Meningitis

Bacteria have crossed into the subarachnoid space and turned the cerebrospinal fluid into an abscess.

THE STORY IN ONE SENTENCE

Bacteria have crossed into the subarachnoid space and turned the cerebrospinal fluid into an abscess.

“Bacterial meningitis is an infection in the fluid around the brain. Antibiotics cannot wait.”

Normal anatomy — the body's job

The blood-brain barrier (a tight seal in the blood vessels around the brain) keeps the cerebrospinal fluid (CSF) sterile. CSF is the clear, watery cushion that wraps the brain and spinal cord, absorbs shocks, delivers nutrients, and washes away waste. Small immune guard cells called microglia patrol the brain and handle tiny threats. This seal and cushion keep the brain clean and protected. When this job fails, bacteria break through and enter the CSF — that is the upstream break we are about to trace.

BODY'S JOB

The blood-brain barrier's job is to filter what can reach the brain, letting nutrients in and keeping germs out. CSF cushions the brain against bumps, carries oxygen and sugar to brain cells, and flushes out waste products into the bloodstream. Microglia sweep up debris and respond to small infections. If you don't know this normal job first, the weird findings in meningitis seem confusing.

1. Blood vessels around the brain have tight junctions (cells locked like bricks) that form the blood-brain barrier.
2. CSF is made in the choroid plexus (tiny sponge-like tissue inside the brain's fluid chambers) and flows around the brain and spinal cord in the subarachnoid space (the thin gap between two meningeal layers).
3. CSF drains back into the bloodstream through tiny valves called arachnoid granulations, completing a clean loop.

4. Microglia (resident brain immune cells) patrol for damage and germs; if they find a small threat, they eat it and signal for help.
5. This system keeps the brain sterile, cushioned, and nourished — like a closed aquarium with filters and guards.

What breaks

The blood-brain barrier breaks or gets bypassed, and bacteria enter the cerebrospinal fluid. Once inside, they multiply fast because CSF has almost no white blood cells or antibodies to stop them. The infection spreads through the fluid around the brain and spinal cord, turning the protective cushion into a swamp of bacteria and pus.

> **CLINICAL TERM**
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> Bacteria enter the CSF via three routes: hematogenous seeding (bloodstream spread from distant infection such as pneumonia or endocarditis), contiguous spread (direct extension from sinusitis, otitis media, or mastoiditis eroding through bone), or direct inoculation (traumatic skull fracture, neurosurgery, or VP shunt breach). CSF is immunologically privileged — it has low complement, low immunoglobulin, and few phagocytes — so bacteria proliferate rapidly. The meninges (pia, arachnoid, and dura) become inflamed, and neutrophils flood the subarachnoid space, forming pus-filled (purulent) leaked fluid (exudate). This break does not stay local. The downstream signal you will actually see at the bedside is meningeal irritation (headache, light sensitivity (photophobia), neck stiffness), fever, and altered mental status. Read all the weird findings backwards from there — they all point to this one broken thing upstream.

How the body compensates

The body fights back by flooding the meninges with white blood cells and inflammatory chemicals to kill bacteria. It tries to keep blood pressure and oxygen delivery steady. But this response itself causes symptoms — fever, stiff neck, headache, confusion — and can tip into brain swelling, seizures, and shock. Vital signs can look okay for a while as the body compensates, but the crash point comes fast once reserves run out.

