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Alcohol and the Brain

The drinking patient as a neurological emergency

Thiamine · Wernicke · Withdrawal · Seizures · Subdural · Sodium · Liver · Toxic alcohols

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About this guide

The confused patient who smells of alcohol, approached as a neurological emergency: the TIMED-S differential, Wernicke's beyond the triad (and after bariatric surgery), thiamine dosing and the glucose myth, withdrawal and seizures by ASAM 2020, subdurals, sodium and osmotic demyelination, hepatic encephalopathy, and the wider neurology of alcohol.

What changed. First edition, expanded from the author's emergency department reference.

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How it was made

Expanded from the author's emergency department reference. The ideas and direction are the author's. AI tools were used for literature search, drafting support and language editing. The author is responsible for the content. See thetract.org/about/policies/#ai.

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A teaching reference, not a protocol. Doses and thresholds are given to aid recognition; check them against current local guidance and prescribing information before acting on them.

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Part I

A neurological patient

The principle · The TIMED-S framework · The first ten minutes

The confused patient who smells of alcohol is one of the most dangerous people in the emergency department, because everyone thinks they already know the diagnosis. Alcohol damages the brain directly, starves it of thiamine, lowers the seizure threshold, makes falls and bleeding more likely, and disturbs sodium, glucose and the liver. Several of these usually happen together. This guide treats the drinking patient as what they are: a neurological patient, until proved otherwise. It expands the author's emergency department reference and checks it against the EFNS guideline on Wernicke's encephalopathy, the 2020 ASAM guideline on alcohol withdrawal, the European hyponatraemia guideline, the EASL and AASLD guidance on hepatic encephalopathy, and the trials behind them.

The principle

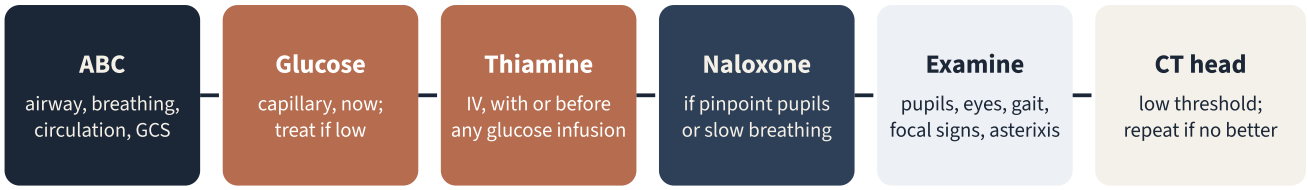
"Intoxication" is a diagnosis of exclusion. In any patient with a history of alcohol use and an altered mental state, assume a structural, metabolic, toxic, infectious and withdrawal cause until each has been excluded. They are not alternatives: in the same patient you may find a subdural haematoma, hypoglycaemia, thiamine deficiency and early withdrawal at once.

1.1 The TIMED-S framework

A differential that is easy to carry in your head, built around what can kill or disable in the next few hours:

Group	Diagnoses	Priority
Toxic and metabolic	Ethanol intoxication · hypoglycaemia · alcoholic ketoacidosis · hyponatraemia · hypomagnesaemia and hypophosphataemia · hepatic encephalopathy · methanol or ethylene glycol	Hypoglycaemia and toxic alcohols: treat now
Infectious	Meningitis and encephalitis · aspiration pneumonia with sepsis-associated encephalopathy · spontaneous bacterial peritonitis	Treat now if suspected
Metabolic-nutritional	Wernicke's encephalopathy · Korsakoff syndrome · Marchiafava–Bignami disease · pellagra	Wernicke: treat on suspicion
Epileptic	Withdrawal seizures · post-ictal state · non-convulsive status epilepticus · seizures from an underlying lesion	Urgent
Drugs and withdrawal	Alcohol withdrawal and delirium tremens · co-ingested benzodiazepines, opioids or other drugs	Urgent
Structural	Subdural, extradural and subarachnoid haemorrhage · contusion · stroke · osmotic demyelination (often iatrogenic) · hydrocephalus	CT first

1.2 The first ten minutes



Do not delay glucose for a hypoglycaemic patient while waiting for thiamine — give both.

Figure 1. The first ten minutes. Each step can be done before a single blood result is back.

Part II

Wernicke and Korsakoff

Who gets it · How it presents · Thiamine · The glucose question

2.1 A clinical diagnosis

Wernicke's encephalopathy is acute thiamine deficiency affecting the mammillary bodies, medial thalami, periaqueductal grey and the floor of the fourth ventricle. It is common, underdiagnosed and treatable, and the price of missing it is permanent amnesia or death.

