

COVID-19

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THE STORY IN ONE SENTENCE

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“COVID-19 means spotting who is sliding toward oxygen failure and stopping the spiral before the lungs fill with fluid and clots.”

Normal anatomy — the body's job

Your airways and lungs are lined with special cells that catch germs, make mucus to trap them, and sweep them out with tiny waving hairs (cilia). Deep in the lungs, millions of thin air sacs (alveoli) let oxygen jump from air into your blood and carbon dioxide (waste gas) jump out — like a screen door that only good air can pass through.

BODY'S JOB

The airway lining (respiratory epithelium) is your first defense: it makes a sticky mucus blanket that traps viruses and bacteria, then the cilia wave together like a crowd doing the wave, pushing the mucus up and out so you swallow it or cough it out. The alveoli are where the magic happens — their walls are only one cell thick, right next to tiny blood vessels (capillaries), so oxygen hops across in a split second and your blood carries it to every organ. At the same time, carbon dioxide (the waste your cells make) hops the other way into the alveoli so you can breathe it out. This swap must happen smoothly and fast, every breath, or your organs run out of fuel.

1. Air enters through your nose or mouth, where hairs and mucus catch big particles.
2. It flows down the trachea (windpipe) into two main bronchi (tubes), one for each lung.
3. Each bronchus branches into smaller and smaller tubes (bronchioles), like a tree spreading into twigs.

4. At the end of the smallest tubes sit clusters of alveoli (air sacs), covered in capillaries (tiny blood vessels).
5. Oxygen crosses the thin alveoli wall into the blood; carbon dioxide crosses back out.
6. Fresh oxygenated blood returns to the heart, which pumps it to your brain, muscles, and every cell.
7. Cilia and mucus sweep out trapped germs and dust back up to your throat, where you swallow or cough them away.

What breaks

The SARS-CoV-2 virus locks onto a doorway called ACE2 on airway and lung cells, slips inside, and makes thousands of copies. The infected cells die and burst, spilling virus onto neighbors. Your immune system charges in with white blood cells and chemicals (cytokines) that cause swelling and mucus in the airways — that is the cough and shortness of breath. If the virus spreads into the deep lung air sacs, immune swelling fills them with fluid (pneumonia), so oxygen cannot cross into your blood. In the sickest patients, the immune response goes too far (cytokine storm), the air sacs collapse, and clots form in lung blood vessels, choking off oxygen completely (ARDS — acute respiratory distress syndrome, meaning the lungs fail).

CLINICAL TERM

SARS-CoV-2 binds ACE2 receptors on respiratory epithelium and type II pneumocytes (cells that line alveoli), enters via endocytosis, replicates, and lyses the host cell. Viral replication triggers innate immune activation: interferons, IL-6, IL-1 β , TNF- α , and chemokines recruit neutrophils and macrophages, causing airway inflammation (tracheobronchitis) and alveolar damage. Cytokine dysregulation (cytokine storm) leads to diffuse alveolar damage, hyaline membrane formation, capillary leak, and V/Q mismatch (ventilation/blood flow (perfusion) mismatch — air sacs filled with fluid cannot load oxygen into blood). Endothelial injury and hypercoagulability cause microthrombi in pulmonary vasculature, worsening low oxygen in the blood (hypoxemia). Severe cases progress to ARDS with bilateral infiltrates, low oxygen in the blood refractory to supplemental oxygen, and multi-organ dysfunction from low oxygen (hypoxia) and systemic inflammation.

How the body compensates

Your body tries to keep oxygen flowing by breathing faster and deeper, pumping your heart harder, and clotting more to seal off damaged vessels. These backup moves can keep your vital signs looking okay for a while, even as the lungs are filling with fluid — that is the dangerous middle, because it hides how close you are to crashing. The crash point is when your lungs run

out of healthy air sacs, your oxygen drops despite fast breathing, and clots or multi-organ failure tip you over the edge.