1. {'step': "Fever spikes as the hypothalamus (the brain's thermostat) raises body temperature to make conditions harder for bacteria to survive.", 'why': "Cytokines (inflammatory alarm chemicals like IL-1 and TNF-alpha) reset the brain's temperature set-point higher, triggering chills and sweating as the body heats up."}
2. {'step': 'Neutrophils (bacteria-eating white blood cells) flood the CSF to surround and kill bacteria, forming pus.', 'why': 'The body tries to wall off the infection, but the flood of cells and chemicals also inflames the meningeal lining and raises pressure inside the skull.'}

3. {'step': 'Blood vessels in the brain dilate and leak fluid into tissue, trying to deliver more immune cells and oxygen, but this causes vasogenic swelling (edema) (brain swelling from leaky vessels).', 'why': 'The brain tissue swells and compresses itself against the rigid skull, raising intracranial pressure and reducing blood flow, which can lead to starved blood flow (ischemia) (tissue starving from lack of blood).'}
4. {'step': 'The heart pumps harder and faster to maintain blood pressure and deliver oxygen to the swelling brain.', 'why': 'This compensation works for a while, but if bacteria spill into the bloodstream and trigger septic shock, blood vessels leak everywhere, blood pressure crashes, and organs begin to fail.'}
5. {'step': "CSF production slows and drainage is blocked by pus, causing hydrocephalus (fluid backup in the brain's chambers).", 'why': 'Pressure inside the skull climbs, squeezing the brainstem (the control center for breathing and heart rate) and risking herniation (the brain pushes down through the skull opening, which is fatal).'}
6. {'step': 'Neurons fire chaotically from inflammation, low oxygen, and metabolic stress, causing seizures.', 'why': "Seizures are the brain's circuits short-circuiting under pressure and inflammation, not a primary infection of the neurons themselves."}
7. {'step': 'The crash point to watch for is: septic shock (blood pressure drops and organs fail), cerebral herniation (brain pushes down and breathing stops), or Waterhouse-Friderichsen syndrome (hemorrhage into both adrenal glands during meningococcemia, causing sudden adrenal crisis and shock).', 'why': 'These are the points of no return. Once the brain herniates or shock sets in, survival depends on seconds. The window to reverse the cascade closes fast.'}
8. {'step': 'How well someone does depends on how fast antibiotics are started and whether steroids are given early to dampen inflammation.', 'why': 'Every hour of delay before antibiotics increases mortality and the risk of permanent complications like hearing loss, brain damage, or limb amputation from clotting.'}
9. {'step': 'Watch the trend, not just one snapshot. A patient with stable vitals but worsening confusion, rising fever, or new seizures is on a downhill slope.', 'why': 'Compensation hides the real severity for a while. A stable patient on a downhill slope is not really stable — they are running out of reserves.'}

Domino effect

1. Bacteria enter the cerebrospinal fluid (CSF) through the bloodstream, a nearby infection, or a break in the skull or spine. — Common bacteria like Streptococcus pneumoniae, Neisseria meningitidis, and Listeria monocytogenes ride the bloodstream from pneumonia or skin infections, or crawl through from ear or sinus infections. Trauma or surgery can also open a direct path. Once inside, bacteria find a space with almost no immune defenses.

