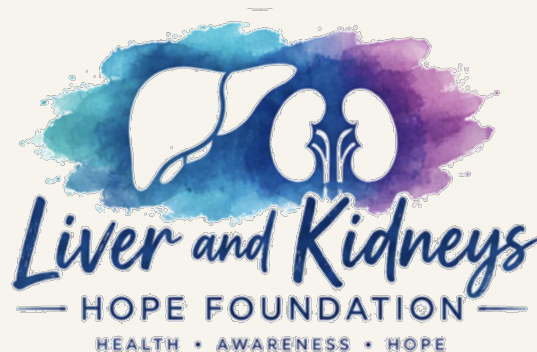




PATIENT & COMMUNITY EDUCATION SERIES

Understanding Hepatic Encephalopathy

A plain-language, comprehensive guide to hepatic encephalopathy — confusion caused by liver dysfunction, how it's graded and treated, and how to navigate daily life safely.

Written for patients, families, and caregivers managing this common complication of liver disease

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A NOTE BEFORE YOU BEGIN

Hepatic encephalopathy is genuinely treatable, and episodes are often reversible with prompt treatment. This guide focuses on HE specifically, as it occurs in cirrhosis and liver failure — our companion cirrhosis and liver failure guides cover the broader conditions it arises from.

SECTION 1 Your Liver: A Quiet, Hardworking Organ

Before discussing what can go wrong, it helps to appreciate just how much your liver does every day — usually without you noticing at all.

Your liver is the largest solid organ inside your body, weighing roughly 1.4 kg (about 3 pounds) in an adult. Scientists have identified more than **500 distinct functions** it performs — including one that's directly relevant to this entire guide: filtering waste products, including ammonia, produced by normal digestion.

The liver as the body's detox filter

As food is digested, bacteria in the gut naturally produce ammonia as a byproduct. In a healthy liver, this ammonia is efficiently converted into a harmless substance called urea, which is then removed by the kidneys. This filtering process happens continuously, day and night, without you ever noticing it.

What happens when this filter is impaired

Hepatic encephalopathy develops when liver disease — most often cirrhosis — impairs this filtering process, allowing ammonia and other substances to build up in the bloodstream and affect brain function. Understanding this single mechanism is the key that unlocks nearly everything else in this guide, including why the treatments described later work the way they do.

DID YOU KNOW?

Blood from the intestines normally passes through the liver before reaching the rest of the body — a kind of built-in checkpoint. In cirrhosis, scarring can both impair the liver's own filtering capacity and create alternate pathways that let blood bypass the liver altogether, compounding the problem in two ways at once.

KEY POINT

Hepatic encephalopathy is not a separate disease from cirrhosis or liver failure — it's a direct consequence of the liver's reduced filtering capacity, which is exactly why treating it focuses on reducing ammonia production and improving its clearance, covered in depth later in this guide.

SECTION 2 What Is Hepatic Encephalopathy?

Hepatic encephalopathy (HE) is a decline in brain function caused by the liver's reduced ability to clear toxins from the blood — most often occurring in people with cirrhosis or liver failure.

"Hepatic" refers to the liver, and "encephalopathy" describes any condition affecting brain function. Put together, hepatic encephalopathy describes brain dysfunction that results specifically from liver disease, rather than from a primary problem within the brain itself.

A spectrum, not a single fixed state

HE ranges from very subtle, barely noticeable changes in thinking, all the way to deep unresponsiveness (coma). This range is captured by a formal grading system, covered in Section 4, which is central to how HE is discussed, tracked, and treated.

A genuinely reversible condition

Unlike some complications of advanced liver disease, hepatic encephalopathy is often substantially or completely reversible with appropriate treatment, particularly when a specific trigger (Section 6) is identified and addressed. This is one of the most important, hopeful facts to hold onto throughout this guide.

KEY POINT

Because HE is generally reversible, episodes are approached as treatable events, not as signs of permanent decline — even when an episode looks frightening in the moment, meaningful recovery is genuinely the expected outcome with proper treatment.

DID YOU KNOW?

HE is sometimes called "portosystemic encephalopathy" in medical literature, reflecting the role that blood bypassing the liver (through the portal-to-systemic circulation) plays in the condition — you may see this term in older records or research articles.