- **The triad is the exception.** In Harper's autopsy series, only 16% of patients with Wernicke's lesions had confusion, eye signs and ataxia together, and most had not been diagnosed in life. Confusion alone is the commonest presentation.
- **Caine's criteria** – two of four: (1) dietary deficiency, (2) eye signs, (3) cerebellar dysfunction, (4) an altered mental state or mild memory impairment. In autopsy-confirmed alcohol-dependent patients they identified about 85% of cases, against about a fifth for the classic triad.
- **Eye signs** to look for: horizontal gaze-evoked nystagmus, bilateral sixth nerve weakness, conjugate gaze palsy, vertical nystagmus. Pupils are usually spared.
- **MRI** may show symmetrical T2/FLAIR signal in the medial thalami, mammillary bodies, periaqueductal grey and tectal plate. A normal MRI does not exclude it, and treatment must never wait for the scan.

Not only in people who drink

- Bariatric surgery, especially with early vomiting – common in Saudi Arabia and the Gulf.
- Hyperemesis gravidarum and any prolonged vomiting.
- Cancer, chemotherapy, and long periods of poor intake or parenteral nutrition without vitamins.
- Refeeding after starvation, dialysis, and severe illness in the ICU.
- In these patients the diagnosis is made later than in alcohol-related cases, because no one thinks of it.

2.2 Thiamine: how much

No randomised trial has established the right dose; the recommendations rest on pharmacology and expert consensus. Oral thiamine is poorly absorbed in people who drink heavily, so treatment of suspected Wernicke's is parenteral.

Situation	Commonly recommended
Suspected or established Wernicke's	EFNS: 200 mg IV three times daily, before any carbohydrate, continued until there is no further improvement. Many UK centres use 500 mg IV three times daily for 2–3 days, then 250 mg daily for 3–5 days. Then oral thiamine long term.
At risk, no signs (malnourished, severe withdrawal, ICU)	Parenteral prophylaxis; ASAM notes a commonly used regimen of 100 mg IV or IM daily for 3–5 days, with no agreed dose. Higher doses are reasonable when the risk is high.
Always alongside	Check and correct **magnesium**: thiamine-dependent enzymes need it, and response to thiamine may fail if it stays low. Correct phosphate and potassium.
Safety	Serious reactions to IV thiamine are very rare. Give it diluted over about 30 minutes, with resuscitation equipment available.

"Thiamine before glucose" — what the evidence says

The teaching comes from case reports and animal studies. A 2012 review found nineteen papers on the question, none of them a trial or a controlled cohort, and concluded that there is no evidence to justify delaying glucose in a hypoglycaemic patient. ASAM states that thiamine may be given before, with, or after glucose. The safe practice is simple: **treat hypoglycaemia at once, and give thiamine at the same time.** What should be avoided is a long glucose infusion in an at-risk patient with no thiamine at all.

2.3 Korsakoff syndrome

Korsakoff syndrome is the chronic amnestic state that follows untreated or undertreated Wernicke's encephalopathy: a severe inability to form new memories, a retrograde amnesia with a temporal gradient, and relatively preserved other cognitive functions and social manner. Confabulation is classically described but is less constant than teaching suggests, and fades with time. It is rarely an emergency diagnosis. The emergency question is whether the patient in front of you has an acute Wernicke's that can still be treated — and that question should be answered with thiamine.

Part III

Withdrawal, seizures and delirium tremens

The timeline · Assessing severity · Treatment · Withdrawal seizures

3.1 The timeline

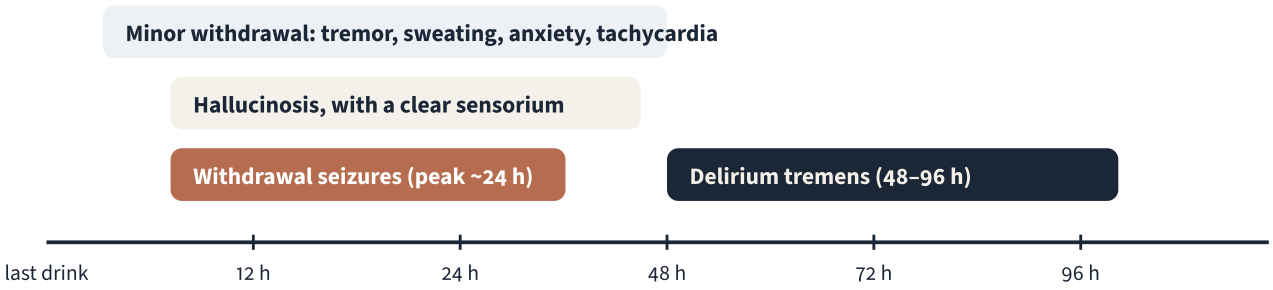


Figure 2. Typical timing after the last drink or a sharp reduction. Timings vary, and a patient may present at any point on the line. Delirium tremens is delirium with autonomic hyperactivity and is the dangerous end of the spectrum.