1. {'label': 'Fast breathing (Tachypnea) (fast breathing)', 'mechanism': 'Low blood oxygen (hypoxemia) triggers chemoreceptors in your brain and neck arteries, which command your diaphragm to contract faster and deeper — you breathe 20, 30, even 40 times per minute to try to pull in more oxygen and blow off carbon dioxide.'}
2. {'label': 'Increased work of breathing', 'mechanism': 'Swollen, stiff lungs (low compliance) require more muscle force to inflate; you use accessory muscles (neck, ribs, belly) and you may see retractions (skin sucking in between ribs) and nasal flaring — this burns energy and tires you out.'}
3. {'label': 'A fast heart rate (Tachycardia) (fast heart rate)', 'mechanism': 'Your heart pumps faster (100–120+ bpm) to deliver the little oxygen you have to starving organs — this keeps blood pressure stable for a while but eventually exhausts the heart.'}
4. {'label': 'Vasoconstriction and shunting', 'mechanism': 'Blood vessels in fluid-filled lung areas clamp down (hypoxic vasoconstriction) to redirect blood to healthier alveoli; your body also shunts blood to the brain and heart first, sacrificing skin, gut, and kidneys (you look pale, cold, mottled).'}- 5. {'label': 'Hypercoagulability', 'mechanism': 'Inflammation activates clotting proteins (fibrinogen, Factor VIII, von Willebrand factor) and platelets stick together more — this tries to seal off leaky, damaged blood vessels but creates clots (DVT, PE, stroke) as a dangerous side effect.'}
- 6. {'label': 'Fever and immune activation', 'mechanism': 'Cytokines (IL-1, IL-6) reset your hypothalamus thermostat higher to speed up immune reactions and slow viral replication — fever is your body fighting, but it also increases oxygen demand and worsens fatigue.'}
- 7. {'label': 'The dangerous middle', 'mechanism': 'All these compensations can keep your oxygen saturation at 90–94%, heart rate at 100, and blood pressure normal even while half your lungs are filled with fluid — you look stable, but you are one step from crashing. The crash comes when compensation maxes out: breathing exhausts you (fatigue, CO₂ rises), heart cannot keep up (shock), or a clot cuts off blood flow (PE, stroke).'}- 8. {'label': 'The crash point', 'mechanism': 'Oxygen saturation drops below 88% despite high-flow oxygen, respiratory rate climbs above 30, you cannot speak full sentences, confusion sets in (brain low oxygen (hypoxia)), blood pressure falls (shock), or you stop breathing (respiratory arrest) — this is ARDS and multi-organ failure; without ICU support (mechanical ventilation, pressors), death follows quickly.'}

Domino effect

1. SARS-CoV-2 binds ACE2 and invades airway cells — The virus lands on your nose, throat, or lung lining and locks onto ACE2 receptors (the front door). It slips inside, hijacks the cell's factory, and makes thousands of virus copies. The cell bursts open, spilling virus onto neighbor cells — this is the first domino that starts the whole cascade.
2. Upper airway inflammation: sore throat, cough, fever, loss of smell — Infected cells die and release danger signals. Your immune system sends white blood cells and chemicals (cytokines) that cause swelling and mucus. Swollen airways trigger cough and sore throat. Inflammation in the nose blocks smell receptors — you lose your sense of smell (anosmia). Cytokines reset your brain's thermostat higher — you get a fever. At this stage, most people stop here and recover in 5–7 days.
3. Virus spreads deeper: pneumonia (lung air sacs fill with fluid) — If your immune system cannot stop the virus in the upper airways, it travels down the bronchioles into the alveoli (air sacs). Infected alveolar cells die, and inflammation makes capillaries leak fluid and protein into the air sacs (like a wet sponge filling with water). Oxygen cannot jump across the thick, fluid-filled wall into your blood — your oxygen saturation drops, you breathe faster and harder, and chest X-ray shows white patches (infiltrates). This is pneumonia.
4. Cytokine storm: immune system overreacts and attacks everything — In some patients (especially older adults, people with diabetes, obesity, or lung disease), the immune response spins out of control. Macrophages and T cells dump massive amounts of IL-6, IL-1 β , TNF- α (inflammatory chemicals) into the bloodstream. This cytokine storm damages not just virus-infected cells but healthy tissue too — blood vessels leak everywhere, blood pressure drops (shock), and organs (heart, kidneys, liver, brain) start to fail from inflammation and low oxygen.
5. ARDS: lungs stiffen, oxygen saturation crashes despite high-flow oxygen — Widespread alveolar damage (diffuse alveolar damage) causes the air sacs to collapse, fill with debris, and form thick membranes (hyaline membranes) that block oxygen transfer. The lungs become stiff and heavy (like trying to inflate a leather bag instead of a balloon). Even 100% oxygen through a mask cannot raise your oxygen saturation above 88% because the alveoli are not working — this is ARDS (acute respiratory distress syndrome). Without a ventilator to force air in and hold the alveoli open (positive pressure), you cannot survive.