SECTION 3

Why It Happens: Ammonia, Toxins & the Brain

Understanding the biology behind hepatic encephalopathy explains both why it happens and why the mainstay treatments work the way they do.

The central role of ammonia

Gut bacteria break down protein from digested food, producing ammonia as a natural byproduct. A healthy liver converts this ammonia into urea, which is harmlessly excreted by the kidneys. When liver function is impaired, or when blood bypasses the liver due to portal hypertension (covered in our companion cirrhosis guide), ammonia isn't adequately cleared and builds up in the bloodstream.

How ammonia affects the brain

Elevated ammonia crosses into the brain, where it's thought to disrupt normal brain cell function and communication between brain cells, in ways researchers continue to study. This disruption is what produces the spectrum of symptoms — from subtle confusion to deep unresponsiveness — described in Section 4.

Ammonia isn't the whole story

While ammonia is the best-understood and most clinically useful factor, researchers believe other substances and processes — including inflammation throughout the body, and imbalances in gut bacteria — also contribute to hepatic encephalopathy, which is part of why blood ammonia levels don't always perfectly match how confused someone appears (Section 9).

KEY POINT

Because ammonia arising from gut bacteria and digestion is central to this process, treatments that target the gut — reducing ammonia production and absorption there — form the cornerstone of HE treatment, covered fully in Sections 13–14.

DID YOU KNOW?

This gut-liver-brain connection is sometimes called the "gut-liver-brain axis" in medical literature, and it's an active area of ongoing research, including into the role of the gut microbiome in HE.

SECTION 4

Grading Severity: From Subtle to Coma

Because HE spans such a wide range of severity, clinicians use a standardized grading system — one of the most useful pieces of vocabulary you'll encounter in this whole guide.

The West Haven criteria

Grade	General presentation
Minimal (Grade 0)	No obvious clinical symptoms, but subtle deficits detectable on specific testing — covered in Section 5.
Grade 1	Mild confusion, shortened attention span, subtle changes in mood or sleep pattern; often described by family as the person "just not seeming like themselves."
Grade 2	More noticeable confusion, lethargy, or personality change; may have a flapping hand tremor (asterixis); disorientation to time is common.
Grade 3	Marked confusion or significant drowsiness, but still responsive to voice or physical stimulation.
Grade 4	Unresponsive (coma) — the most severe grade, requiring emergency, intensive care.

Why this grading matters day to day

This scale gives patients, families, and clinicians a shared, specific language for describing severity and tracking change over time — "he's at about a Grade 1 today, better than yesterday's Grade 2" conveys far more useful information than "he seems a bit confused."

KEY POINT

Learning to roughly recognize these grades yourself, if you're a patient or caregiver, is one of the most practical skills you can develop — it helps you communicate clearly and urgently with the care team, and helps you track whether treatment is working.

DID YOU KNOW?

Grade can change within hours, in either direction — improving quickly with treatment of a trigger, or worsening if a trigger goes unaddressed. This is part of why frequent reassessment, not a single snapshot, guides care.

SECTION 5

Minimal Hepatic Encephalopathy: The Hidden Form

Before any obvious symptoms appear, many people with cirrhosis have a subtler form of HE that can still meaningfully affect daily functioning — including, importantly, driving safety.

What makes it "minimal"

Minimal hepatic encephalopathy (MHE) causes no obvious confusion that family or the person themselves would necessarily notice in conversation. Instead, it shows up as subtle deficits in attention, reaction time, and certain types of coordination — detectable only through specific psychometric or computerized testing designed to pick up on these changes.

Why it matters despite being "hidden"

Research has shown that MHE can meaningfully affect a person's ability to perform complex tasks safely, including driving. Because it produces no obvious outward signs, it can go completely unrecognized unless specifically tested for — which is exactly why awareness of it matters, even for patients who feel and seem completely normal.

How it's identified

Specific tests — including certain paper-and-pencil timed tasks and computerized testing tools designed for this purpose — can detect MHE, though it isn't universally screened for in every patient with cirrhosis. Your hepatologist can advise whether testing for MHE is relevant to your situation, particularly if there are any concerns about driving or complex daily tasks.

TALK WITH YOUR CLINICIAN

If you have cirrhosis and are wondering about your fitness to drive, or have noticed subtle changes in concentration or reaction time, it's worth raising directly — testing for minimal HE may be relevant, and this is exactly the kind of practical, safety-focused conversation your care team wants to have with you.