3.2 Assessing severity

- **Predict risk early.** ASAM recommends identifying patients at risk of severe or complicated withdrawal with a structured tool such as **PAWSS**, and by history: previous delirium tremens or withdrawal seizures, heavy daily drinking, older age, concurrent illness, and a high blood alcohol level with symptoms already present.
- **Measure the response.** The **CIWA-Ar** monitors severity and guides symptom-triggered treatment. It needs a patient who can answer questions; in a drowsy, delirious or intubated patient use RASS and a delirium tool instead.
- **Withdrawal is also a diagnosis of exclusion.** The commonest error in this whole field is to label new agitation or confusion "withdrawal" and stop looking. Subdural haematoma, meningitis, hepatic encephalopathy, hypoxia and sepsis all present this way.

3.3 Treatment

Drug	Place (ASAM 2020)
Benzodiazepines	First line: they reduce withdrawal seizures and delirium. Symptom-triggered dosing guided by CIWA-Ar is preferred to fixed schedules where staff can assess reliably; front-load a long-acting drug (diazepam, chlordiazepoxide) in severe withdrawal. In liver failure or the elderly, lorazepam or oxazepam avoid accumulation.
Phenobarbital	An acceptable alternative or adjunct in severe or benzodiazepine-resistant withdrawal, in settings experienced with its use and able to monitor respiration.
Alpha-2 agonists (clonidine, dexmedetomidine)	Adjuncts for persistent autonomic hyperactivity only. Never as monotherapy: they do not prevent seizures or delirium tremens.
Antipsychotics	Adjuncts for hallucinosis or agitation not controlled by adequate benzodiazepines; never alone, because they lower the seizure threshold and do not treat withdrawal.
Carbamazepine, gabapentin	Alternatives in mild to moderate withdrawal when benzodiazepines cannot be used; not first line.

Drug	Place (ASAM 2020)
Everyone	Thiamine, magnesium, phosphate, fluids, nutrition, and a calm, well-lit room.

3.4 Withdrawal seizures

Withdrawal seizures are usually generalised tonic–clonic, single or a brief cluster, and occur within about 6–48 hours of the last drink. In a randomised trial of 186 patients who presented with an alcohol-related seizure, 2 mg of intravenous lorazepam reduced the rate of a second seizure in the emergency department from 24% to 3%, and reduced admissions.

- Treat the seizure and the withdrawal with a benzodiazepine. Phenytoin does not prevent withdrawal seizures and is not indicated for them.
- A **first** seizure in a drinker still needs the work-up of any first seizure, including imaging: head injury, subdural haematoma and old contusions are common causes.
- **Focal onset, status epilepticus, a seizure outside the withdrawal window, or a slow recovery** point to another cause. Non-convulsive status epilepticus is a recognised cause of "persistent intoxication".

Part IV

The structural causes

Subdural haematoma · Head injury · When to scan, and when to scan again

4.1 Why subdurals are so common

Alcohol shrinks the brain and stretches the bridging veins; it causes falls; and liver disease causes thrombocytopenia and coagulopathy. The history of a fall is often absent, forgotten or denied. Heavy drinking is also associated with a higher risk of intracerebral haemorrhage.

Clues that should lower your threshold even further

- Any focal sign, unequal pupils, or a GCS that is falling or fluctuating.
- Headache or vomiting, any visible injury, any period that no one witnessed.
- Anticoagulants, antiplatelets, or known cirrhosis.
- A patient who is not improving at the rate the blood alcohol level predicts.

4.2 Imaging

Request non-contrast CT of the head in any patient with an altered mental state and a history of alcohol use when there is a head injury or possible fall, a focal sign, anticoagulation or coagulopathy, a seizure, or failure to improve during observation. Repeat it within hours when the picture changes or does not fit a first normal scan. Check the platelet count and coagulation, and reverse coagulopathy according to local protocol when there is bleeding. Discuss with neurosurgery any haematoma with mass effect, midline shift or a falling GCS.

"Sober up and reassess" is a plan only if someone reassesses

Blood alcohol falls by roughly 15–20 mg/dL per hour in most adults, faster in heavy drinkers. A patient who is no better after several hours of observation, or whose level does not explain the depth of the coma, has another cause until proved otherwise.