6. **Hypercoagulability:** blood clots form in lungs, legs, brain — Inflammation activates your clotting system (tissue factor, platelets, fibrinogen) and damages the endothelium (inner lining of blood vessels), making it sticky. Tiny clots (microthrombi) form in lung capillaries, worsening oxygen transfer. Bigger clots form in leg veins (DVT — a clot in a deep leg vein (deep vein thrombosis)), break off, and travel to the lungs (PE — pulmonary embolism (a clot lodging in a lung artery)), choking off blood flow and oxygen. Clots can also block brain arteries (stroke), causing sudden weakness or speech loss. COVID-19 patients have 2-6 times higher clot risk than other pneumonias.
7. **Multi-organ failure:** heart, kidneys, brain, gut all suffer — Low oxygen (hypoxemia) and systemic inflammation hit every organ. The heart muscle swells (myocarditis) and pumps weakly, blood pressure drops (cardiogenic shock). Kidneys cannot filter waste (acute kidney injury, AKI) — you make little urine, toxins build up. Brain gets low oxygen and inflammatory signals — confusion, delirium, or unresponsiveness (encephalopathy). Gut lining is damaged (you get diarrhea, belly pain). Liver enzymes rise. This is multi-organ dysfunction syndrome (MODS) — the final domino before death if not reversed in the ICU.
8. **Long COVID and MIS-C:** delayed problems weeks to months later — Some adults never fully recover — they have fatigue, shortness of breath, brain fog, and exercise intolerance for months (Long COVID). Mechanisms are unclear but likely include lingering virus bits (antigens), autoimmunity (antibodies attacking self), and microvascular damage (tiny blood vessels stay inflamed). In children, 2-6 weeks after mild infection, some develop MIS-C (Multisystem Inflammatory Syndrome in Children): high fever, rash, red eyes, belly pain, and shock from antibodies and immune complexes attacking heart and blood vessels — similar to Kawasaki disease. This can cause coronary artery aneurysms (dangerous bulges) if not treated urgently with IVIG (antibodies) and steroids.

Symptoms · what the patient feels, and why

- **Fever:** The immune system raises body temperature to fight the virus (a defense response that makes it harder for the virus to multiply).
- **Cough (usually dry):** The virus irritates the airways, triggering a reflex to try to clear them out.
- **Loss of smell or taste:** The virus infects the cells that support smell nerves in the nose, blocking signals from reaching the brain.
- **Shortness of breath or fast breathing:** Inflammation in the lungs (swelling in the tiny air sacs) makes it harder to pull oxygen into the blood.
- **Fatigue (feeling very tired):** The body uses a lot of energy to fight the infection, leaving less energy for normal activities.
- **Headache:** Inflammation chemicals released during the infection irritate pain-sensing nerves in the head.

- Belly pain, nausea, or diarrhea: The virus can infect cells lining the intestines, causing inflammation and disrupting normal digestion.
- Low oxygen level (hypoxia) without feeling very short of breath (silent low oxygen (hypoxia)): Lung inflammation reduces oxygen transfer, but the brain's alarm for breathlessness sometimes does not fire early enough.

