Community-Acquired Pneumonia

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THE STORY IN ONE SENTENCE

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“The lung's exchange counter is underwater. Every breath still moves air in and out—but the oxygen can't reach the blood.”

Normal anatomy — the body's job

Air moves down the trachea (windpipe), into the bronchi (the two main branch-tubes), and into smaller and smaller tubes until it reaches the alveoli—millions of tiny, thin-walled sacs wrapped by capillaries (the smallest blood vessels). Oxygen crosses that paper-thin wall into the blood; carbon dioxide crosses out. Mucus and cilia (tiny hairs that sweep like brooms) clean the airway on the way in. White blood cells stand guard to catch germs.

BODY'S JOB

Move air to the alveoli and let oxygen cross into the blood without inflammation or liquid getting in the way.

1. Nose and mouth — Warms, adds moisture, and filters incoming air.
2. Trachea (windpipe) — A single tube carrying air down into the chest.
3. Bronchi (branch-tubes) — Split into right and left main airways—one for each lung.
4. Bronchioles (smaller tubes) — Keep branching into narrower and narrower passages.
5. Alveoli (air sacs) — Millions of tiny balloon-like sacs—this is where oxygen actually crosses over into the blood.
6. Capillaries (tiny blood vessels) — Blood picks up oxygen and drops off carbon dioxide through the alveolar wall.

7. Blood returns to the heart and pumps out to the body — Fresh oxygen is delivered to every organ.

What breaks

A bacterium or virus makes it past the nose, mucus, cilia, cough, and immune cells and lands in some alveoli. The immune system floods those alveoli with neutrophils (the white blood cells that eat bacteria), plasma (the liquid part of blood), and inflammatory signals. The flooded alveoli look solid-white on a chest X-ray (called consolidation) and cannot do gas exchange anymore.

> **CLINICAL TERM**
> Alveolar filling with inflammatory leaked fluid (exudate) causes ventilation/blood flow (perfusion) (V/Q) mismatch and low oxygen in the blood (hypoxemia) (low oxygen in the blood). Most common pathogens: Streptococcus pneumoniae, Haemophilus influenzae, Mycoplasma pneumoniae, respiratory viruses (influenza, RSV, SARS-CoV-2).

How the body compensates

The body senses low oxygen and speeds up breathing to move more air past the healthy alveoli that still work. The heart rate rises to deliver more blood to the parts of the lung that are still clear. The immune system pours in more neutrophils and raises body temperature to help kill the germ—good for fighting the infection but worse for gas exchange in the short term because more alveoli fill with liquid.

1. Breathing rate speeds up (tachypnea) to compensate for lost gas-exchange surface.
2. Heart rate rises (tachycardia) to deliver more blood to working alveoli.
3. White blood cell count climbs (leukocytosis with neutrophil predominance).
4. Fever and shivering ramp up to boost the immune fight and make it harder for bacteria to grow.
5. If untreated: the flooded area grows, oxygen drops further, and the person may need supplemental oxygen or a ventilator to breathe.

Domino effect

1. Bacteria or virus enters the lung (by breathing it in, by mouth germs slipping into the airway during sleep, or rarely by spreading through the bloodstream). — The nose, cilia, mucus, cough reflex, and alveolar macrophages (the white cells that patrol the air sacs) are breached or overwhelmed.

2. The germ starts to multiply inside the alveoli. — The air sacs are warm, moist, and rich in nutrients—a perfect place for bacteria to grow.
3. Alveolar macrophages detect the germ and release cytokines (chemical alarm signals); neutrophils (infection-fighting white cells) and plasma flood into the alveoli. — The immune system is walling off and killing the invader.
4. Alveoli fill with leaked fluid (exudate) (a mix of white cells, dead bacteria, and liquid)—they cannot do gas exchange. — Liquid replaces air; oxygen cannot cross a flooded membrane.
5. Oxygen level in the blood drops (hypoxemia). — Fewer working alveoli means less surface for oxygen to cross over.
6. The body compensates: breathing speeds up, heart rate rises, and fever appears. — Trying to move more air through healthy alveoli, deliver more blood, and heat up to slow bacterial growth.
7. Cough (often bringing up yellow, green, or rust-colored coughed-up mucus (sputum)), chest pain (pleuritic—sharp with each breath), and fatigue show up. — Airway irritation triggers cough; inflammation of the pleura (the lining around the lung) causes sharp chest pain; low oxygen and the immune battle drain energy.
8. If untreated: bacteria can spill into the bloodstream (bacteremia) causing sepsis (body-wide inflammation and organ failure), the lung can collapse from pus in the pleural space (empyema), or breathing can fail entirely (respiratory failure requiring a ventilator). — Severe cases overwhelm local defenses and spread the immune storm and infection throughout the body.