Part V

Metabolic and toxic causes

Sodium · Glucose and ketoacidosis · The liver · Toxic alcohols · Infection

5.1 Sodium, and the danger of correcting it

Hyponatraemia is common: poor intake, beer potomania, vomiting, diuretics and SIADH. Patients with alcohol use disorder, malnutrition, hypokalaemia or liver disease are at the **highest risk of osmotic demyelination** if the sodium rises too fast.

- Severe symptoms (seizures, coma): give hypertonic saline in boluses to raise the sodium by about 5 mmol/L, then slow down.
- Limit the rise. The European guideline sets 10 mmol/L in the first 24 hours and 8 in each 24 hours after; in high-risk patients, including those with alcohol use disorder, aim for **no more than 8 mmol/L in 24 hours**, and lower targets are often recommended.
- **Beer potomania** corrects itself dangerously fast once the patient receives saline and food and a water diuresis begins. Measure sodium every few hours and be ready to stop the rise with water or desmopressin.
- Osmotic demyelination appears 2–6 days after the correction: dysarthria, dysphagia, quadriparesis, a locked-in state. It can mimic the illness the patient came in with.

5.2 Glucose and ketoacidosis

Alcohol suppresses gluconeogenesis, and a malnourished drinker can become severely hypoglycaemic hours after the last drink. Check a capillary glucose in every patient, more than once.

Alcoholic ketoacidosis follows a binge, then vomiting and poor intake: tachycardia, abdominal pain, a high anion gap, ketones, and a glucose that is normal or low – which separates it from diabetic ketoacidosis. Standard nitroprusside tests detect acetoacetate, not beta-hydroxybutyrate, and can underestimate the ketosis. Treatment is dextrose-containing fluids with saline, thiamine, and replacement of potassium, magnesium and phosphate; insulin is not needed.

5.3 Hepatic encephalopathy

- Look for cirrhosis, asterixis, jaundice, ascites – and above all a **precipitant**: gastrointestinal bleeding, infection including spontaneous bacterial peritonitis, constipation, dehydration, diuretics, hypokalaemia, sedatives.
- **Ammonia** does not grade severity and a high level does not make the diagnosis. EASL advises that a **normal** ammonia in a patient thought to have overt encephalopathy should prompt a search for another cause.

- Treat the precipitant; lactulose titrated to two or three soft stools a day; rifaximin added to prevent recurrence after an episode.
- Do not attribute confusion in a cirrhotic drinker to the liver without considering Wernicke's, subdural haematoma, infection and hyponatraemia. All are more common in exactly this patient.

5.4 Toxic alcohols and infection

- **Methanol and ethylene glycol**: a high anion gap metabolic acidosis with a raised osmolal gap, visual symptoms (methanol) or renal failure and oxalate crystals (ethylene glycol). Contact poisons services early; fomepizole or ethanol, and dialysis.
- **Meningitis and encephalitis**: drinkers are at higher risk and the signs are blunted. Fever, neck stiffness, or encephalopathy with no explanation should lead to blood cultures, antibiotics and aciclovir without delay, and a lumbar puncture once CT has excluded a contraindication when one is needed.
- **Aspiration pneumonia and sepsis** are common causes of encephalopathy in this group.

Part VI

The wider neurology of alcohol

Marchiafava–Bignami disease · The other syndromes you will meet

6.1 Marchiafava–Bignami disease

A rare demyelination and necrosis of the corpus callosum in malnourished heavy drinkers. The acute form presents with impaired consciousness, seizures, dysarthria or a picture that mimics Wernicke's, stroke or psychiatric illness; the chronic form with dementia, gait disorder and signs of interhemispheric disconnection. CT is often normal or non-specific. MRI shows callosal lesions, classically in the central layers with sparing of the dorsal and ventral rims. There is no proven treatment; a review of imaging-confirmed cases found better outcomes in patients given parenteral thiamine early, so treat as for Wernicke's.

6.2 Other alcohol-related syndromes

Syndrome	What you see
Cerebellar degeneration	Wide-based gait and leg ataxia with relatively normal arms and speech — the anterior superior vermis is affected first. Often coexists with Wernicke's.
Peripheral neuropathy	Painful, length-dependent sensory and later motor neuropathy; toxic and nutritional causes overlap.
Compression neuropathies	Radial nerve palsy ("Saturday night palsy"), peroneal palsy after prolonged lying.
Myopathy	Acute necrotising myopathy with rhabdomyolysis after a binge; chronic proximal myopathy.
Nutritional optic neuropathy	Painless, bilateral, symmetrical loss of central vision with central or centrocaecal scotomas.
Pellagra	Niacin deficiency: encephalopathy with dermatitis on sun-exposed skin and diarrhoea — and sometimes no skin signs at all. Treat with nicotinamide.
Acquired hepatocerebral degeneration	Parkinsonism, ataxia and cognitive decline in chronic liver disease, with T1 hyperintensity of the globus pallidus.