2. Bacteria multiply rapidly in the CSF, doubling every 20–30 minutes. — CSF normally has fewer than 5 white blood cells per microliter and low levels of antibodies and complement proteins (immune tools that tag and kill germs). Without these defenses, bacteria grow unchecked and spread through the subarachnoid space (the fluid-filled gap around the brain and spinal cord).
3. The body detects bacteria and floods the meninges (the protective layers around the brain) with neutrophils (bacteria-eating white blood cells) and releases inflammatory chemicals like TNF-alpha and interleukin-1. — This immune storm tries to kill bacteria and wall off the infection, but the flood of cells and chemicals inflames the meninges, causing severe headache, light sensitivity (photophobia) (light hurts the eyes), and a stiff neck (nuchal rigidity) (stiff neck from irritated meningeal lining pulling on neck muscles).
4. Inflammation makes blood vessels in the brain leaky and swollen, causing vasogenic swelling (edema) (brain swelling from fluid leaking out of vessels) and vasculitis (inflamed, narrowed blood vessels). — The brain tissue swells and presses against the rigid skull, raising intracranial pressure (ICP). Blood flow is reduced in inflamed vessels, causing starved blood flow (ischemia) (tissue starving from lack of oxygen). This leads to confusion, lethargy, and eventually coma as brain cells struggle.
5. Pus clogs the normal flow and drainage of CSF, causing obstructive hydrocephalus (backup of fluid in the brain's chambers). — CSF is made continuously in the choroid plexus and drains through tiny valves into the bloodstream. When pus blocks these pathways, pressure inside the skull climbs even higher, squeezing the brainstem (the control center for breathing and heart rate) and risking herniation (the brain pushes down through the skull opening, which is fatal).
6. Neurons fire chaotically under the stress of inflammation, low oxygen, and metabolic chaos, triggering seizures. — Seizures are the brain's circuits short-circuiting, not a direct infection of neurons. Cytokines (inflammatory chemicals), low glucose in the CSF, and starved blood flow (ischemia) all lower the seizure threshold and cause abnormal electrical storms in the cortex (the brain's outer layer).
7. If bacteria spill heavily into the bloodstream, they trigger septic shock: blood vessels leak, blood pressure crashes, and organs are starved of oxygen. — Endotoxins (toxins released when bacteria die) activate the immune system everywhere, causing widespread inflammation. Blood vessels dilate and leak fluid into tissues, blood pressure drops, and the heart cannot pump enough oxygen to the brain, kidneys, and liver. In meningococcemia (Neisseria meningitidis in the blood), this can also cause purple bleeding blotches (purpura) fulminans (massive clotting and bleeding in the skin and organs) and Waterhouse-Friderichsen syndrome (hemorrhage into both adrenal glands, causing sudden loss of stress hormones and fatal shock).

8. Every symptom — fever, headache, stiff neck, confusion, seizures — is the body's emergency response trying to wall off infection and keep the brain alive, not the infection itself. — The treatment must break the chain at multiple points: antibiotics (like ceftriaxone or vancomycin) kill the bacteria (the root cause), dexamethasone (a steroid) dampens the inflammatory overreaction and reduces hearing loss, and careful fluid management and ICP monitoring protect the brain from swelling and herniation. Each targets a different domino to stop the cascade before permanent damage or death.

Symptoms · what the patient feels, and why

- Fever: Bacteria or viruses in the meninges (the thin protective layers around the brain and spinal cord) trigger the immune system to release chemicals called pyrogens that reset the brain's thermostat higher, raising body temperature to try to fight the infection.
- Severe headache: The infection causes the meninges to swell and stretch, and extra fluid builds up in the tight space around the brain, pressing on pain-sensitive tissues and nerves.
- Stiff neck (cannot bend the neck forward comfortably): Inflamed meninges along the spinal cord tighten the muscles in the back of the neck reflexively to protect the irritated tissue, making it painful and difficult to flex the neck.
- Altered mental status (confusion, sleepiness, hard to wake up, or acting unlike themselves): Infection and swelling disrupt normal brain function, and toxins from bacteria or an overactive immune response interfere with the neurons (nerve cells) that control thinking, attention, and awareness.
- Petechial or purpuric rash (tiny red or purple spots that do not fade when you press on them): Neisseria meningitidis bacteria (meningococcus) can damage blood vessel walls and trigger clotting problems, causing blood to leak into the skin in small spots; this is a sign of meningococcemia (the bacteria spreading in the bloodstream).
- Light sensitivity (Photophobia) (eyes hurt or water in bright light): Inflamed meninges make the brain and nerves extra sensitive, so normal light levels feel painful because the optic nerve (the nerve that carries signals from the eye to the brain) and surrounding tissues are irritated.
- Nausea and vomiting: Increased pressure inside the skull from swelling and extra fluid irritates the vomiting center in the brainstem (the bottom part of the brain that controls automatic body functions).
- Positive Kernig sign (pain or resistance when the knee is straightened while the hip is bent): Stretching the leg pulls on the inflamed nerve roots and meninges along the spinal cord, causing sharp pain that makes the muscles resist.
- Positive Brudzinski sign (hips and knees bend automatically when the neck is flexed forward): Bending the neck stretches the inflamed spinal meninges, and the body reflexively bends the legs to slacken the spinal canal and reduce the painful pull.