Focused exam

- Oxygen level below 94% on room air, or patient is breathing fast or using neck muscles to breathe: COVID-19 damages the tiny air sacs in the lungs (alveoli) where oxygen crosses into blood. When oxygen drops below 94%, it means enough lung tissue is hurt that the body cannot pull in enough oxygen anymore. This is the clearest sign that the infection has moved deep into the lungs and is causing real danger right now.
- Dry cough that started 2–5 days ago: The virus lands in the nose or throat, grabs onto a doorway on cells called ACE2, and starts copying itself. The cough is the body trying to clear out infected airway cells. A dry cough (not bringing up mucus) is classic for COVID-19 because the virus attacks the lining of the airways before it fills them with fluid.
- Fever between 100°F and 103°F: Fever means the immune system has recognized the virus and is turning up the body's thermostat to slow down viral copying and speed up immune cells. Fever by itself does not tell you how sick someone is, but it confirms the body is fighting an infection.
- Cannot smell or taste anything, even though the nose is not blocked: COVID-19 attacks the support cells around smell nerves high in the nose, cutting off their ability to send signals to the brain. This finding is so specific to COVID-19 (compared to flu or cold) that it is one of the strongest clues pointing to this virus.
- Belly pain, nausea, or diarrhea (especially in children): ACE2 doorways are also found on cells lining the gut, so the virus can attack there too. When someone has belly symptoms along with fever and cough, it tells you the virus is hitting more than just the lungs and helps explain why some people feel sicker than their lung symptoms suggest.
- Oxygen level is low but the person does not feel very short of breath (silent low oxygen (hypoxia)): COVID-19 can quietly damage deep lung tissue and block oxygen from crossing into blood without triggering the normal alarm signals that make you feel breathless. This mismatch means you must always measure oxygen with a pulse oximeter — you cannot trust how the person feels alone.

Diagnostics · every test answers a question

- SARS-CoV-2 PCR (nasal swab) → Is COVID-19 virus present in the nose or throat right now?

- SARS-CoV-2 rapid antigen test (nasal swab) → Is there enough virus in the nose to be contagious and likely causing symptoms?
- Pulse oximetry (oxygen saturation, SpO₂) → Are the lungs getting enough oxygen into the blood?
- Chest X-ray → Are the lungs filling with fluid or showing patches of inflammation?
- C-reactive protein (CRP) → How much inflammation is the body making in response to the virus?
- D-dimer → Is the blood clotting system turned on, raising the risk of dangerous clots?
- Complete blood count (CBC) → Are white blood cells, red blood cells, and platelets at normal levels?
- Ferritin → Is there hidden, severe inflammation that could lead to organ damage?

Treatment logic

- Nirmatrelvir-ritonavir (Paxlovid) started within 5 days of symptom onset for high-risk patients (age ≥65, diabetes, obesity, immunocompromised) → Paxlovid blocks the enzyme the virus uses to copy itself inside human cells, cutting viral reproduction by about 90%. Giving it early (within 5 days) cuts the risk of hospitalization and death by roughly 50% in high-risk people because it stops the virus before it overwhelms the lungs.
- Remdesivir (antiviral IV infusion) for hospitalized patients or high-risk outpatients within 7 days of symptoms → Remdesivir jams the virus's copying machinery (RNA polymerase), slowing down how fast the virus spreads through the lungs. In hospitalized patients, it shortens recovery time by about 5 days and may reduce mortality when started early.
- Dexamethasone 6 mg once daily (oral or IV) for hospitalized patients requiring oxygen → Dexamethasone is a steroid that turns down the immune system's attack signals (cytokines), which stops the lungs from swelling so much and lets oxygen cross into blood better. It reduces death by about one-third in patients on oxygen and by one-fifth in patients on ventilators.
- Baricitinib (JAK inhibitor) or tocilizumab (IL-6 blocker) for hospitalized patients with rising oxygen needs despite steroids → These drugs block specific immune signals (JAK pathways or interleukin-6) that drive runaway inflammation. They help prevent progression to mechanical ventilation and lower mortality in patients whose lungs are still getting worse even on dexamethasone.

- Preventive (Prophylactic)-dose anticoagulation (blood thinner, usually low-molecular-weight heparin) for most hospitalized patients → COVID-19 triggers widespread clotting in small blood vessels, especially in the lungs. Blood thinners reduce the risk of pulmonary embolism (a clot lodging in a lung artery) (clot in lung arteries) and a clot in a deep leg vein (deep vein thrombosis) (clot in leg veins), which can suddenly drop oxygen levels or cause death.
- Supplemental oxygen (nasal cannula, high-flow nasal cannula, or non-invasive ventilation) to keep $\text{SpO}_2 \geq 92\%$ → Providing extra oxygen keeps vital organs (brain, heart, kidneys) supplied with oxygen while the lungs heal. It buys time for antivirals and immune modulators to work.
- Mechanical ventilation (intubation and ventilator) when oxygen levels stay below 90% despite maximum non-invasive support → A ventilator takes over the work of breathing, pushing air into the lungs with precise pressure and oxygen levels. This keeps oxygen flowing to the body when the patient's own breathing muscles and damaged lungs cannot do the job anymore.

