

Newborn jaundice

All newborns have some yellow color in their skin from bilirubin, because their liver is still learning its job and red blood cells are breaking down faster than before birth.

THE STORY IN ONE SENTENCE

All newborns have some yellow color in their skin from bilirubin, because their liver is still learning its job and red blood cells are breaking down faster than before birth.

“Newborn yellowing of the skin and eyes (jaundice) — most is normal, some crosses into the brain and causes permanent damage, and the bilirubin curve shows you which.”

Normal anatomy — the body's job

Red blood cells in a newborn break down faster than before birth, releasing bilirubin. The liver wraps that bilirubin in a water-friendly coat (conjugation), sends it into the bile, and the bile flows into the gut. Bacteria in the gut finish breaking it down so it leaves in the stool. A newborn's liver is slower and the gut bacteria are just arriving, so the system runs slower than an adult's.

BODY'S JOB

The liver's job is to grab bilirubin from the blood, attach a sugar tag to it so it dissolves in water, and dump it into bile. The bile flows into the small intestine, where gut bacteria turn the bilirubin into a form that leaves in the poop. This keeps bilirubin from building up and crossing into the brain.

1. Red blood cells break down → bilirubin (yellow waste) is released into the blood.
2. Bilirubin floats in the blood stuck to a protein (albumin) so it can travel.
3. The liver grabs bilirubin, attaches a sugar tag to it (conjugation), and the tagged bilirubin becomes water-soluble.
4. The liver dumps the tagged bilirubin into bile, which flows through bile ducts into the small intestine.
5. Gut bacteria break down the bilirubin into a form called urobilinogen and stercobilin, which leaves in the stool and gives it the normal brown color.

6. If any step slows down or breaks, bilirubin stacks up in the blood and the skin turns yellow.

What breaks

Yellowing of the skin and eyes (Jaundice) happens when bilirubin is made too fast for the liver to handle, or the liver and gut are too slow or blocked to clear it. Either way, bilirubin piles up in the blood.

CLINICAL TERM

Newborn yellowing of the skin and eyes (jaundice) results from an imbalance between bilirubin production and elimination. Production rises because fetal red blood cells have a shorter lifespan and hemolyze faster after birth. Elimination is slower because hepatic UDP-glucuronosyltransferase (the enzyme that conjugates bilirubin) is immature, gut flora are not yet established, and enterohepatic recirculation is higher. When production exceeds clearance, unconjugated (lipophilic) bilirubin accumulates in the blood. If the level exceeds the binding capacity of albumin or if the blood-brain barrier is compromised, free bilirubin crosses into the brain and deposits in the basal ganglia, hippocampus, and brainstem nuclei, causing kernicterus (acute bilirubin encephalopathy progressing to chronic neurologic injury).

How the body compensates

The body cannot compensate for high bilirubin. There is no feedback loop to speed up the liver or block bilirubin from crossing into the brain. Vital signs stay normal until kernicterus begins, so you must track the bilirubin number and the baby's behavior — sleepiness and poor feeding are the only early warnings before permanent damage sets in.

1. {'step': 'The body tries to bind bilirubin to albumin in the blood.', 'why': 'Albumin grabs bilirubin so it stays in the bloodstream and doesn't cross into tissues. This works until all the albumin binding sites are full or something knocks bilirubin off.'}
2. {'step': 'No other backup exists.', 'why': 'Unlike other metabolic problems, there is no hormone or reflex to speed up the liver or block bilirubin from entering the brain. The only protection is keeping the blood level below the threshold where free bilirubin rises.'}
3. {'step': 'Heart rate, blood pressure, and temperature stay normal even as bilirubin climbs.', 'why': 'Bilirubin is a toxin to the brain, not a shock or infection signal, so vital signs don't change until the baby is critically ill from kernicterus.'}
4. {'step': 'The first clue is behavior: the baby becomes sleepy, floppy, and feeds poorly.', 'why': 'Early bilirubin damage to the brainstem and basal ganglia dulls reflexes and arousal before it destroys cells completely. This is the narrow window to intervene.'}