- Seizures: Inflammation, swelling, and chemical imbalances around the brain lower the seizure threshold, making neurons fire in chaotic bursts instead of their normal coordinated pattern.

Focused exam

- Neck stiffness (cannot touch chin to chest): Bacteria in the cerebrospinal fluid (the clear liquid cushioning the brain and spinal cord) make the meninges (the protective layers around the brain and spine) swell and hurt. The neck muscles lock up to guard the spinal cord from being stretched. This is the signature sign of meningitis.
- Kernig sign (pain shoots down the leg when you straighten the knee while the hip is bent): Straightening the leg pulls on the swollen meninges through the nerves, causing sharp pain. This specific finding points directly to infection in the cerebrospinal fluid.
- Brudzinski sign (when you bend the neck forward, the knees pull up on their own): The inflamed meninges send a reflex signal through the spinal cord that automatically pulls the knees up to protect the spine from being stretched. Another meningitis-specific sign.
- Petechial or purpuric rash (tiny red or purple spots that do not fade when you press on them): Meningococcal bacteria (Neisseria meningitidis) release poisons that punch holes in blood vessel walls, causing bleeding into the skin. This rash means bacteria are flooding the bloodstream and septic shock (when blood pressure crashes and organs start failing) is minutes to hours away.
- Light sensitivity (Photophobia) (bright light hurts the eyes so much the patient hides from it): The swollen meninges irritate the optic nerve (the nerve that carries signals from the eye to the brain) and make it fire too easily. Pure sign of meningeal inflammation.
- Altered mental status (confused, very sleepy, or unresponsive): Bacteria and the immune response cause the brain itself to swell (cerebral swelling (edema)). Swelling squeezes brain cells and disrupts the electrical signals between them. This means the infection has spread beyond the meninges into the brain tissue.

Diagnostics · every test answers a question

- Lumbar puncture with cerebrospinal fluid (CSF) analysis: cell count, glucose, protein, Gram stain, and culture → Is there infection in the cerebrospinal fluid, and is it bacterial, viral, or something else?
- Blood cultures (two sets from different sites, drawn before antibiotics) → Are bacteria in the bloodstream, and which ones?
- Serum procalcitonin → Is this bacterial or viral? Procalcitonin rises sharply only when bacteria release toxins.

- CSF polymerase chain reaction (PCR) for herpes simplex virus (HSV) and enteroviruses → Is this actually herpes or a common virus pretending to be bacterial meningitis?
- Serum lactate → Is the body in septic shock? When bacteria flood the blood, oxygen delivery crashes and cells switch to emergency energy-making that produces lactate.
- Head CT scan before lumbar puncture (only if altered mental status, new seizure, focal neurologic signs, or concern for mass) → Is the brain so swollen or shifted that doing a lumbar puncture could cause the brainstem to herniate (get squeezed down through the skull opening)?