Medications

Nirmatrelvir-ritonavir (Paxlovid) · Antiviral protease inhibitor combination

Nirmatrelvir blocks a scissors-like enzyme the COVID virus uses to cut big protein chains into working pieces inside your cells; without those pieces, the virus cannot make copies of itself. Ritonavir slows down the liver so nirmatrelvir stays in your blood longer and can keep fighting the virus.

1. COVID virus enters your cell and releases its instruction manual (RNA).
2. Your cell reads the manual and makes long chains of viral protein.
3. The virus has a special cutting tool (protease enzyme) that chops those chains into smaller working parts.
4. Nirmatrelvir locks onto that cutting tool and stops it from working.
5. Without the cut pieces, the virus cannot build new copies of itself.
6. Ritonavir blocks liver enzymes (CYP3A) that would normally break down nirmatrelvir, so the drug stays in your blood at a high enough level to keep blocking the virus.

SAFETY WATCH

Ritonavir interacts with dozens of common drugs (statins, blood thinners, seizure medicines, transplant drugs, some heart medicines) by changing how fast your liver breaks them down; your doctor must check every medicine you take before prescribing this. · Can cause a metallic or bitter taste in your mouth and diarrhea. · Not safe if your kidneys or liver are severely damaged. · Symptoms may come back (rebound) in about 2 to 8 out of every 100 people a few days after finishing the medicine; this is usually mild and goes away on its own.

Molnupiravir · Antiviral nucleoside analog

This drug looks like one of the building blocks (nucleosides) the virus uses to copy its genetic instruction manual (RNA). When the virus accidentally uses molnupiravir instead of the real block, it makes so many mistakes in the copies that the new viruses cannot work.

1. COVID virus enters your cell and starts copying its RNA instruction manual.
2. The virus uses an enzyme (RNA polymerase) that grabs building blocks to write new RNA.
3. Molnupiravir looks so much like a real building block that the enzyme grabs it by mistake.
4. Once molnupiravir is added to the new RNA strand, it causes errors every time the strand is copied again.
5. The errors pile up until the new virus copies are so broken they cannot infect other cells.

SAFETY WATCH

Should not be used during pregnancy because it could theoretically harm a developing baby by causing genetic errors; women who can become pregnant should use reliable birth control during treatment and for 4 days after the last dose. · Not recommended in people under 18 because bone and cartilage are still growing and animal studies showed possible harm. · May cause diarrhea, nausea, and dizziness.

Remdesivir · Antiviral nucleotide analog

Like molnupiravir, remdesivir is a fake building block. When the virus tries to copy its genetic instructions (RNA), remdesivir gets added to the new strand and then stops the copying machine from adding any more blocks, so the virus cannot finish making copies of itself.

1. COVID virus RNA polymerase (the copying machine) starts building a new RNA strand.
2. Remdesivir looks like the building block adenosine, so the enzyme adds it to the growing chain.
3. Once remdesivir is in place, the enzyme tries to add the next block but gets stuck and cannot continue.

4. The incomplete RNA strand is useless, so the virus cannot make working copies.

SAFETY WATCH

Can cause nausea and elevated liver enzymes (your doctor checks blood tests). · Rarely causes kidney problems or slow heart rate; hospital staff monitor you during the infusion. · Infusion reactions (rash, low blood pressure, trouble breathing) can happen but are uncommon.

Dexamethasone · Corticosteroid (immune suppressor)

Dexamethasone is a strong copy of cortisol, a hormone your body makes to calm down inflammation. It tells many immune cells to stop releasing chemicals (cytokines) that cause swelling, fluid leak, and damage in the lungs. This keeps the immune system from attacking your own lung tissue while it fights the virus.