5. {'step': 'If not treated, the baby then becomes stiff and arches backward (opisthotonus).', 'why': 'Continued bilirubin toxicity kills neurons in the basal ganglia, flipping muscle tone from floppy to rigid and locking the baby into abnormal postures.'}
6. {'step': 'Finally, breathing and temperature control fail.', 'why': 'Brainstem nuclei that control vital functions die, and the baby becomes critically ill or dies.'}
7. {'step': 'The crash point to watch for is kernicterus.', 'why': 'Once bilirubin crosses into the brain and starts killing neurons, the damage is permanent. You must catch it in the sleepy, floppy phase — not wait for stiffness or seizures.'}
8. {'step': 'Track the bilirubin curve over time, not just one number.', 'why': 'A level that is safe today can become dangerous tomorrow if it is rising fast. Use the Bhutani nomogram (hour-specific bilirubin zones) to see if the baby is on a safe or risky trajectory.'}

Domino effect

1. Red blood cells break down faster than the newborn liver can clear bilirubin. — Fetal red blood cells have a shorter lifespan (70–90 days vs. 120 in adults) and hemolyze faster after birth. At the same time, the liver enzyme UDP-glucuronosyltransferase is still ramping up, so the liver is slow to tag and dump bilirubin. This mismatch means bilirubin stacks up in the blood.
2. Unconjugated (untagged) bilirubin rises in the blood. — The liver hasn't attached the sugar tag yet, so this bilirubin is greasy (lipophilic) and can dissolve into fat-rich tissues like the brain. Most of it binds to albumin and stays in the blood, but if production is too high or albumin is too low, free bilirubin starts to rise.
3. Free bilirubin crosses the blood-brain barrier and enters the brain. — The blood-brain barrier normally blocks large or water-loving molecules, but unconjugated bilirubin is small and greasy, so when the free fraction climbs — either because total bilirubin is very high or something displaces it from albumin (sepsis, too much acid in the blood (acidosis), certain drugs) — it slips through and dissolves into the fatty myelin sheaths and cell membranes of neurons.
4. Bilirubin deposits in the basal ganglia, hippocampus, and brainstem nuclei, poisoning neurons. — These brain regions are rich in fat and have high metabolic activity. Bilirubin disrupts mitochondria (the cell's power plants), triggers inflammation, and kills neurons directly. The basal ganglia control movement, and the brainstem nuclei control breathing, swallowing, eye movement, and arousal.
5. The baby becomes sleepy, floppy (hypotonic), and feeds poorly (acute bilirubin encephalopathy, early phase). — Damaged brainstem nuclei send weak or garbled signals, so the baby loses muscle tone, suck-swallow coordination fails, and arousal drops. This is the warning phase — the damage is starting but not yet permanent if treated fast.

6. The baby then becomes stiff (hypertonic), arches backward (opisthotonus), and may have a high-pitched cry or seizures (intermediate phase). — Continued bilirubin toxicity kills more neurons in the basal ganglia (especially the globus pallidus and subthalamic nucleus), causing loss of inhibitory control over muscles. Opposing muscle groups contract at once, locking the baby into rigid, arched postures. The cry becomes shrill because laryngeal control is lost.
7. If untreated, the baby progresses to seizures, pauses in breathing (apnea), temperature instability, and death or permanent disability (advanced phase and chronic kernicterus). — Brainstem damage spreads to the respiratory and temperature-control centers. The baby cannot breathe on their own, cannot regulate body heat, and may die. Survivors are left with choreoathetoid cerebral palsy (writhing, involuntary movements), sensorineural hearing loss (auditory nerve damage), upward gaze palsy (eye-movement nerve damage), and intellectual disability.
8. None of this is random — each step forces the next because bilirubin is directly poisoning brain cells, and there is no way to reverse neuron death once it happens. — The sequence is biochemical and anatomical. High bilirubin → free bilirubin crosses into brain → neurons die → circuits break → permanent disability. The only way to stop it is to lower bilirubin before free levels rise into the toxic range, using phototherapy (light breaks down bilirubin in the skin), IV immunoglobulin (for immune red blood cell destruction (hemolysis)), or exchange transfusion (replace the baby's blood with donor blood to drop bilirubin fast).

Symptoms · what the patient feels, and why

- Yellow color in the skin and the whites of the eyes (jaundice): Bilirubin (a yellow waste made when old red blood cells break down) builds up faster than the baby's liver can clean it out. A newborn's liver is still learning its job, so bilirubin can pile up and stain the skin and eyes yellow.
- Yellowing that starts in the first 24 hours of life: When yellowing of the skin and eyes (jaundice) appears this early, it usually means red blood cells are breaking apart too fast — often because the mother's and baby's blood types don't match (like Rh or ABO mismatch) or because the baby has an infection. The flood of bilirubin overwhelms the newborn liver right away.
- Yellow color that spreads down below the chest and belly, reaching the legs and arms: Bilirubin moves from the bloodstream into the skin. It appears first in the face, then spreads downward as the level in the blood climbs higher. When you see yellow below the belly button, it means the bilirubin level is getting dangerously high.