Syndrome	What you see
Alcohol-related brain damage	Cognitive impairment, especially executive and memory, which may partly recover with abstinence and nutrition.

Part VII

Putting it together

The work-up · Six principles · Cases

7.1 The work-up

Bedside	**Capillary glucose, full observations including temperature, GCS trend, pupils, eye movements and nystagmus, gait if safe, asterixis, focal signs, signs of injury**
Blood	Full blood count, urea and electrolytes, magnesium, phosphate, calcium, glucose, liver function, coagulation, blood alcohol, venous gas with lactate, anion and osmolal gaps, ammonia only if the picture is hepatic, blood cultures if febrile, toxicology as indicated
ECG	Arrhythmia, QTc (electrolytes, before any antipsychotic)
Imaging	CT head on the indications in section 4.2; MRI when Wernicke's, Marchiafava–Bignami or osmotic demyelination is suspected — never delaying treatment
Further	Lumbar puncture when infection is possible; EEG for fluctuating or unexplained coma, or a slow recovery after a seizure

Six principles

1. Thiamine early, parenterally, and generously – and never let it delay glucose.
2. Wernicke's is a clinical diagnosis; treat on suspicion.
3. Scan the head liberally; falls are forgotten and coagulopathy is common.
4. "Intoxication" and "withdrawal" are diagnoses of exclusion.
5. Correct sodium slowly; these are the patients at highest risk of osmotic demyelination.
6. Reassess: this patient changes over hours, and the trajectory is the diagnosis.

7.2 Cases

Case 1

A 48-year-old man is brought in "drunk again". He is drowsy and smells of alcohol. His blood alcohol is modest. Four hours later he is drowsier, and the nurse notices the left pupil is larger than the right.

A. Continue observation; he is sleeping it off

B. Urgent CT head

C. Give lorazepam for withdrawal

D. Check ammonia

B. A falling GCS with anisocoria and a level that does not explain the coma: a subdural or extradural haematoma until proved otherwise. Scan now, check coagulation, and involve neurosurgery.

Case 2

A 36-year-old woman three months after a sleeve gastrectomy has vomited for two weeks. She is confused and unsteady, with horizontal nystagmus on lateral gaze. She does not drink alcohol.

A. Functional disorder
B. Wernicke's encephalopathy
C. Vestibular neuritis
D. Dehydration only

B. Three of Caine's four criteria. Give high-dose IV thiamine at once, correct magnesium, and only then start dextrose-containing fluids. Wernicke's after bariatric surgery is well described and easily missed.

Case 3

A 52-year-old man has a generalised tonic–clonic seizure in the waiting room, 30 hours after his last drink. He recovers fully within 20 minutes. He has had similar seizures before during withdrawal, and his CT is normal.

A. Load with phenytoin
B. Lorazepam 2 mg IV and treat the withdrawal
C. Discharge with no treatment
D. Start levetiracetam long term

B. A typical withdrawal seizure with a normal scan and a known pattern. Lorazepam reduces early recurrence; phenytoin does not prevent withdrawal seizures. Treat the withdrawal, give thiamine, and observe.

Case 4

A 58-year-old man with a sodium of 108 mmol/L after weeks of drinking beer and eating little is given saline. Twelve hours later his sodium is 119 mmol/L and he is passing large volumes of dilute urine.

A. Continue; he is improving
B. Stop the rise: the correction is already above the safe limit for a high-risk patient
C. Give furosemide
D. Start fluid restriction only

B. A rise of 11 mmol/L in 12 hours in a patient at the highest risk of osmotic demyelination. Stop the saline, consider water and desmopressin to halt or reverse the rise, and measure sodium every 2–4 hours.

Case 5

A 61-year-old woman with cirrhosis is confused and has asterixis. Her ammonia is normal. She fell at home two days ago.

A. Hepatic encephalopathy; start lactulose and wait

B. Ammonia excludes encephalopathy entirely

C. Look for another cause: CT head, thiamine, infection screen including ascitic tap, sodium

D. Withdrawal; give diazepam

C. A normal ammonia should make you doubt hepatic encephalopathy as the sole explanation, and the fall and coagulopathy make a subdural likely. Lactulose may still be appropriate, but only alongside the search.

Appendix

Sources

Guidelines, trials and reviews

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