Treatment logic

- Ceftriaxone 2 grams IV every 12 hours PLUS vancomycin 15–20 mg/kg IV every 8–12 hours, started within 30 minutes of suspicion (ideally before lumbar puncture if any delay) → Kills Streptococcus pneumoniae (the most common cause, a tough bacteria that hides in a thick capsule), Neisseria meningitidis (the one that causes the purple rash and septic shock), and covers drug-resistant strains that shrug off weaker antibiotics. Stops bacteria from multiplying in the cerebrospinal fluid before the infection triggers unstoppable brain swelling.
- Ampicillin 2 grams IV every 4 hours added to the regimen if the patient is over 50 years old, pregnant, or has a weakened immune system → Covers Listeria monocytogenes, a bacteria that hides in deli meats and soft cheeses and sneaks into the brain in older adults and people with weak immune systems. Ceftriaxone and vancomycin completely miss Listeria.
- Dexamethasone 10 mg IV given 15–20 minutes before or at the same time as the first antibiotic dose → Blocks the inflammatory cascade triggered when antibiotics break open bacterial cell walls and spill toxins into the cerebrospinal fluid. Reduces swelling of the meninges and brain (cerebral swelling (edema)), lowers pressure inside the skull, and cuts the risk of hearing loss and brain damage. Interrupts the second domino (the immune overreaction) without touching the first (the bacteria themselves).
- Acyclovir 10 mg/kg IV every 8 hours if herpes simplex encephalitis cannot be ruled out yet (e.g., focal neurologic signs, temporal lobe changes on imaging, or CSF red blood cells) → Stops herpes simplex virus from copying itself inside brain cells. Herpes encephalitis looks identical to bacterial meningitis at first but needs a completely different drug. Covers this dangerous look-alike while waiting for CSF PCR results.
- Elevate the head of the bed 30 degrees, keep carbon dioxide in the normal range ($PaCO_2$ 35–40 mmHg by adjusting the ventilator if intubated), treat fever aggressively, and give sedation or pain control as needed → Lowers pressure inside the skull (intracranial pressure) by improving blood flow out of the brain, preventing the brain from herniating (getting squeezed down through the skull opening). Buys time for antibiotics to work and prevents secondary injury from swelling.

- Report confirmed or suspected meningococcal meningitis to public health immediately and give prevention (prophylaxis) (rifampin 600 mg by mouth every 12 hours for 2 days, or ciprofloxacin 500 mg by mouth once) to close contacts within 24 hours → Clears Neisseria meningitidis bacteria from the nose and throat of family members, roommates, and healthcare workers before the bacteria seed their bloodstream and cause a second case.

Medications

Ceftriaxone · Third-generation cephalosporin antibiotic

Ceftriaxone punches holes in the walls that bacteria need to stay alive. Without a strong wall, the bacteria burst and die. It works especially well against the most common germs that cause meningitis—Streptococcus pneumoniae (the pneumonia bug) and Neisseria meningitidis (the meningococcal bug)—because it crosses from the bloodstream into the fluid around the brain and spinal cord even when inflammation is present.

1. Ceftriaxone travels through the blood and crosses into the cerebrospinal fluid where bacteria are multiplying.
2. It locks onto proteins in the bacterial cell wall called penicillin-binding proteins.
3. This stops the bacteria from building new wall pieces as they try to grow and divide.
4. The weak, incomplete wall cannot hold the bacteria together, so they rupture and die.
5. The immune system then clears the dead bacteria and debris from the CSF.

> **SAFETY WATCH**
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> Allergic reactions: Some people allergic to penicillin may also react to ceftriaxone; watch for rash, swelling, or trouble breathing. · Do NOT mix ceftriaxone with calcium-containing IV fluids in newborns—it can form dangerous crystals. · Can cause diarrhea, including severe infection with Clostridioides difficile (C. diff) if used for a long time. · Rarely causes gallbladder sludge or stones, especially in children or adults getting high doses.

Vancomycin · Glycopeptide antibiotic

Vancomycin attacks a different part of the bacterial wall than ceftriaxone does, blocking the bacteria from linking wall pieces together. It is added to ceftriaxone because some strains of Streptococcus pneumoniae have become resistant to other antibiotics, and vancomycin can still kill them. Together, ceftriaxone and vancomycin cover almost all the bacteria that cause bacterial meningitis in adults and children.

1. Vancomycin enters the bloodstream and crosses into the inflamed meninges and CSF.
2. It binds tightly to the building blocks (D-alanyl-D-alanine) that bacteria use to weave their cell wall.