Symptoms · what the patient feels, and why

- Cough that brings up thick, green or yellow mucus: The infection fills the tiny air sacs (alveoli) in the lung with fluid and pus. The body tries to cough it all out to clear the airway.
- Fever with shaking chills: The immune system raises the body's temperature to slow down germs and speed up infection-fighting cells. The shaking happens when the body's thermostat suddenly jumps higher.
- Shortness of breath and fast breathing: Infected air sacs fill with fluid instead of air, so less oxygen can cross into the blood. The body breathes faster to try to make up for it.
- Sharp chest pain that gets worse when you breathe in deeply or cough: The infection spreads to the pleura (the thin lining around the lung). When inflamed, the two layers rub together with each breath, causing sharp pain.
- Extreme tiredness and weakness: Fighting infection takes huge energy. Plus, less oxygen reaches muscles and the brain because the lungs aren't working well.

- Confusion or changes in thinking (especially in older adults): Low oxygen in the blood and inflammatory chemicals (cytokines released by the immune system) change how the brain works.
- Low oxygen level on pulse oximeter (SpO_2 below 92%): So many air sacs are flooded that not enough oxygen can pass from the lungs into the bloodstream.
- Low blood pressure and altered mental status together: The infection has spread into the bloodstream (sepsis). Inflammatory chemicals cause blood vessels to leak and widen, dropping pressure. The brain doesn't get enough blood flow.

Focused exam

- Crackles (rales) on lung listening with a stethoscope (auscultation): Tiny air sacs (alveoli) are filled with pus or fluid; when they pop open during the breath, you hear crackles. This is the classic exam sign that pneumonia is in the lungs.
- Decreased breath sounds + dullness to percussion: The lung is either filled with solid infection (consolidation) or surrounded by fluid (effusion). Either way, air can't move through that part. Dull sound when you tap means something thick is blocking the normal hollow echo.
- Respiratory rate > 22 breaths per minute, oxygen saturation (SpO_2) < 92%: The infected lung can't move oxygen into the blood well. The body tries to make up for it by breathing faster. Oxygen below 92% means the lungs are failing to do their job and the person needs extra oxygen right away.
- Blue lips or fingertips (cyanosis), using neck and shoulder muscles to breathe, or leaning forward with hands on knees (tripod position): The person is running out of oxygen and the breathing muscles are exhausted. This is a sign that the lungs are about to stop working (respiratory failure). The person needs ICU care immediately, possibly a breathing tube.
- New confusion or change in thinking: In older adults, confusion may be the ONLY sign of pneumonia — sometimes even more obvious than cough or fever. It happens because low oxygen and the body's stress response (infection signals) scramble brain function.

Diagnostics · every test answers a question

- Chest X-ray (posterior-anterior and lateral views) → Is there a white patch (consolidation) in the lung? Is there fluid around the lung (effusion)? Is there a hole or air pocket (cavitary lesion)?
- Pulse oximetry ± arterial blood gas (ABG) → Is oxygen getting into the blood? Is carbon dioxide (the waste gas) building up because the lungs can't blow it out?
- Complete blood count (CBC) with differential → Is the immune system fighting? Are there enough white blood cells (WBCs), and what kind are they?

- Blood cultures × 2 sets (from different sites, before antibiotics if possible) → Has the infection spread into the bloodstream (bacteremia)?
- Coughed-up mucus (Sputum) Gram stain and culture → What germ is causing the pneumonia? Will it respond to the antibiotic we started?
- Respiratory viral panel (influenza A/B, RSV, SARS-CoV-2) → Is this pneumonia caused by a virus instead of bacteria?
- Serum lactate and procalcitonin → Is the body in shock (not enough blood reaching the tissues)? Is this bacterial or viral?
- Urine antigen tests (Streptococcus pneumoniae, Legionella pneumophila serogroup 1) → Is the pneumonia caused by pneumococcus or Legionella? (These germs shed proteins in the urine.)