KEY POINT

Minimal HE is treatable using the same approaches covered later in this guide, and treatment has been shown to improve the specific deficits it causes — this isn't a hidden, untreatable problem, just an under-recognized one.

SECTION 6

Causes & Common Triggers

While the underlying cause of hepatic encephalopathy is liver disease itself, specific triggers very often set off an individual episode — and identifying that trigger is one of the most important parts of treating any given episode.

Common triggers

Trigger	Why it matters
Infection	One of the most common triggers; the body's inflammatory response to infection can worsen HE independent of the infection's direct effects.
Gastrointestinal bleeding	Blood in the digestive tract is broken down by gut bacteria into ammonia, providing a large, sudden ammonia load.
Constipation	Slower transit through the gut allows more time for ammonia-producing bacteria to act, and for ammonia to be absorbed.
Dehydration	Can occur from vomiting, diarrhea, or excessive diuretic use (for ascites), and can worsen HE.
Certain medications	Particularly sedatives, sleeping pills, and opioid pain medications, which can directly worsen confusion.
Electrolyte imbalances	Particularly low sodium or potassium, sometimes related to diuretic medications.
Not taking prescribed HE treatment	Missing doses of lactulose or rifaximin (Sections 13–14) is a common, addressable trigger.
New or worsening kidney function	Reduces the body's ability to clear toxins overall.

KEY POINT

Because triggers are so often identifiable and treatable, finding and addressing the trigger behind any given episode is just as important as treating the confusion itself — and often produces the fastest improvement.

DID YOU KNOW?

Sometimes no specific trigger is identified despite a thorough search — this doesn't mean one didn't exist, and treatment proceeds effectively regardless, focused on reducing ammonia through the approaches in Sections 13–14.

SECTION 7 How Common Is It? Who Is Affected

Hepatic encephalopathy is a common complication for people with cirrhosis, particularly as liver disease advances — making it one of the more frequently encountered issues covered in this entire guide series.

A substantial proportion of people with cirrhosis will experience at least one episode of overt (clinically noticeable) hepatic encephalopathy over the course of their disease, and minimal HE, as discussed in Section 5, is thought to affect an even larger share of people with cirrhosis, given how subtle it can be.

Who is at higher risk

- **People with more advanced cirrhosis**, particularly decompensated disease.
- **People who have had a prior episode of HE**, since having one episode increases the likelihood of future ones.
- **People with significant portal hypertension** or prior procedures (such as TIPS, covered in our companion cirrhosis guide) that reroute blood around the liver.
- **Older adults**, who may be somewhat more susceptible to episodes.

DID YOU KNOW?

Because HE recurrence risk is significant after a first episode, ongoing preventive treatment — covered in Sections 13–14 — is typically continued indefinitely after a first episode, not just during active symptoms.

KEY POINT

If you have cirrhosis, understanding HE — even before ever experiencing an episode — is genuinely worthwhile preparation, given how common it is across the course of this disease.

SECTION 8

Signs & Symptoms: What Families Notice First

Family members and caregivers are very often the first to notice the early signs of hepatic encephalopathy — sometimes before the patient themselves is aware anything has changed.

Early, subtle signs

- **Changes in sleep pattern**, such as sleeping during the day and being awake at night (sometimes an early, specific clue).
- **Shortened attention span** or difficulty concentrating.
- **Subtle personality or mood changes** — irritability, apathy, or seeming "not quite themselves."
- **Mild forgetfulness** or trouble with simple tasks that were previously easy.
- **Slightly slurred or slowed speech**.

More noticeable signs

- **Clear disorientation** — to time, place, or situation.
- **A flapping hand tremor (asterixis)**, best seen by asking the person to hold their arms out with hands bent back at the wrist.
- **Significant drowsiness** or difficulty staying awake.
- **Uncharacteristic behavior**, which can occasionally include agitation.

WHEN TO SEEK URGENT CARE

Significant confusion, difficulty waking someone, or any rapid worsening warrants prompt medical attention — treatment works best when started early, and HE can worsen quickly if a trigger goes unaddressed.

KEY POINT

Because patients themselves often can't recognize these changes in the moment, caregiver observations are genuinely valuable clinical information — trust your instincts if something seems different, and share it with the care team.