- Baby is feeding poorly or skipping feeds: Very high bilirubin can make a baby sluggish and weak, so they lose interest in eating. Also, if the baby isn't getting enough milk, they make less stool — and bilirubin leaves the body mainly through stool — so the level climbs even higher.
- Baby is much more sleepy than usual, hard to wake up, or very floppy: When bilirubin reaches a very high level, it can cross into the brain and slow down brain signals. The baby becomes too drowsy to wake for feeds and loses normal muscle tone.
- High-pitched cry that sounds different from the usual newborn cry: Bilirubin irritating parts of the brainstem (the base of the brain that controls breathing and reflexes) can change the baby's cry, making it sound shrill or piercing — a sign that the brain is under stress.
- Arching of the head and neck backward (opisthotonus): Severe bilirubin toxicity in the basal ganglia (deep brain centers that control movement) and the brainstem causes the muscles along the back to tighten and pull the baby's body into a stiff, arched posture.
- Pale or clay-colored stool and dark urine that lasts beyond the first few days: If bile (which carries bilirubin) cannot flow out of the liver because of a blockage (like biliary atresia, where the bile ducts are missing or scarred shut), bilirubin backs up into the blood instead of coloring the stool brown. The stool turns pale, and bilirubin spills into the urine, making it dark.

Focused exam

- Yellow skin that started in the first 24 hours of life: Yellowing of the skin and eyes (Jaundice) this early means red blood cells are breaking down too fast — much faster than a newborn liver can handle. This is red blood cell destruction (hemolysis) (red cells popping open), not the slow, normal yellowing of the skin and eyes most babies get a few days later. The fast breakdown dumps too much bilirubin (the yellow waste from old red cells) into the blood before the liver can clean it out.
- Yellow color spreads down the body — face first, then chest, then belly, then legs and feet: The farther down the yellow goes, the higher the bilirubin number. Yellow that reaches the feet means bilirubin is dangerously high — often above 15-20 mg/dL — and getting close to the level where it can cross into the brain and damage the basal ganglia (the brain's movement-control center). This is the body's map of how bad things are.
- Baby is very sleepy, hard to wake, or won't latch and feed well: These are the first warning signs that bilirubin is starting to poison the brainstem (the part of the brain that keeps you awake and breathing) and the basal ganglia. Sleepiness and poor feeding come before the baby gets stiff or arches. Missing this means missing the window to stop kernicterus (permanent brain damage from bilirubin).

- High-pitched cry or arching of the back and neck (opisthotonos): These mean bilirubin has already damaged the basal ganglia and brainstem. This is kernicterus happening right now. The damage is starting and may become permanent — causing lifelong movement problems, hearing loss, and trouble thinking.
- Cephalohematoma (blood clot between the skull bone and the scalp) or visible bruising from delivery: These pockets of trapped blood are full of red blood cells that will break down over the next few days. Each broken cell releases bilirubin. This extra load feeds more yellow into the system, making yellowing of the skin and eyes (jaundice) worse and longer. It is an upstream source that the liver cannot keep up with.
- Baby not gaining weight or losing more than 10% of birth weight by day 3: Poor feeding means not enough milk is moving through the gut. Without milk, bilirubin sits in the intestines and gets reabsorbed back into the blood instead of leaving in the poop. This is breastfeeding yellowing of the skin and eyes (jaundice) — a type where the body recycles the yellow waste it should be throwing out. The cycle feeds itself: bilirubin makes the baby sleepy, sleepy babies feed poorly, poor feeding traps more bilirubin.
- Pale stools (white or clay-colored) and dark urine after the first few days of life: Pale stools mean bilirubin is not leaving the liver and reaching the gut. Dark urine means bilirubin is backing up and spilling into the kidneys instead. This points to biliary obstruction — the tubes that carry bile out of the liver are blocked or missing. The most urgent cause is biliary atresia (the bile ducts never formed right), which needs surgery before 8 weeks or the liver will scar permanently.

