

Infective Endocarditis

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart has four valves that open and close to keep blood flowing in the right direction. Each valve is lined with a smooth, slippery layer called the endocardium (the inside skin of the heart). Normally this lining is so smooth that bacteria flowing by in the blood cannot stick. But if a valve is already damaged (from past rheumatic fever, wear and tear, or a birth defect) or if a person has an artificial valve, the surface becomes rough or uneven—like a scratched countertop instead of a polished one. When bacteria enter the bloodstream (from a dental cleaning, an infected cut, injecting drugs, or a medical procedure like a catheter or scope), they can now land on that rough spot and stick. Once stuck, the bacteria multiply and call in platelets and fibrin (the blood's clotting material) to form a lumpy, fragile clump called a vegetation. This clump keeps growing, damages the valve so it leaks, and sheds pieces into the bloodstream that travel to the brain, skin, kidneys, and other organs. The result is fever, feeling wiped out, a new heart murmur (the sound of blood leaking backward), and strange skin spots or painful finger bumps from those traveling clumps. The body tries to wall off the infection with inflammation and fever, and the heart tries to pump harder to make up for the leaky valve, but without antibiotics to kill the bacteria and sometimes surgery to replace the valve, the infection keeps spreading and the heart eventually fails.

What you might feel

- Fever that won't quit
- New heart murmur or a murmur that sounds different
- Tiny red or purple spots on the skin (petechiae) or painful red bumps on fingers and toes (Osler nodes)
- Painless red spots on the palms or soles (Janeway lesions)
- Splinter hemorrhages—tiny red lines under the nails
- Sudden stroke or patches of vision loss
- Shortness of breath and swollen ankles (heart failure)
- Night sweats and weight loss you didn't plan

- Joint pain or back pain
- Blood in the urine or sudden flank pain

Call the doctor right away if...

- 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

What the care team will do

- Obtain at least three sets of blood cultures from different sites before starting any antibiotics, spacing them 30 minutes apart
- Start IV antibiotics within one hour after blood cultures are drawn, using vancomycin plus ceftriaxone for empiric coverage
- Monitor temperature every 4 hours and immediately report new fever spikes or persistent fever after 72 hours of antibiotics
- Perform focused neurologic checks every 4 hours, looking for confusion, facial droop, arm drift, slurred speech, or vision changes
- Assess heart sounds every shift and immediately report new murmurs or changes in existing murmurs
- Monitor urine output hourly and check creatinine and BUN daily, reporting output below 0.5 milliliters per kilogram per hour
- Watch for sudden severe back pain, abdominal pain, or cold painful legs, and check pedal pulses every shift
- Maintain strict IV site care using sterile technique, change tubing per protocol, and use antimicrobial-coated central lines if long-term access is needed

Medicines you may be given — and why

Vancomycin

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.

Ceftriaxone

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.

Gentamicin

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.

Nafcillin or Cefazolin (for methicillin-sensitive *Staphylococcus aureus*, MSSA)

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.

Penicillin G or Ceftriaxone (for viridans group streptococci and *Streptococcus gallolyticus*)

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.

Ampicillin plus Gentamicin or Ampicillin plus Ceftriaxone (for *Enterococcus* species)

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.

Where this information comes from

- 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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