SECTION 9

How Hepatic Encephalopathy Is Diagnosed

Diagnosing HE combines clinical assessment, blood tests, and, importantly, ruling out other possible causes of confusion — since HE is generally a diagnosis reached by putting the whole picture together, not from any single test.

Clinical assessment

The most important diagnostic tool is a careful clinical examination — assessing orientation, attention, and checking for the characteristic flapping tremor (asterixis) — which is how the West Haven grade (Section 4) is actually determined at the bedside.

Blood ammonia level

An elevated blood ammonia level can support the diagnosis, but it's an imperfect test — some people with clear HE have normal ammonia levels, and some people with elevated ammonia have no symptoms at all. Because of this, ammonia level is used as a supporting piece of information, not a definitive, stand-alone test.

Other blood tests

Blood tests are also used to look for common triggers, including markers of infection, kidney function, and electrolyte levels (particularly sodium), since identifying a treatable trigger is central to the diagnostic process.

KEY POINT

HE is fundamentally a clinical diagnosis — meaning it's based primarily on how a person actually presents, supported by tests, rather than confirmed by any single lab value. This is worth knowing if a normal ammonia level ever seems to conflict with an otherwise clear clinical picture.

DID YOU KNOW?

Because ammonia level doesn't always correlate perfectly with symptom severity, some specialists don't routinely check it once the diagnosis is otherwise clear, focusing instead on clinical grade and trigger identification.

SECTION 10 Ruling Out Other Causes of Confusion

Because confusion can result from many causes beyond liver disease, and because someone with cirrhosis can develop any of these other causes too, ruling them out is a genuinely important part of diagnosis, not just a formality.

Other causes that can mimic or coexist with HE

- **Alcohol intoxication or withdrawal**, particularly relevant for alcohol-related liver disease.
- **Sedative or opioid medication effects.**
- **Stroke or other neurological events**, which require entirely different, urgent treatment.
- **Low blood sugar.**
- **Infections affecting the brain** (such as meningitis), which are less common but important not to miss.
- **Electrolyte imbalances** unrelated to HE itself.
- **Underlying dementia or other cognitive conditions**, particularly in older patients.

KEY POINT

This is exactly why new confusion in someone with cirrhosis is evaluated thoroughly in a medical setting, rather than automatically assumed to be HE — getting the diagnosis right shapes everything about treatment that follows.

DID YOU KNOW?

Sometimes more than one cause contributes at once — for example, an infection triggering both a fever and HE simultaneously. Thorough evaluation helps make sure every contributing factor gets addressed.

SECTION 11 Possible Complications & Safety Risks

Beyond the confusion itself, hepatic encephalopathy carries some specific safety risks worth understanding and actively planning around.

Falls and injury

Confusion, drowsiness, and impaired coordination during an HE episode significantly raise the risk of falls and related injuries — a genuinely important, practical safety consideration covered further in Section 17.

Aspiration risk

Significant drowsiness or confusion can impair safe swallowing, raising the risk of food or liquid entering the airway — part of why more severe episodes are managed in a hospital setting with close monitoring.

Progression to more severe grades

Without treatment of the underlying trigger, HE can progress from milder to more severe grades, potentially reaching unresponsiveness (coma) in the most serious cases — which is why prompt evaluation and treatment of any new or worsening episode matters.

Impact on daily functioning and independence

Recurrent HE, even when each individual episode resolves, can affect a person's confidence, independence, and ability to safely manage certain tasks — a real quality-of-life consideration that deserves open discussion with your care team.

SEEK EMERGENCY CARE FOR SEVERE SYMPTOMS

Significant unresponsiveness, difficulty breathing, or any sudden, severe worsening requires immediate emergency medical attention.

KEY POINT

Understanding these risks isn't meant to be frightening — it's meant to support practical planning, covered throughout the rest of this guide, that genuinely reduces them.

SECTION 12

Treatment & Management: The Big Picture

Treating hepatic encephalopathy rests on a few clear, well-established pillars — and the overall outlook with treatment is genuinely good.

Pillar 1 — Identify and treat the trigger

As covered in Section 6, addressing whatever set off the episode — infection, bleeding, constipation, dehydration, or another cause — is often the single most impactful step, and frequently produces improvement within hours to a couple of days.

Pillar 2 — Reduce ammonia production and absorption

This is achieved primarily through lactulose (Section 13) and, for recurrent episodes, rifaximin (Section 14) — the two mainstay medications in HE treatment.