3. This stops the bacteria from linking new wall pieces into the growing wall.
4. Without a complete, strong wall, the bacteria cannot survive and they break apart.
5. The immune system removes the debris.

SAFETY WATCH

Red man syndrome: If vancomycin is given too fast through the IV, it can cause flushing, itching, and a red rash on the face, neck, and chest. Slow the infusion to prevent this. · Kidney damage: Vancomycin can harm the kidneys, especially if given with other drugs that stress the kidneys or if the patient is dehydrated. Blood tests monitor kidney function. · Hearing loss: Rarely, vancomycin can damage the inner ear and cause ringing in the ears or permanent hearing loss, especially at high doses or in people with kidney problems. · Check vancomycin blood levels (trough) to keep the dose effective but safe.

Ampicillin · Penicillin-type antibiotic

Ampicillin is added to the antibiotic mix when the patient is over 50 years old, pregnant, or has a weakened immune system (from cancer, HIV, transplant drugs, or steroids) because these groups are at higher risk for Listeria monocytogenes meningitis. Listeria is resistant to ceftriaxone and vancomycin, but ampicillin kills it by breaking down its cell wall. Ampicillin also covers some other bacteria, making the initial treatment as broad as possible until lab tests identify the exact germ.

1. Ampicillin travels from the blood into the CSF.
2. It binds to penicillin-binding proteins on the bacterial wall.
3. This stops the bacteria from cross-linking the wall pieces as they grow.
4. The wall becomes weak and the bacteria burst from internal pressure.
5. Dead bacteria are cleared by the immune system.

SAFETY WATCH

Allergic reactions: Anyone allergic to penicillin should not get ampicillin; reactions range from rash to life-threatening anaphylaxis (throat swelling, shock). · Ampicillin can cause a bright red, flat rash in people with mononucleosis (mono); this rash is not a true allergy. · Diarrhea is common; severe or bloody diarrhea may signal C. diff infection. · Rarely can cause seizures if very high doses are used or if kidney function is poor.

Acyclovir · Antiviral nucleoside analog

Acyclovir is started immediately if the doctor cannot yet rule out herpes simplex virus (HSV) encephalitis, a viral brain infection that looks very similar to bacterial meningitis at first but needs antiviral treatment, not antibiotics. HSV encephalitis can cause fever, confusion,

seizures, and brain swelling; without acyclovir, it can lead to permanent brain damage or death. Acyclovir works by sneaking into infected brain cells and stopping the virus from copying its DNA, which halts the infection.

1. Acyclovir enters the bloodstream and crosses into the brain and CSF.
2. Inside cells infected by HSV, the virus's own enzyme (thymidine kinase) accidentally activates acyclovir.
3. Activated acyclovir looks like a natural DNA building block, so the virus tries to use it when copying its genetic code.
4. Once acyclovir is inserted into the growing viral DNA chain, the chain stops—no more copying can happen.
5. The virus cannot multiply, and the infection is controlled while the immune system clears infected cells.

> **SAFETY WATCH**
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> Kidney damage: Acyclovir can form crystals in the kidneys if the patient is dehydrated or the drug is given too fast. IV fluids are given alongside acyclovir to protect the kidneys. · Neurologic side effects: Rarely, acyclovir can cause confusion, tremors, hallucinations, or seizures, especially in people with kidney problems where the drug builds up. · Nausea and vomiting are common. · Check kidney function before starting and monitor during treatment.

Dexamethasone · Corticosteroid (synthetic steroid hormone)

Dexamethasone is a powerful anti-inflammatory steroid given before or with the first dose of antibiotics to reduce brain swelling and damage in bacterial meningitis, especially pneumococcal meningitis caused by Streptococcus pneumoniae. When bacteria die from antibiotics, they release toxins and debris that trigger a massive inflammatory response in the tight space around the brain. This inflammation can damage nerves, cause hearing loss, and lead to stroke or death. Dexamethasone dampens that inflammatory surge, protecting the brain and reducing the risk of long-term disability like deafness or cognitive problems.