Diagnostics · every test answers a question

- Total serum bilirubin (TSB) and direct (conjugated) bilirubin → How high is the bilirubin, and is it the kind the liver has processed (direct) or the kind still waiting to be cleaned (indirect)?
- Blood type and Coombs test (direct antiglobulin test, DAT) on baby and mother → Is the mother's immune system attacking the baby's red blood cells because their blood types do not match?
- Complete blood count (CBC) with reticulocyte count → Are red blood cells breaking down too fast, and is the bone marrow working overtime to replace them?
- G6PD enzyme level (glucose-6-phosphate dehydrogenase) → Does the baby have a hidden genetic weakness that makes red blood cells fragile and easy to break?
- Thyroid function tests (TSH and free T4) → Is the baby's thyroid working? A low thyroid slows the whole body down, including the liver's ability to process bilirubin.
- Abdominal ultrasound → Are the bile ducts and gallbladder present and normal, or are they missing or blocked?

- Transcutaneous bilirubin (TcB) screening → Can we estimate the bilirubin level through the skin without drawing blood, especially for quick screening or repeated checks?

Treatment logic

- Phototherapy (blue-spectrum light at 460–490 nm wavelength) → Blue light hits bilirubin sitting in the baby's skin and changes its shape into lumirubin and other forms that dissolve in water. These new forms can leave the body through the kidneys (in urine) and gut (in stool) without needing the liver to process them first. This drops the bilirubin level and stops it from crossing into the brain. Intensive phototherapy uses multiple lights or a fiberoptic blanket wrapped around the baby to cover more skin and work faster.
- Optimize breastfeeding frequency (8–12 times per day) or add formula supplementation → More milk in the gut means more stool, and bilirubin leaves the body in poop. Milk also speeds up gut movement, which flushes bilirubin out before it can be reabsorbed back into the blood. This directly lowers the bilirubin level by cutting off the recycling loop. Lactation support — helping the mother with latch, positioning, and milk supply — is key. If breastfeeding is not working yet, formula supplementation (even 10–20 mL after each breastfeeding attempt) keeps the gut moving and the bilirubin dropping.
- Intravenous immunoglobulin (IVIG, 0.5–1 g/kg over 2 hours) → IVIG floods the baby's blood with normal antibodies that block and distract the bad antibodies attacking the baby's red blood cells. This stops immune red blood cell destruction (hemolysis) — fewer red cells break, so less bilirubin gets made. It is used when the Coombs test is positive (ABO or Rh incompatibility) and bilirubin is rising fast despite phototherapy. IVIG can reduce the need for exchange transfusion by cutting the source of bilirubin at the root.
- Exchange transfusion (double-volume exchange: 160–200 mL/kg over 2–4 hours) → Exchange transfusion removes the baby's blood in small batches and replaces it with donor blood. This pulls out the bilirubin already in the bloodstream and removes the antibody-coated red cells that are causing red blood cell destruction (hemolysis). It is the emergency brake — used when bilirubin is at or near the level where brain damage starts (usually > 20–25 mg/dL in a term baby, or lower if the baby is premature or sick), or when phototherapy and IVIG have failed to stop a fast rise. It drops the bilirubin by 40–50% immediately.
- Kasai portoenterostomy (hepatportoenterostomy) surgery for biliary atresia → Surgery removes the blocked or scarred bile ducts outside the liver and sews a loop of intestine directly onto the liver. This creates a new drainage path for bile to leave the liver and reach the gut, bypassing the missing or damaged ducts. If done before 8 weeks of life, it restores bile flow in 60–80% of babies and prevents the liver from scarring into cirrhosis. The earlier it is done (ideally before 60 days), the better it works.

- Ursodeoxycholic acid (ursodiol, 10–20 mg/kg/day in 2–3 divided doses) → Ursodiol is a bile acid that makes bile thinner and easier to flow. It protects liver cells from toxic bile and helps bile drain out of the liver. It is used after Kasai surgery for biliary atresia, or in babies with other causes of cholestasis (bile not flowing), to reduce liver inflammation and slow scarring. It also helps with itching caused by bile acids building up in the skin.
- Fat-soluble vitamin supplementation (vitamins A, D, E, K) for cholestasis → When bile is not flowing into the gut, the gut cannot absorb fats or the vitamins that dissolve in fat (A, D, E, K). Without these vitamins, babies develop bleeding (low vitamin K), weak bones (low vitamin D), vision problems (low vitamin A), and nerve damage (low vitamin E). Giving water-soluble forms or high doses of these vitamins bypasses the bile problem and prevents deficiency complications.