Pillar 3 — Supportive care

This includes ensuring adequate hydration and nutrition, safety precautions during confusion (Section 17), and close monitoring, particularly for more severe episodes.

Pillar 4 — Ongoing prevention after an episode

Because recurrence risk is significant after a first episode, preventive treatment is generally continued indefinitely afterward, not stopped once symptoms resolve.

A WORD OF CAUTION

Specific treatment decisions depend on severity, likely trigger, and your overall clinical picture. **This guide cannot recommend specific treatments or doses.** These decisions belong with your treating clinician.

KEY POINT

HE treatment is one of the more reliably effective areas of liver disease management — most episodes respond well to this combination of approaches, and recurrence is genuinely preventable with consistent ongoing treatment.

SECTION 13

Lactulose: The Cornerstone Treatment

Lactulose is the single most important, most widely used medication in hepatic encephalopathy treatment, and understanding how it works helps explain why consistent use matters so much.

How lactulose works

Lactulose is a synthetic sugar that isn't absorbed by the body. Instead, it travels to the colon, where it is fermented by gut bacteria, producing an acidic environment that both reduces ammonia-producing bacteria and converts ammonia into a form that's trapped in the gut rather than absorbed into the bloodstream. It also acts as a laxative, helping physically flush ammonia-containing waste out of the body faster.

Dosing: aimed at a specific effect, not a fixed amount

Unlike many medications dosed by weight or a fixed schedule, lactulose is generally titrated to achieve a specific effect — typically **two to three soft, formed bowel movements per day** — with the dose adjusted up or down by your clinician based on how your body responds. Too little won't adequately clear ammonia; too much can cause excessive diarrhea, dehydration, and electrolyte problems that can actually worsen HE.

KEY POINT

Because lactulose dosing is about hitting a specific target — not a one-size-fits-all amount — tracking and reporting your actual bowel movement frequency to your care team is genuinely important information, not a minor detail.

DID YOU KNOW?

Lactulose has been used for HE treatment for decades and remains a cornerstone of care precisely because it directly and effectively targets the ammonia-production mechanism described in Section 3.

TALK WITH YOUR CLINICIAN

If you're having too few or too many bowel movements on your current dose, say so directly — this is exactly the kind of practical feedback that helps your care team adjust your dose appropriately.

Because lactulose can taste quite sweet and syrupy, some patients find it more tolerable mixed with a small amount of water or juice, or taken chilled — small adjustments like these can make a real difference to long-term adherence without changing the medication's effectiveness. If taste or texture is genuinely getting in the way of taking it consistently, mention this to your care team rather than simply skipping doses, since alternative formulations or timing strategies are sometimes available.

SECTION 14 Rifaximin & Other Medications

For people with recurrent episodes despite lactulose, or who don't tolerate it well, additional medications offer real, effective options.

Rifaximin

Rifaximin is an antibiotic that works specifically within the gut, poorly absorbed into the rest of the body, which reduces the population of ammonia-producing bacteria in the intestine. It's most commonly added alongside lactulose, particularly for people who've had recurrent episodes, and has been shown to meaningfully reduce the risk of future episodes when used this way.

Other approaches sometimes considered

- **L-ornithine L-aspartate (LOLA)**, a supplement that supports the body's own ammonia processing pathways, used in some settings and regions.
- **Zinc supplementation**, since zinc deficiency is common in cirrhosis and may play a supporting role in ammonia metabolism.
- **Branched-chain amino acids**, considered in some specific situations, particularly around nutrition (Section 16).

KEY POINT

Rifaximin and lactulose are typically used together, not as alternatives to each other, for anyone with recurrent episodes — this combination approach reflects strong evidence for reducing future episodes more effectively than either medication alone.

A WORD OF CAUTION

Whether additional medications are appropriate for you depends on your specific situation and how you've responded to lactulose. **This guide cannot recommend specific medications or doses** — these decisions belong with your treating clinician.

SECTION 15 Treating & Preventing Triggers

Because triggers so often drive individual episodes, actively managing them — both during an episode and as ongoing prevention — is just as important as the medications covered in the previous two sections.