1. COVID triggers a strong immune reaction; white blood cells flood the lungs and release chemicals (interleukins, TNF, and others) that cause inflammation.
2. These chemicals make tiny blood vessels leaky, so fluid fills the air sacs (alveoli) and oxygen cannot get into your blood (this is called ARDS—acute respiratory distress syndrome, or severe lung injury).
3. Dexamethasone enters immune cells and turns on genes that make calming proteins and turns off genes that make inflammatory chemicals.
4. Inflammation drops, blood vessels become less leaky, and the air sacs can start to clear, making it easier to breathe.

SAFETY WATCH

Can raise blood sugar (a problem for people with diabetes); blood sugar should be checked regularly. · Can cause confusion, mood swings, or trouble sleeping. · Increases the risk of new infections (bacterial, fungal) because it weakens the immune defense; watch for fever, new cough, or worsening symptoms. · Long-term use (weeks) can weaken bones, raise blood pressure, and cause stomach ulcers, but short courses (10 days or less) for COVID carry lower risk.

Baricitinib · JAK inhibitor (immune modulator)

Baricitinib blocks enzymes called JAK1 and JAK2 that sit inside immune cells and pass along signals from inflammatory messengers (cytokines) outside the cell. By blocking these enzymes,

the drug stops the cell from getting the 'attack' message, so the immune system calms down and causes less lung damage.

1. COVID infection causes immune cells to release many cytokines (IL-6, IL-1, interferons, and others).
2. These cytokines float to other cells and attach to receptors on the cell surface.
3. The receptor sends a signal inward through JAK enzymes, which then activate STAT proteins.
4. STAT proteins move into the cell nucleus and turn on genes that make even more inflammatory chemicals, creating a vicious cycle (cytokine storm).
5. Baricitinib blocks the JAK enzymes, so the signal never gets through and the cell stays calm.

SAFETY WATCH

Can raise the risk of serious infections (including tuberculosis reactivation, fungal infections, and herpes zoster/shingles) because it weakens part of the immune defense; screening for latent TB and hepatitis is often done before starting. · May increase the chance of blood clots (deep vein clot formation (thrombosis), pulmonary embolism (a clot lodging in a lung artery)), especially in people with other clot risk factors; many hospitalized COVID patients already get blood thinners, which helps reduce this risk. · Can cause low blood counts (fewer red cells, white cells, or platelets); blood tests are checked regularly. · May raise cholesterol levels over time.

Tocilizumab · Monoclonal antibody (IL-6 receptor blocker)

Tocilizumab is a lab-made antibody that sticks to the receptor for interleukin-6 (IL-6), a powerful inflammatory messenger. By covering the receptor, it stops IL-6 from delivering its 'attack' signal, so immune cells make less inflammation and the lungs take less damage.

1. Severe COVID triggers immune cells to pour out large amounts of IL-6.
2. IL-6 travels to many cells and binds to IL-6 receptors on their surface.
3. The receptor sends a signal into the cell that ramps up inflammation, causes fever, makes blood vessels leaky, and tells the liver to make acute-phase proteins.
4. Tocilizumab antibodies bind to the IL-6 receptor first, blocking IL-6 from attaching.
5. Without the IL-6 signal, cells produce less inflammation and the cytokine storm calms.

SAFETY WATCH

Increases the risk of serious bacterial, fungal, and viral infections because it dampens immune defense; watch for new fever, worsening breathing, or signs of infection. · Can cause infusion reactions (fever, chills, rash, low blood pressure) during or shortly after the IV dose. · May raise liver enzymes and lower white blood cell or platelet counts; blood tests are monitored. · Can mask fever, making it harder to detect a new infection early.

Heparin or low-molecular-weight heparin (enoxaparin) · Anticoagulant (blood thinner)

These drugs boost the activity of antithrombin, a natural protein in your blood that stops clotting factors (especially factor Xa and thrombin) from turning liquid blood into a solid clot. COVID makes blood stickier and more likely to clot inside vessels; heparin helps keep blood flowing smoothly.

1. COVID infection damages the lining (endothelium) of blood vessels and activates platelets and clotting factors.
2. The blood becomes hypercoagulable (extra sticky), and tiny clots (microthrombi) form in small vessels, blocking oxygen delivery.
3. Heparin molecules bind to antithrombin and change its shape so it works hundreds of times faster.
4. Supercharged antithrombin grabs and stops factor Xa and thrombin, two key enzymes in the clotting cascade.
5. New clots cannot form as easily, and existing small clots are less likely to grow.