Medications

Phototherapy (blue light treatment) · Non-drug light therapy

Bright blue light (wavelength 460–490 nanometers) shines through the baby's skin and changes the shape of bilirubin (the yellow pigment) into a form called lumirubin, which the kidneys can wash out in urine without needing the liver. This skips the normal liver pathway and lowers the level of bilirubin in the blood before it can climb high enough to hurt the brain.

1. Blue light photons hit unconjugated bilirubin molecules floating in blood under the skin.
2. The light energy twists the bilirubin molecule into isomers (lumirubin and photobilirubin), which are water-soluble.
3. The kidneys filter these new forms straight into urine.
4. Bilirubin levels drop, keeping the brain safe from toxic buildup (kernicterus).

SAFETY WATCH

Dehydration: the light and extra urine loss can dry the baby out, so nurses watch weight and wet diapers closely. · Bronze baby syndrome: rare dark gray-brown skin color if direct bilirubin is high; the therapy is usually stopped. · Eye protection: the baby's eyes are covered with soft patches because bright light can hurt the retina. · Temperature swings: the lights can warm or cool the baby, so temperature is checked often. · Loose stools: faster gut movement from the light; usually harmless.

Intravenous immune globulin (IVIG) · Immune modulator (pooled human antibodies)

IVIG is a mix of antibodies collected from thousands of healthy donors. When a mother's antibodies are attacking the baby's red blood cells (immune red blood cell destruction

(hemolysis), like in ABO or Rh incompatibility), IVIG floods the baby's system with neutral antibodies that block the mother's attack antibodies from sticking to and destroying more red cells. This slows the breakage of red cells, so less bilirubin is released and the yellowing of the skin and eyes (jaundice) does not climb as fast.

1. Mother's antibodies (anti-A, anti-B, or anti-D) coat the baby's red cells, marking them for destruction.
2. IVIG antibodies bind to Fc receptors on macrophages (the immune cells that eat tagged red cells).
3. The macrophages become less able to recognize and destroy the baby's red cells.
4. Red blood cell destruction (Hemolysis) (red-cell breakage) slows, so less heme is released and less bilirubin is made.
5. Bilirubin rise slows, buying time for phototherapy to catch up and avoiding the need for exchange transfusion.

SAFETY WATCH

Infusion reactions: fever, chills, or flushing during the IV; nurses slow the drip if this happens. · Volume overload: IVIG is a fluid load, so the care team watches for signs the baby's heart or lungs are struggling (fast breathing, poor feeding). · Red blood cell destruction (Hemolysis) (rare paradox): in very rare cases IVIG itself can trigger red-cell breakdown if there is a mismatch; labs are rechecked after the dose. · Infection risk (theoretical, extremely low): pooled human product, screened heavily but not zero risk. · Only given once or twice: this is a short-term rescue, not a daily medicine.

Exchange transfusion · Procedural intervention (blood replacement)

A doctor takes out small portions of the baby's blood (which is full of bilirubin and sometimes the mother's attacking antibodies) through an umbilical vein catheter and replaces it with fresh donor blood. This physically removes bilirubin, washes out the antibodies causing red blood cell destruction (hemolysis), and gives the baby healthy red cells that will not be attacked. It is the fastest way to drop bilirubin when levels are so high that kernicterus (brain damage) is imminent, or when phototherapy and IVIG cannot keep up.

1. A catheter is placed in the umbilical vein (the soft spot in the belly button).
2. Small aliquots (5–20 mL at a time) of the baby's blood are withdrawn.
3. An equal volume of donor blood (type O negative or crossmatch-compatible, irradiated to prevent graft-versus-host disease) is infused.
4. The cycle repeats until about twice the baby's blood volume has been exchanged.
5. Bilirubin level drops by roughly 50% immediately; maternal antibodies are diluted; healthy red cells restore oxygen-carrying capacity.

SAFETY WATCH

Infection: any line into the blood can carry bacteria; strict sterile technique is used. · Electrolyte shifts: calcium, potassium, and glucose can swing; labs are checked during and after. · Blood clots or air bubbles: rare but serious; the team watches the line closely. · Heart rhythm changes: especially if calcium or potassium shifts; continuous heart monitor is on. · Bleeding: from the catheter site or inside the belly; the team checks for swelling and blood counts. · Graft-versus-host disease: prevented by irradiating (zapping) the donor blood before transfusion. · Only done in the NICU by experienced teams; this is reserved for life-threatening yellowing of the skin and eyes (jaundice).