During an active episode

Alongside starting or increasing lactulose, the care team will typically look for and treat any identifiable trigger — treating an infection with antibiotics, addressing gastrointestinal bleeding, correcting electrolyte imbalances, and reviewing medications for anything that might be contributing (particularly sedatives or opioids).

Preventing future episodes

- **Consistent adherence to lactulose and rifaximin**, if prescribed — missed doses are themselves a common trigger.
- **Preventing constipation** through consistent lactulose dosing.
- **Being cautious with sedatives, sleep aids, and opioid pain medications**, discussing any new prescription with your hepatologist first.
- **Prompt treatment of infections**, including staying current on recommended vaccinations.
- **Staying well-hydrated**, while following any specific fluid guidance related to ascites management.
- **Regular follow-up** to monitor for and address electrolyte imbalances, particularly if on diuretics for ascites.

KEY POINT

Many HE episodes are genuinely preventable through consistent attention to these factors — this is an area where patient and caregiver engagement in daily management makes a real, measurable difference.

TALK WITH YOUR CLINICIAN

Before starting any new medication, including over-the-counter sleep aids or pain relievers, check with your hepatologist — some common options can meaningfully raise HE risk.

SECTION 16 Diet & Nutrition: Getting Protein Right

Nutrition and HE have a complicated history — outdated advice once recommended restricting protein, but current understanding is quite different, and worth knowing clearly.

Why protein restriction fell out of favor

Because protein digestion is a source of ammonia, doctors once commonly recommended restricting protein intake for people with HE. Research over the past couple of decades has shown this approach actually worsens outcomes for most patients, by contributing to

malnutrition and muscle loss — problems that are already common in cirrhosis and that themselves worsen HE risk.

Current recommendations

- **Adequate protein intake is now generally recommended**, not restricted, for the large majority of patients — specific targets are best set by a dietitian based on your body weight and overall condition.
- **Smaller, more frequent meals**, including a bedtime snack, help offset cirrhosis's effect on overnight energy storage (covered in our companion cirrhosis guide) and may support steadier ammonia handling.
- **Plant-based or dairy protein sources** are sometimes preferred by some specialists for certain patients, as they may be somewhat better tolerated, though this is individualized rather than a universal rule.

KEY POINT

If you've heard that protein should be restricted with HE, this reflects outdated guidance. Current best practice favors adequate protein intake, tailored by a dietitian, alongside the medications covered earlier in this guide.

TALK WITH A PROFESSIONAL

A registered dietitian experienced in liver disease can set specific, personalized protein and calorie targets for you — this is one of the more valuable referrals available in HE and cirrhosis care generally.

SECTION 17

Safety at Home: Driving, Falls & Daily Life

Because HE, including its minimal form, can affect judgment, reaction time, and coordination, a few practical safety conversations are worth having proactively, not just after a problem occurs.

Driving

Even minimal HE, which produces no obvious outward symptoms, has been linked to impaired driving performance in research studies. This is a genuinely important, sensitive topic worth raising directly and honestly with your hepatologist — they can help assess whether testing for minimal HE (Section 5) is relevant to you, and can offer guidance specific to your situation and local regulations.

Fall prevention

- **Clear walkways** of clutter, loose rugs, and cords, particularly at night.
- **Adequate lighting**, including night lights for bathroom trips.
- **Grab bars in bathrooms**, a simple, effective modification.
- **Supervision or check-ins** during any period of known active confusion.

Medication safety

Someone experiencing active confusion may not reliably manage their own medications correctly — a caregiver or a pill organizer system can help reduce the risk of missed or doubled doses during this time.

Financial and important decisions

During an active episode, judgment and decision-making capacity can be genuinely impaired. It's reasonable to postpone significant financial or legal decisions until an episode has clearly resolved, and to loop in a trusted family member if something urgent can't wait.

KEY POINT

These aren't meant as alarming precautions — they're practical, common-sense steps that meaningfully reduce real risks, and having them in place before they're needed is far easier than scrambling during an actual episode.

It's worth acknowledging that conversations about driving in particular can feel difficult, since driving often represents independence in a very concrete, personal way. Bringing this up with your care team doesn't mean an automatic restriction — it means getting an honest, individualized assessment based on your specific situation, which may well conclude that driving remains safe for you, particularly if your HE is well controlled. The goal of raising it is accurate information, not a foregone conclusion.

SECTION 18

Monitoring, Recurrence & Your Care Team

HE is monitored as an ongoing part of cirrhosis care, not treated as a one-time problem to resolve and forget.

