood during dental work or tooth-brushing) are usually very sensitive to penicillin, making it the gold standard. Ceftriaxone is an alternative, especially for once-daily dosing convenience.

1. The drug binds to penicillin-binding proteins (PBPs) that assemble the peptidoglycan wall.
2. Wall synthesis stops.
3. The bacterium's internal pressure bursts the weak wall, killing the cell.
4. Strep do not make beta-lactamase, so penicillin works perfectly without resistance.

SAFETY WATCH

Penicillin allergy: can cause rash, hives, or (rarely) anaphylaxis; always ask about allergy history. · High-dose penicillin G (used in endocarditis) can rarely cause seizures, especially in kidney failure or if given into the brain space (never give intrathecal). · Ceftriaxone shares the safety profile listed above (gallbladder sludge, *C. diff* risk, calcium interaction in neonates). · Both can cause diarrhea or secondary yeast infections by disrupting normal gut and skin bacteria.

Ampicillin plus Gentamicin or Ampicillin plus Ceftriaxone (for *Enterococcus* species) · Aminopenicillin (ampicillin) combined with an aminoglycoside (gentamicin) or third-generation cephalosporin (ceftriaxone), both beta-lactams plus synergy agent

Enterococcus is naturally tough—it resists many antibiotics and tolerates cell-wall agents alone. Ampicillin punches holes in the enterococcal wall, and gentamicin (or ceftriaxone, for *E. faecalis*) rushes through those holes to jam the bacteria's protein factory. Together, they kill enterococcus; alone, neither works well. This synergy is the cornerstone of enterococcal endocarditis treatment.

1. Ampicillin binds to penicillin-binding proteins and weakens the enterococcal wall.
2. The weakened wall lets gentamicin (or ceftriaxone) enter the bacterium more easily.
3. Gentamicin jams the ribosome, stopping protein production, or ceftriaxone further disrupts wall synthesis.
4. The combination kills the bacterium, whereas either drug alone would only slow it down.

SAFETY WATCH

Gentamicin nephrotoxicity and ototoxicity (see gentamicin entry above); monitor levels and renal function closely. · Ampicillin can cause allergic reactions (rash, anaphylaxis); also commonly causes a non-allergic rash, especially in patients with mononucleosis (not a true allergy). · Prolonged aminoglycoside use (4–6 weeks) increases toxicity risk; some protocols use shorter courses (2 weeks) when possible. · Ceftriaxone is an option for *E. faecalis* without high-level gentamicin resistance and may be safer for longer treatment.

Red flags

- 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

Nursing priorities

1. Obtain at least three sets of blood cultures from different sites before starting any antibiotics, spacing them 30 minutes apart — 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 — 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 — 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
4. Perform focused neurologic checks every 4 hours, looking for confusion, facial droop, arm drift, slurred speech, or vision changes — Embolic stroke happens in 25 percent of patients when vegetation pieces break off and block brain arteries; catching stroke early allows thrombolysis (clot-busting drugs) within 4.5 hours or lets the team decide if emergency valve surgery is needed to prevent more traveling clots (emboli)
5. Assess heart sounds every shift and immediately report new murmurs or changes in existing murmurs — A new regurgitant murmur (whooshing sound from blood leaking backward) or a murmur that gets louder means the valve is being destroyed and cannot close properly; this is a Duke major criterion and signals need for urgent echocardiogram and possible surgery
6. Monitor urine output hourly and check creatinine and BUN daily, reporting output below 0.5 milliliters per kilogram per hour — Kidney failure happens in endocarditis from traveling clots (emboli) blocking kidney arteries, immune complexes damaging kidney filters, or antibiotic toxicity from vancomycin and gentamicin; catching this early allows dose adjustment to prevent permanent kidney damage
7. Watch for sudden severe back pain, abdominal pain, or cold painful legs, and check pedal pulses every shift — Traveling clots (Emboli) can travel to arteries anywhere in the body causing acute limb starved blood flow (ischemia) (blocked leg artery), splenic tissue death from blocked blood flow (infarction) (dead spleen tissue), or renal tissue death from blocked blood flow (dead kidney tissue); these need emergency vascular surgery or interventional radiology to restore blood flow

8. Maintain strict IV site care using sterile technique, change tubing per protocol, and use antimicrobial-coated central lines if long-term access is needed — Patients need 4 to 6 weeks of continuous IV antibiotics so line infections can introduce new bacteria and cause repeat endocarditis; keeping the IV site clean prevents catheter-related bloodstream infections that would restart the whole infection

Sources

- AHA/ACC 2023 Infective Endocarditis Guideline
- European Society of Cardiology 2023 Endocarditis Guidelines
- Infectious Diseases Society of America (IDSA)
- UpToDate Clinical Guidelines

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