1. Dexamethasone enters the bloodstream and quickly reaches the inflamed tissues around the brain.
2. It enters immune cells and binds to receptors inside the cells.
3. This binding turns off genes that produce inflammatory chemicals like cytokines (chemical messengers that ramp up inflammation) and prostaglandins.
4. Blood vessel walls become less leaky, so less fluid leaks into the brain tissue and less swelling occurs.

5. Immune cells release fewer enzymes and toxins that would otherwise damage brain tissue and nerves, especially the auditory nerve (the nerve for hearing).

SAFETY WATCH

High blood sugar: Dexamethasone can spike blood glucose, especially in people with diabetes; blood sugar must be monitored and treated. · Weakened immune response: Because it suppresses the immune system, dexamethasone can make it harder to fight off other infections if used for a long time, but the short course used in meningitis (2–4 days) is generally safe. · Stomach irritation: Can cause stomach pain or bleeding, especially if the patient is also on NSAIDs (ibuprofen, naproxen). · Mood changes, insomnia, and increased appetite are common even with short-term use. · Should be given before or with the first antibiotic dose for maximum benefit; if started late, the benefit is less clear.

Red flags

- Altered mental status, confusion, or hard to wake up
- Seizure
- Petechial or purpuric rash that does not blanch (does not turn white when pressed)
- Signs of shock (very low blood pressure, fast heart rate, cold hands and feet, pale or mottled skin)
- Focal neurologic deficits (weakness, numbness, or vision loss on one side)

Nursing priorities

1. Obtain blood cultures and ensure antibiotics start within 30 minutes of arrival, before any imaging or lumbar puncture — Every hour of antibiotic delay increases mortality by 8 percent; blood cultures before antibiotics help identify the bacteria later but treatment cannot wait for test results
2. Administer dexamethasone 10 mg IV before or with the first antibiotic dose — Dexamethasone (a steroid) reduces inflammation in the subarachnoid space and decreases hearing loss and death in bacterial meningitis, but only works if given early before antibiotics
3. Monitor neurological status every 1 to 2 hours using Glasgow Coma Scale and pupil response — Rising intracranial pressure (ICP) from brain swelling and pus in CSF causes herniation (brain pushes through skull openings), which kills patients; early signs include decreased alertness and unequal pupils

4. Elevate the head of the bed to 30 degrees and keep the patient's head midline — Head elevation helps venous blood drain from the brain through jugular veins, which lowers intracranial pressure; turning the head to the side kinks the veins and raises pressure
5. Place the patient in droplet precautions with a mask until bacterial meningitis is ruled out or 24 hours of antibiotics are complete — Neisseria meningitidis and Streptococcus pneumoniae spread through respiratory droplets; isolation protects staff and other patients from catching life-threatening infection
6. Maintain normothermia with acetaminophen or cooling blankets, targeting temperature below 38°C (100.4°F) — Fever increases the brain's metabolic demand for oxygen and raises intracranial pressure; each degree Celsius of fever worsens brain swelling and secondary injury
7. Give IV fluids to maintain euvolemia with normal saline, avoiding both dehydration and fluid overload — Dehydration drops cerebral blood flow (perfusion) pressure (the pressure pushing blood into the brain), but overhydration worsens cerebral swelling (edema) (brain swelling); goal is normal blood pressure and urine output
8. Minimize stimulation by dimming lights, reducing noise, and clustering care activities — The inflamed meninges make the brain hypersensitive; stimulation triggers pain and can raise intracranial pressure through agitation and increased metabolic demand

Sources

- IDSA Clinical Practice Guidelines for Bacterial Meningitis 2004 (updated 2017)
- CDC Meningitis Guidelines
- Cochrane Acute Bacterial Meningitis Reviews
- WHO Guidelines on Meningitis

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