

# Hepatic Encephalopathy

A plain-English guide for you and your family

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## WHAT IS HAPPENING

Normally, the liver works like a filter. Gut bacteria break down protein from food and make ammonia. That ammonia travels in the blood to the liver, where healthy liver cells change it into urea (a safe waste product that leaves in the pee). This keeps ammonia levels low and the brain safe. When the liver is badly damaged—like from cirrhosis (scarring of the liver)—or when blood finds a shortcut around the liver through abnormal vessels (called shunts), ammonia and other toxins skip the filter and pour straight into the rest of the body. Something then tips the balance: bleeding in the gut, an infection, dehydration, constipation (which leaves more protein in the gut for bacteria to chew on), or sedative medicines that slow the brain. Ammonia climbs even higher. The brain gets confused first—small memory slips, trouble focusing, changes in personality. Sleep flips upside down (awake at night, sleepy all day). Then comes a flapping hand tremor (asterixis), slurred speech, and deeper confusion. If ammonia keeps rising, the person can slip into a coma. In sudden (acute) liver failure, ammonia can cause the brain to swell dangerously inside the skull. Every one of these signs is the brain reacting to poison in the blood—not separate problems, but one chain you can interrupt by lowering ammonia, treating the trigger, and supporting the liver.

## What you might feel

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- Confusion and trouble thinking clearly
- Sleeping during the day but awake at night (flipped sleep schedule)
- Hand tremor or flapping (asterixis)
- Slurred speech or mumbling
- Personality changes (irritability, apathy, or acting out of character)
- Slow responses or seeming 'out of it'
- Unsteady walk or trouble balancing
- Deep sleepiness or coma (in severe cases)

## Call the doctor right away if...

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- Sudden deep confusion or inability to wake the person (coma) — the brain is being poisoned fast and may swell or stop working.
- New seizures or uncontrolled shaking — extremely high ammonia or swelling in the brain can trigger abnormal electrical storms.
- Vomiting blood or black tarry stools in a person with liver disease — bleeding inside the gut dumps more protein (which becomes ammonia) into the system and can trigger or worsen brain poisoning.
- Fever and worsening confusion together — may signal an infection (like spontaneous bacterial peritonitis) that is pushing the sick liver over the edge.
- Very slow breathing or gasping — the brainstem centers that control breathing may be shutting down from severe toxin buildup.

## What the care team will do

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- Check airway and breathing every 15 to 30 minutes in confused or sleepy patients; place patient on their side; have suction ready; check oxygen saturation
- Give lactulose every hour until the first bowel movement, then adjust to get two to three soft stools per day; give by mouth, feeding tube, or enema if patient cannot swallow
- Look for and report bleeding signs including checking all stools for black color or blood, measuring blood pressure and heart rate every 2 to 4 hours, and watching for pale skin or low hemoglobin
- Monitor potassium and sodium levels daily and replace as ordered; give IV fluids carefully to avoid overload in patients with fluid buildup in the belly (ascites); measure strict intake and output
- Watch for infection signs including temperature every 4 hours, check belly for new pain or tenderness, send urine and blood cultures if fever occurs, and report white blood cell count changes
- Hold all sedating medicines including opioids, benzodiazepines, and sleeping pills; check medication list with pharmacy; use non-drug methods for pain and anxiety like repositioning and family presence
- Assess mental status using West Haven grades every 4 to 8 hours; test for asterixis by having patient hold arms out with wrists bent back; document sleep-wake pattern; use confusion assessment tools
- Give rifaximin 550 mg by mouth twice daily as ordered for grade 2 or higher encephalopathy or frequent repeat episodes; continue even after mental status improves

## Medicines you may be given — and why

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### Lactulose

Lactulose traps ammonia (the toxic waste that builds up when the liver fails) inside the colon and pulls it out of the body in stool. It does this two ways: first, gut bacteria break lactulose down into acids that make the colon more acidic, and ammonia cannot cross back into the blood in that acid environment—it stays trapped. Second, lactulose draws water into the colon (acts like a gentle laxative), so stool moves faster and carries the ammonia out before the body can reabsorb it. The goal is three soft bowel movements every day to keep ammonia low.

### Rifaximin

Rifaximin kills or slows down the gut bacteria that make ammonia from protein in food. Normally, when you eat protein, bacteria in the colon break it down and release ammonia as waste. In liver failure, the liver cannot clean that ammonia out of the blood, so it builds up and poisons the brain. Rifaximin reduces the number of ammonia-producing bacteria, so less ammonia is made in the first place. Because rifaximin is not absorbed into the blood, it works only in the gut and causes very few side effects in the rest of the body.

### Zinc sulfate (supplemental zinc)

Zinc is a mineral the liver needs to run enzymes that help clear ammonia. One key enzyme, ornithine transcarbamylase, works in the urea cycle (the liver's ammonia-disposal system). Many patients with cirrhosis are low in zinc because the sick liver does not store or use it well. Giving extra zinc can help the remaining liver cells work a bit better at turning ammonia into urea (a safer waste product that leaves in urine). Zinc will NOT cure hepatic encephalopathy or replace lactulose, but it may help as an add-on in patients who are zinc-deficient.

### L-ornithine L-aspartate (LOLA)

LOLA provides two amino acids (ornithine and aspartate) that help the body detoxify ammonia through two backup pathways outside the damaged liver. Ornithine feeds into the urea cycle in any remaining healthy liver cells, helping turn ammonia into urea. Aspartate helps muscle cells convert ammonia into glutamine (a safe storage form of nitrogen) so ammonia is pulled out of the blood. Muscles act like a sponge for ammonia when the liver cannot do its job. LOLA is used in some countries (especially in Europe) but is NOT FDA-approved in the United States; guidelines vary on its use.

### Treat the trigger (antibiotics, blood products, electrolyte repletion, stop sedatives)

Hepatic encephalopathy almost always has a trigger that tips a barely-compensated liver over the edge. The most common triggers are infection (which releases inflammatory signals that

worsen brain swelling and increase ammonia production), gastrointestinal bleeding (blood in the gut is digested into protein, which gut bacteria turn into ammonia), dehydration or electrolyte imbalances (low potassium or sodium worsen confusion and can stress the kidneys), constipation (ammonia sits in the colon longer and gets reabsorbed), and sedating medications like benzodiazepines or opioids (these directly suppress brain function and unmask encephalopathy). Fixing the trigger is AS important as lowering ammonia—if you only give lactulose but leave an infection untreated, the encephalopathy will not improve. Antibiotics kill the infection, stopping the inflammatory cascade. Blood transfusion or endoscopy stops GI bleeding, cutting off the protein load to the gut. IV fluids and electrolyte replacement restore kidney function and help the body clear toxins. Stopping sedatives lets the brain wake up.

## Where this information comes from

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- AASLD/EASL Practice Guideline on Hepatic Encephalopathy 2014
- American Journal of Gastroenterology
- Hepatology journal (official journal of AASLD)
- UpToDate clinical decision support

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