SAFETY WATCH

Main risk is bleeding (bruising, blood in urine or stool, bleeding gums, or internal bleeding); the dose is carefully chosen to balance clot prevention and bleeding risk. · Can cause heparin-induced a low platelet count (thrombocytopenia) (HIT), a rare condition where the immune system attacks platelets and actually increases clotting; if platelet count drops significantly, the drug is stopped immediately. · Enoxaparin is cleared by the kidneys, so the dose must be reduced in people with kidney disease to avoid bleeding. · Rarely, can cause a mild allergic reaction at the injection site (redness, itching).

Monoclonal antibody therapies (bebtelovimab, and earlier sotrovimab, casirivimab-imdevimab, bamlanivimab-etesevimab—now largely inactive against current variants) · Monoclonal antibodies (passive immunotherapy)

These are lab-made copies of human antibodies that stick tightly to the spike protein on the surface of the COVID virus. By covering the spike, they prevent the virus from grabbing onto ACE2 receptors on your cells, so the virus cannot enter and cannot make copies of itself.

1. COVID virus uses its spike protein like a key to unlock ACE2 receptors (the lock) on your cells.
2. Monoclonal antibodies bind to the spike protein and cover the part (receptor-binding domain) that would normally attach to ACE2.
3. The virus can no longer grab onto cells, so it floats harmlessly in your blood and tissue fluid.
4. Your immune cells recognize the antibody-coated virus and clear it away faster.

SAFETY WATCH

Infusion reactions (itching, rash, fever, nausea, dizziness, or trouble breathing) can happen during or within 24 hours of the IV or subcutaneous dose; patients are observed for at least an hour. · Rarely, severe allergic reactions (anaphylaxis) can occur; emergency equipment is kept nearby. · Because most monoclonal antibodies no longer work against current variants, they are not routinely authorized or recommended as of 2024–2025; check the latest FDA and NIH guidelines before using.

Supplemental oxygen (nasal cannula, high-flow nasal cannula, non-rebreather mask, or mechanical ventilator) · Supportive therapy (not a drug)

COVID damages the air sacs (alveoli) in your lungs so oxygen cannot pass easily into your blood. Giving extra oxygen through a tube, mask, or breathing machine raises the amount of oxygen in the air you breathe (from the normal 21% up to 100%), which pushes more oxygen across the damaged lung membrane into your bloodstream and keeps your organs alive while your lungs heal.

1. COVID causes inflammation and fluid to fill the air sacs, creating a barrier between air and blood.
2. Oxygen in room air (21%) cannot cross that thick barrier fast enough to keep your blood oxygen level normal.
3. Delivering higher concentrations of oxygen increases the pressure gradient, driving more oxygen molecules across the damaged membrane.
4. Your blood oxygen level rises, and your heart, brain, kidneys, and other organs get enough oxygen to keep working.

5. In severe cases, positive pressure (from a ventilator) helps hold the air sacs open and recruits collapsed areas, improving oxygen exchange even more.

SAFETY WATCH

Nasal cannula and masks are safe and comfortable for most people. · High-flow nasal oxygen can dry out the nose and throat, so the oxygen is warmed and humidified. · Mechanical ventilation (breathing machine) requires a tube in your windpipe (intubation) and sedation; risks include ventilator-associated pneumonia, lung injury from pressure (barotrauma), and muscle weakness from prolonged sedation. · Giving too much oxygen can rarely suppress breathing drive in people with chronic lung disease (very uncommon in acute COVID).

COVID-19 vaccines (mRNA vaccines: Pfizer-BioNTech, Moderna; protein subunit: Novavax) · Vaccine (active immunization)

The vaccine delivers instructions (mRNA) or a piece of the spike protein itself into your body. Your immune system sees the spike protein (or the protein your cells make from the mRNA) as foreign, learns to recognize it, and builds an army of memory B cells and T cells. If the real virus enters later, those memory cells respond fast and stop the virus before it can cause serious illness.