Treatment logic

- Empiric antibiotics (outpatient: amoxicillin 1 g three times daily, or doxycycline 100 mg twice daily; hospitalized non-ICU: ceftriaxone 1–2 g daily + azithromycin 500 mg daily; ICU: ceftriaxone + azithromycin, or a respiratory fluoroquinolone like levofloxacin) → Kills the most common bacteria that cause community-acquired pneumonia (Streptococcus pneumoniae, Haemophilus influenzae, atypical germs like Mycoplasma and Legionella) while you wait for culture results. Starts working within hours and reduces the risk of death.
- Supplemental oxygen (nasal cannula 1–6 L/min → high-flow nasal cannula up to 60 L/min → non-invasive positive pressure ventilation (BiPAP/CPAP) → intubation and mechanical ventilation) → Raises the amount of oxygen in the blood while the infected lung heals. Keeps the brain, heart, and kidneys fed with oxygen so they don't fail.
- Intravenous (IV) fluids (normal saline or lactated Ringer's, typically 30 mL/kg bolus in sepsis, then maintenance rate) → Restores blood volume and blood pressure if the person is dehydrated or in septic shock. Thins thick mucus in the airways so it's easier to cough up. Helps deliver antibiotics and oxygen to the tissues.
- Fever reducers (acetaminophen 650–1,000 mg every 6 hours, or ibuprofen 400–600 mg every 6–8 hours) → Lowers fever and makes the person more comfortable. Reduces how hard the body is working (metabolic demand), which can help if the heart or lungs are struggling.
- Chest physiotherapy, incentive spirometry (breathing exercises), and upright positioning → Helps the person cough up thick mucus (secretions). Opens up collapsed or sticky air sacs (atelectasis). Sitting upright lets the diaphragm (main breathing muscle) work better and gravity helps drain secretions.

- Corticosteroids (e.g. methylprednisolone 0.5 mg/kg/day or prednisone 50 mg daily for 5–7 days in severe CAP) → Calms the body's overwhelming immune response (inflammation) in the lungs. May shorten time on oxygen and reduce risk of respiratory failure in severe cases. Evidence is mixed; use is selective (IDSA 2019 suggests considering in refractory septic shock).
- Vaccination (pneumococcal: PCV15/PCV20 or PPSV23; influenza yearly; COVID-19 per CDC schedule) → Prevents the majority of severe bacterial and viral pneumonias in adults, especially in those over 65 or with chronic illness. PCV20 covers the 20 most common Streptococcus pneumoniae strains. Flu and COVID vaccines prevent the viruses that often lead to secondary bacterial pneumonia.

Medications

Amoxicillin (± azithromycin) · Beta-lactam antibiotic (± macrolide antibiotic)

Amoxicillin punches holes in the bacteria's outer wall so the germ bursts and dies. Azithromycin adds cover for sneaky bugs (like Mycoplasma and Chlamydia) that hide inside cells and don't have the same kind of wall.

1. Amoxicillin sticks to tiny pegs called penicillin-binding proteins on the bacteria's wall.
2. Those pegs normally help lock wall pieces together (cross-linking).
3. Without the pegs working, the wall can't hold its shape.
4. Water rushes in and the bacterium pops like a water balloon.
5. Azithromycin slips into atypical bacteria and grabs the 50S piece of the ribosome.
6. The ribosome can't make new proteins, so the bug can't grow or survive.

> **SAFETY WATCH**
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> Always ask about penicillin allergy before giving amoxicillin—some people get hives or even breathing trouble. · Azithromycin can stretch out the heart's QT wave (the reset phase on an EKG), which raises the risk of a dangerous rhythm in older adults or people on other QT-stretching drugs—check an EKG first in those cases. · Stomach upset (nausea, loose stool) is common with both drugs. · Very rare: severe skin rash (Stevens-Johnson syndrome) or liver irritation.

Doxycycline · Tetracycline antibiotic

Doxycycline sneaks into bacteria and jams the ribosome (the germ's protein-making machine) so the bug can't build the proteins it needs to live. Works well against both typical bacteria and the atypical bugs that hide inside cells.

1. Doxycycline enters the bacterium and locks onto the 30S part of the ribosome.

2. The ribosome reads genetic instructions (mRNA) to build proteins.
3. Doxycycline blocks the messenger from attaching, so no new proteins get made.
4. Without proteins, the bacteria can't repair themselves or multiply, and they die off.

> **SAFETY WATCH**
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> Can make skin very sensitive to sun—wear sunscreen and a hat. · Take with a full glass of water and stay upright for 30 minutes to avoid throat irritation (esophagitis). · Nausea is common; taking it with food helps (but avoid dairy, calcium, or iron within 2 hours—they block absorption). · Never use in pregnancy or kids under 8—can permanently yellow teeth and slow bone growth. · Rare: severe skin reaction (photo-onycholysis, nail lifting) if you get a lot of sun.

Ceftriaxone (± azithromycin) · Third-generation cephalosporin (± macrolide antibiotic)

Ceftriaxone breaks the bacteria's cell wall so the germ bursts. It's stronger than amoxicillin and covers more bacteria, so it's used when someone is sick enough to need IV antibiotics in the hospital. Azithromycin adds cover for atypical bugs.

