

Newborn jaundice

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.

THE ONE-LINE TRUTH

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 chain — how it unfolds

1. Red blood cells break down faster than the newborn liver can clear bilirubin. — why: 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. — why: 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. — why: 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. — why: 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). — why: 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). — why: 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). — why: 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. — why: 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).

Spot it — what you see, 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.

Red flags — act now

- 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.

Treat it — in this order

1. 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.

2. 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.
3. 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.
4. 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.
5. 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.

Your top priorities at the bedside

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. — why: 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. — why: 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. — why: 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.