What ongoing monitoring involves

- **Regular clinical check-ins**, including simple questions about sleep, concentration, and daily functioning, not just formal testing.
- **Periodic reassessment of medication doses**, particularly lactulose, based on bowel movement frequency.

- **Ongoing attention to potential triggers**, including medication reviews and electrolyte monitoring.
- **For some patients**, periodic testing for minimal HE, particularly if driving or complex tasks are a concern.

Who might be on your team

Hepatologist / gastroenterologist

Primary care provider

Registered dietitian

Nurse educator

Pharmacist

Family caregivers

KEY POINT — CAREGIVERS ARE PART OF THE TEAM

Because family members often notice changes before the patient does, keeping a simple log of sleep patterns, bowel movements, and any subtle behavior changes can be genuinely valuable information to bring to appointments.

MAKE THE MOST OF APPOINTMENTS

Worth asking regularly: "Are we still hitting the target number of bowel movements with my current lactulose dose?" and "Has anything changed that we should discuss?"

SECTION 19 Special Situations

Hepatic encephalopathy management looks somewhat different across certain groups and circumstances.

Older adults

Distinguishing HE from age-related cognitive changes or dementia can be more complex in older patients, making thorough evaluation particularly important, and sometimes requiring closer coordination with a geriatrician or neurologist.

After TIPS placement

A TIPS procedure (covered in our companion cirrhosis guide), which reduces portal pressure by rerouting blood flow, can increase HE risk, since it creates an additional pathway for blood — and the ammonia it carries — to bypass the liver. This risk is discussed as part of TIPS candidacy evaluation, and HE occurring after TIPS is managed with the same principles covered throughout this guide.

Alcohol-related liver disease

Distinguishing HE from alcohol intoxication or withdrawal can be genuinely challenging in this population, making an honest, detailed history of recent alcohol use an important part of accurate diagnosis and treatment.

Recurrent, difficult-to-control HE

For a minority of patients who continue to have frequent episodes despite lactulose and rifaximin, further specialist evaluation may explore additional contributing factors or consider other interventions, including, in appropriate candidates, transplant evaluation.

TALK WITH YOUR CLINICIAN

If any of these situations apply to you or a loved one, mention it directly — it may change how episodes are evaluated or managed.

SECTION 20 Supporting a Loved One Through HE

Caring for someone through episodes of confusion is emotionally demanding in a particular way — it can feel like watching a loved one temporarily become someone else, which is disorienting and often frightening.

What caregivers commonly experience

Fear, exhaustion, grief, and sometimes a strange sense of loneliness — even when surrounded by medical staff — are all completely normal responses. Watching a loved one experience confusion, personality changes, or disorientation can be genuinely hard to witness, even knowing intellectually that it's often reversible.

Practical approaches during an episode

- **Stay calm and speak simply and gently** — reorienting the person to time and place (reminding them where they are, what day it is) can help without being confrontational.
- **Don't argue or try to reason extensively** with confused thinking — gentle redirection tends to work better than correction.
- **Keep the environment calm and free of unnecessary stimulation.**
- **Track what you observe** — timing, specific behaviors, and any potential triggers — this information genuinely helps the care team.
- **Know your own limits** and don't hesitate to seek help, including emergency care, if a situation feels beyond what you can safely manage at home.



IF YOU ARE STRUGGLING

If you are experiencing persistent low mood, hopelessness, or thoughts of harming yourself — whether as a patient or a caregiver — please reach out to a healthcare professional or a crisis line. The **988 Suicide & Crisis Lifeline** (call or text 988 in the United States) is free, confidential, and available 24/7. You do not have to manage this alone.

COMMUNITY MATTERS

Connecting with other caregivers navigating HE — through support groups or organizations like The Liver and Kidneys Hope Foundation · can genuinely ease the particular isolation this role can bring.

SECTION 21 Prevention & Myths vs. Facts

While HE can't always be entirely prevented, several genuinely protective steps substantially reduce both the likelihood and severity of episodes.

Prevention

- **Take lactulose and rifaximin consistently**, if prescribed, even when feeling well.
- **Attend regular follow-up appointments** to monitor for and adjust treatment as needed.
- **Be cautious with sedatives, sleep aids, and opioid medications** — always check with your hepatologist first.
- **Stay current on vaccinations** and seek prompt treatment for any infection.
- **Maintain adequate nutrition**, including sufficient protein, rather than restricting it.
- **Manage constipation proactively** through your prescribed lactulose regimen.