1. Ceftriaxone grabs onto penicillin-binding proteins in the bacterial wall.
2. Those proteins normally glue wall pieces together.
3. Without the glue, the wall can't hold pressure.
4. The bacterium swells and bursts.
5. Azithromycin locks onto the 50S ribosome piece in atypical bugs and stops protein-making so they can't survive.

> **SAFETY WATCH**
>
> Ask about penicillin or cephalosporin allergy—about 10 percent of people allergic to penicillin may also react to ceftriaxone. · Can cause loose stool or diarrhea (including C. diff colitis if used for a long time). · Rare: gallbladder sludge (stones) in babies or people on very high doses. · Check azithromycin QT safety as above.

Levofloxacin or Moxifloxacin · Fluoroquinolone antibiotic

Levofloxacin and moxifloxacin twist and snap the bacteria's DNA so the germ can't copy itself or fix damage. They cover both common bacteria and atypical bugs in one pill, so they're used when someone can't take beta-lactams or needs single-drug coverage.

1. Bacteria unwind their coiled DNA using an enzyme called DNA gyrase (topoisomerase II).
2. The fluoroquinolone locks onto the gyrase while it's holding the DNA open.

3. The DNA breaks and can't re-seal properly.
4. The bacterium can't copy its genes or repair itself, so it dies.

SAFETY WATCH

Black-box FDA warnings: can rupture tendons (especially the Achilles), damage nerves (peripheral neuropathy), worsen myasthenia gravis, cause mood changes or confusion. · Avoid in people over 60, those on steroids, or anyone with tendon trouble. · Can stretch the QT interval—check EKG in older adults or those on other QT drugs. · Nausea, headache, and dizziness are common. · Rare but serious: aortic aneurysm or tear (don't use in people with known aneurysm or Marfan syndrome). · Can raise blood sugar in diabetics and lower it in others.

Oseltamivir (Tamiflu) · Neuraminidase inhibitor (antiviral for influenza)

Oseltamivir traps brand-new flu virus copies inside the infected cell so they can't escape and spread to healthy cells. It shortens flu symptoms by about 1 day if started within 48 hours of getting sick, and it helps prevent severe pneumonia when flu is the cause or a partner infection.

1. After the flu virus copies itself inside a cell, new virus particles bud off the cell surface.
2. Budding uses an enzyme called neuraminidase to cut the virus free.
3. Oseltamivir blocks neuraminidase.
4. New virus particles stay stuck to the cell and can't infect neighbors.
5. Your immune system catches up and clears the infection before it spreads further.

SAFETY WATCH

Nausea and vomiting are the most common side effects—taking it with food helps. · Rare reports of confusion, hallucinations, or unusual behavior in children and teens (though it's hard to tell if that's the flu itself or the drug). · Safe in pregnancy and recommended if a pregnant person has flu. · Adjust dose in people with kidney disease.

Red flags

- Oxygen saturation below 90% on room air, or needing more and more oxygen → Give more oxygen right away. Consider moving to the ICU and using non-invasive breathing support like BiPAP.
- Signs of sepsis: blood pressure below 90 systolic, blood lactate above 2, confusion or very sleepy → Start the sepsis bundle within the first hour: IV fluids, blood cultures, and broad antibiotics immediately.

- Large pocket of fluid around the lung (pleural a fluid collection (effusion)) or pus in that space (empyema) → Insert a chest tube to drain the fluid. Send a sample to the lab to check for bacteria.
- Hole or cavity inside the lung on chest X-ray or CT → Broaden antibiotics to cover mouth bacteria and resistant bugs. Isolate the patient and test for TB. Call pulmonology.
- No improvement after 48 to 72 hours on the right antibiotics → Repeat chest imaging. Review culture results. Consider a CT scan and switch to broader antibiotics.

Nursing priorities

1. Assess respiratory status and oxygen saturation every 2–4 hours — Deterioration can be silent — early O_2 escalation prevents intubation.
2. Give antibiotics on time — first dose within 4 hours of arrival — Every hour of delay increases mortality.
3. Position: head of bed 30–45°, encourage incentive spirometry — Opens collapsed alveoli, improves gas exchange.
4. Encourage hydration, monitor for fluid overload — Dehydration thickens secretions; over-hydration worsens swelling (edema).
5. Screen for sepsis at every reassessment — Pneumonia is the #1 cause of sepsis in older adults.
6. Educate on vaccine follow-up (pneumococcus, flu, COVID) — Cuts recurrence and severity of the next episode.

Sources

- ATS/IDSA Community-Acquired Pneumonia Guideline 2019
- IDSA/ATS Hospital-Acquired and Ventilator-Associated Pneumonia Guideline 2016
- Cochrane systematic reviews on pneumonia treatment
- CDC pneumonia prevention and treatment resources

Last reviewed: 2026-02-28