1. mRNA vaccines: A tiny fat bubble (lipid nanoparticle) carries mRNA into your muscle cells. The cells read the mRNA and make copies of the COVID spike protein, then display pieces of it on their surface. Immune cells see the spike pieces, recognize them as foreign, and begin making antibodies and training T cells. The mRNA breaks down in a few days, but the memory cells last months to years.
2. Protein subunit vaccine: The vaccine contains lab-made spike protein along with an adjuvant (immune booster). Immune cells pick up the protein, process it, and present it to B and T cells, which learn to recognize and attack it.
3. When real COVID virus later enters your body, memory B cells quickly produce neutralizing antibodies that block the spike from attaching to ACE2, and memory T cells kill infected cells before the virus can spread widely.

SAFETY WATCH

Common side effects: sore arm, fatigue, headache, muscle aches, fever, and chills for 1–2 days (signs your immune system is responding). · Rare: severe allergic reaction (anaphylaxis) within minutes to hours, usually in people with a history of severe allergies; vaccination sites are equipped to treat this immediately. · mRNA vaccines: very rare risk of myocarditis (inflammation of the heart muscle) or pericarditis (inflammation of the sac around the heart), most often in males under 30 after the second dose; most cases are mild and resolve with rest and anti-inflammatory medicine. · All vaccines are monitored through national safety systems (VAERS, V-safe, Vaccine Safety Datalink) to detect and investigate any new safety signals.

Red flags

- Trouble breathing, chest pain, or blue lips or face (means oxygen is too low)
- Confusion or trouble waking up (means the brain is not getting enough oxygen)
- Sudden worsening around day 7 to 10 of illness (may signal severe lung inflammation or a clot forming)

Nursing priorities

1. Check oxygen saturation (SpO₂) with pulse oximeter every 4 hours and immediately if patient reports shortness of breath — COVID-19 damages the alveoli (tiny air sacs in lungs) where oxygen enters the blood. Oxygen levels below 94% mean the lungs are failing and the patient needs supplemental oxygen or higher level of care.
2. Place patient on contact and airborne precautions with N95 mask for staff and keep patient in negative-pressure room if available — SARS-CoV-2 spreads through tiny droplets (aerosols) that float in the air. Airborne precautions protect other patients and healthcare workers from catching the virus.
3. Monitor respiratory rate and work of breathing every 4 hours, watching for use of neck muscles, flaring nostrils, or inability to speak full sentences — Increased work of breathing means the lungs are stiff from inflammation (pneumonitis) and the patient is tiring out. This predicts respiratory failure and need for mechanical ventilation (breathing machine).
4. Encourage prone positioning (lying on stomach) for 2 to 4 hours at a time if oxygen is low but patient is not intubated — Proning improves ventilation-blood flow (perfusion) matching (gets oxygen to the parts of lung that still have good blood flow). This simple position change can raise oxygen levels 10 to 20 points and prevent need for intubation.

5. Administer dexamethasone 6 mg once daily exactly as ordered for hospitalized patients on oxygen, and never give steroids to patients not requiring oxygen — Dexamethasone reduces cytokine storm (overactive immune response causing lung damage) in patients with severe COVID-19. It lowers death risk by one-third. But giving steroids too early to mild cases may help the virus replicate and cause harm.
6. Assess and document venous thromboembolism (VTE) risk daily and ensure preventive (prophylactic) anticoagulation (blood thinner shots) is ordered for all hospitalized patients unless contraindicated — COVID-19 activates the clotting cascade (blood clotting system) and damages blood vessel walls. Without prevention, 20 to 30% of hospitalized patients develop dangerous clots in legs or lungs.
7. Monitor fluid balance closely and avoid giving too much IV fluid unless patient is truly dehydrated — COVID-19 pneumonia causes capillary leak (fluid leaking from blood vessels into lungs). Excess IV fluids worsen pulmonary edema (fluid flooding the lungs) (water in the lungs) and make oxygenation harder.
8. Screen daily for delirium (sudden confusion) using a validated tool and reorient patient frequently if confused — Low oxygen (Hypoxia) (low oxygen), systemic inflammation, and isolation cause delirium in up to 60% of hospitalized COVID-19 patients. Delirium increases fall risk, length of stay, and long-term cognitive problems.

Sources

- CDC COVID-19 Guidelines
- NIH COVID-19 Treatment Guidelines
- WHO COVID-19 Clinical Management
- AAP COVID-19 Interim Guidance

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