Myths vs. facts

Myth	Fact
"Confusion from liver disease is permanent brain damage."	HE is often substantially or completely reversible with proper treatment, especially when a trigger is identified and addressed.
"I should restrict protein to prevent HE."	Current guidance favors adequate protein intake for most patients — restriction can worsen malnutrition and actually increase HE risk.
"If I feel completely fine, I don't need to worry about HE."	Minimal HE causes no obvious symptoms but can still affect tasks like driving — it's worth discussing with your clinician even without noticeable symptoms.
"Lactulose is just a laxative for constipation."	While it does cause bowel movements, its primary purpose in HE is reducing ammonia absorption — the laxative effect is part of how it works, not a side effect.
"Once you've had one HE episode, there's nothing to be done to prevent more."	Consistent use of lactulose, often combined with rifaximin, substantially reduces the risk of future episodes.
"Ammonia level always tells you how confused someone is."	Ammonia level is a useful supporting clue, but it doesn't always match symptom severity — clinical assessment matters more.
"A person with HE should be left alone to sleep it off."	New or worsening confusion warrants medical evaluation, since identifying and treating the underlying trigger is what actually resolves an episode.

KEY POINT

If you remember one thing from this page: hepatic encephalopathy is common, often reversible, and — with consistent treatment and attention to triggers — genuinely manageable and preventable.

CLOSING

Questions, Glossary, Resources & Disclaimer

Questions you might ask your doctor

- What grade of HE was I (or my loved one) at during this episode, and how has that changed?
- Was a specific trigger identified, and how is it being treated?

- Am I on the right lactulose dose — are we hitting the target number of bowel movements?
- Would rifaximin be appropriate for me, given my history of episodes?
- Should I be tested for minimal HE, particularly regarding driving safety?
- What specific signs should prompt us to call, or seek emergency care, before our next visit?

Glossary

Term	Plain-language meaning
Ammonia	A waste product from digestion that a healthy liver clears; builds up and affects the brain in HE.
Asterixis	A flapping hand tremor, a characteristic sign of hepatic encephalopathy.
Lactulose	A laxative medication that reduces ammonia absorption; the cornerstone HE treatment.
Minimal hepatic encephalopathy (MHE)	A subtle form of HE with no obvious symptoms but detectable effects on attention and reaction time.
Rifaximin	A gut-targeted antibiotic that reduces ammonia-producing bacteria; often used alongside lactulose.
West Haven criteria	The standard grading scale for HE severity, from Grade 0 (minimal) to Grade 4 (coma).

About The Liver and Kidneys Hope Foundation

The Liver and Kidneys Hope Foundation (TLHF) is a Nevada-based 501(c)(3) nonprofit dedicated to liver-health education, community support, and outreach. Through educational materials like this guide, community programming, our chapter network, volunteer efforts, and fundraising initiatives, we work to raise awareness of liver conditions and support the people affected by them, including those managing hepatic encephalopathy and the families who support them. The Foundation is not a medical provider and does not deliver clinical care.

A NOTE ON SOURCES AND CURRENCY

This guide reflects widely accepted, general medical understanding of hepatic encephalopathy intended for patient education. It does not cite individual studies, and research into its mechanisms and treatment continues to evolve. For the most current, individualized information, your healthcare team is the best resource.



Full disclaimer

PLEASE READ

This publication is provided by The Liver and Kidneys Hope Foundation for **educational purposes only**. It is **not medical advice** and is not a substitute for professional diagnosis, treatment, or the judgment of a qualified, licensed healthcare provider who knows your personal history. The Liver and Kidneys Hope Foundation is **not a medical provider** and does not practice medicine, provide clinical services, or establish a provider-patient relationship through this material.

Do not use this guide to diagnose or treat a health problem or disease, and do not disregard or delay seeking professional medical advice because of something you have read here. Significant confusion, difficulty waking someone, or any rapid worsening of symptoms can indicate a medical emergency — contact your local emergency services immediately.

The Foundation makes no warranties about the completeness, accuracy, or currency of this information and accepts no liability for actions taken based on it. Always consult your own healthcare professional.

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