Red flags

- Yellowing of the skin and eyes (Jaundice) appears in the first 24 hours of life — always needs urgent lab work and close watch because it usually means red cells are breaking down too fast.
- Yellow color spreads to the arms, legs, palms, or soles — sign that bilirubin is climbing to dangerous levels and the baby needs treatment right away.
- Baby is hard to wake, refuses to eat, has a shrill cry, or arches the head and neck backward — these mean bilirubin may be hurting the brain (kernicterus) and the baby needs emergency care.
- Pale, clay-colored stool and dark urine that continue past the first week — may mean bile cannot leave the liver (biliary atresia or other liver problem) and needs urgent evaluation.
- Yellowing of the skin and eyes (Jaundice) lasts longer than two weeks in a full-term baby or three weeks in a premature baby — requires blood tests to check for thyroid problems, liver disease, or infection.

Nursing priorities

1. Assess the baby for acute bilirubin encephalopathy signs every 2 to 4 hours: lethargy, high-pitched cry, arching of the neck and back (opisthotonus), poor feeding, and decreased tone. — Unconjugated bilirubin (fat-soluble form) crosses the blood-brain barrier and deposits in the basal ganglia (brain movement centers), causing irreversible kernicterus; early recognition allows emergency exchange transfusion to prevent permanent damage.
2. Position the baby naked except for diapers and eye shields under phototherapy lights, and turn the baby every 2 to 3 hours to expose maximum skin surface area. — Phototherapy converts unconjugated bilirubin in the skin into water-soluble isomers (changed forms) that the kidneys and liver can eliminate without conjugation (chemical processing); more exposed skin increases the breakdown rate.

3. Monitor the baby's temperature every 3 to 4 hours and watch for signs of overheating or excessive fluid loss during phototherapy. — Phototherapy lights generate radiant heat that can cause hyperthermia (overheating) and increase insensible water loss (evaporation from skin), leading to dehydration that worsens yellowing of the skin and eyes (jaundice) by concentrating bilirubin in the blood.
4. Support and optimize breastfeeding by ensuring 8 to 12 feeds per 24 hours, or supplement with expressed milk or formula if intake is inadequate. — Frequent feeding increases gut motility (bowel movement) and stool passage, which prevents enterohepatic circulation (bilirubin reabsorption from intestine back into blood); colostrum (early breast milk) also acts as a natural laxative.
5. Check transcutaneous bilirubin (TcB) or serum bilirubin levels at the times ordered by the provider, and plot results on the Bhutani nomogram (hour-specific bilirubin chart) to guide treatment decisions. — The hour-specific bilirubin level and the baby's risk factors (gestational age, red blood cell destruction (hemolysis), age in hours) determine the threshold for starting or stopping phototherapy and whether exchange transfusion is needed to prevent kernicterus.
6. Inspect every stool for color and document whether it is yellow, green, or acholic (pale white or clay-colored). — Acholic stools indicate direct (conjugated) hyperbilirubinemia from cholestasis or biliary atresia; if biliary atresia is not surgically corrected with Kasai portoenterostomy before 60 days of life, the baby will progress to cirrhosis (liver scarring) and need transplant.
7. Monitor for rebound hyperbilirubinemia by checking bilirubin 12 to 24 hours after stopping phototherapy, especially in babies with ABO incompatibility, Rh disease, or G6PD deficiency. — Ongoing red blood cell destruction (hemolysis) continues producing bilirubin even after phototherapy stops; rebound above treatment thresholds requires restarting phototherapy to prevent neurotoxicity (brain damage from bilirubin).
8. Educate parents to recognize danger signs at home: increased yellowing, poor feeding, extreme sleepiness, fever, high-pitched cry, or arching, and ensure follow-up within 24 to 48 hours of discharge. — Most kernicterus cases occur in babies who were discharged early without adequate follow-up; parents are the first-line monitors at home, and early recognition with prompt return for care prevents progression to irreversible brain injury.

Sources

- AAP Clinical Practice Guideline: Management of Hyperbilirubinemia in the Newborn Infant 35 or More Weeks of Gestation (2022)
- American Academy of Pediatrics (AAP)
- CDC: Jaundice and Kernicterus
- Cochrane Database: Interventions for neonatal jaundice

Last reviewed: 2026-07-07