

EMERGENCY MEDICINE PRACTICE

AN EVIDENCE-BASED APPROACH TO EMERGENCY MEDICINE

Alcohol-Related Emergencies: A New Look At An Old Problem

3:00 a.m.: Medics bring in a 40-something male “found down” in an alley. He has an empty pint bottle in his pocket and is unable to offer any historical information. In fact, the patient does not respond to verbal stimuli; instead, he moans and curses almost incoherently. The sweet/medicinal smell of emesis and gin is nauseating.

The doctor carries on and notes that the patient's vital signs are normal. The only sign of trauma is a hematoma above his left eye. Because the patient is restless and combative, he is placed in a back room to “sleep it off.”

7:00 a.m.: Change of shift. You arrive in the ED eager for the new day. On turnover, the night doc reports that the patient is doing fine; in fact, after a restless night, he hasn't made a sound for hours. Your optimism evaporates as you instantly realize: “This is a bad sign.”

*“If on my theme I rightly think,
There are five reasons why men drink,
Good wine, a friend, because I'm dry,
Or lest I should be by and by,
Or any other reason why.”
—John Sirmond, 1589-1649*

BOTH in terms of absolute numbers and diversity of problems, Bethanol (and its “toxic cousins,” methanol, ethylene glycol, and isopropanol) is directly and indirectly responsible for a staggering variety of pathology. A review of the literature on alcohol yields more statistics than baseball; suffice it to say that its financial impact on society is staggering. Despite the existence of alcohol throughout history, notwithstanding the frequency with which intoxicated patients present to the ED, there is remarkably little uniformity in the way these patients are managed. This issue of *Emergency Medicine Practice* reviews the recent literature and its implications for the emergency evaluation and treatment of the alcohol abuser.

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CME Objectives

Upon completing this article, you should be able to:

1. safely manage the acutely intoxicated patient in the ED while avoiding common pitfalls;
2. describe the characteristic metabolic disturbance seen in ethylene glycol and methanol toxicity and list the indications for hemodialysis;
3. identify and treat patients with alcoholic ketoacidosis;
4. manage disulfiram-related emergencies; and
5. explain the medicolegal aspects of caring for intoxicated patients, including the use and misuse of blood alcohol levels.

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Ethanol: Background

Definitions

Alcohol tolerance means that over time, the person must consume more alcohol to achieve the same intoxicating effects. With dependence, the drinker develops withdrawal symptoms with cessation of alcohol. Alcohol addiction involves drastic behaviors used to maintain alcohol intake and often involves socially inappropriate behavior.

Epidemiology

Although there is credible evidence that moderate drinking (2-6 drinks per week) is associated with health benefits,¹ daily alcohol intake of greater than two drinks results in increased mortality.^{2,3} For adults in the United States, the 12-month prevalence of alcohol abuse or dependence is estimated to range between 7.4% and 9.7% of the population, with a lifetime prevalence estimated as high as 13.7% to 23.5%.⁴ Alcohol abuse and dependence in adolescents, while less well-defined, no doubt contributes greatly to morbidity and mortality in that age group as well. The prevalence of alcohol dependence or abuse among ED patients is significantly higher, with estimates ranging as high as 40% in some populations.⁵

More than 107,000 alcohol-related deaths are reported each year. Some are medical in nature (as in liver failure), while others are due to trauma. Twenty-five to 40% of trauma patients are injured while under the influence of alcohol.⁶ Despite this phenomenal human toll, there is very little prospective research regarding the emergency management of alcohol-related disease. In a MEDLINE search covering the years 1966 to 2000, none of the 4744 citations under the Medical Subject Headings "guidelines" or "practice guidelines" specifically addressed acute alcohol intoxication. However, six references focused on the management of alcohol withdrawal.⁷⁻¹²

Pharmacokinetics And Metabolism

Ethanol is rapidly absorbed from the gastrointestinal (GI) tract—20% from the stomach and the remainder through the small intestine.¹³ While absorption usually begins in minutes, the presence of food in the stomach, delayed GI motility, underlying GI disease, or co-ingestion of other drugs can delay absorption. Other factors that contribute to delayed effect include the concentration of ethanol in the ingested drink and individual metabolism.¹⁴ In general, one drink (1 ounce of hard liquor, 1 beer, or 1 glass of wine) raises the blood alcohol level 25 mg/dL.

Ethanol is partially oxidized by alcohol dehydrogenase located in the gastric mucosa. This gastric metabolism is reduced in women, however, accounting for enhanced absorption, higher blood ethanol levels, and possibly an increased susceptibility to alcohol-related disease.¹⁵ Alcohol, once absorbed, is rapidly distributed throughout total body water.

More than 90% of absorbed ethanol is oxidized in the liver. This is accomplished via alcohol dehydrogenase (ADH), a separate microsomal ethanol-oxidizing system (MEOS), and a peroxidase-catalase system. The remaining

5%-10% is excreted unchanged through the kidneys and lungs. The ratio between the concentration of ethanol in the intrapulmonary space and the blood is fairly constant, meaning that the measurement of alcohol in the breath accurately reflects blood alcohol concentration.¹⁶

Because the ADH enzyme system becomes saturated at very low blood ethanol concentrations, metabolism moves to zero-order kinetics, so a fixed amount of alcohol is metabolized per unit of time, regardless of concentration. Practically speaking, an average-size non-ethanol-tolerant adult will metabolize ethanol at a rate of 20 mg/dL/h.¹⁴ Chronic drinkers, through induction of the MEOS system, may potentially increase their rate of clearance up to 30 mg/dL/h,^{17,18} though ED studies indicate that there is a high degree of variability.^{19,20} In general, 25 mg/dL/h is a good "rule of thumb" to estimate the rate of ethanol clearance.

The search for agents to accelerate the clearance of ethanol or to reverse its clinical effects has been largely fruitless. Neither naloxone nor flumazenil is effective.²¹⁻²³ While IV fluids are commonly administered to intoxicated patients, there is no evidence that this practice accelerates sobriety. In one crossover volunteer study, IV fluids did not improve ethanol clearance.²⁴

There are well-documented ethnic differences in the response to ethanol. Eighty percent of Asians have been noted to respond with facial flushing and intoxication to doses of ethanol that have no effect on Caucasians; 50% of Japanese are predisposed to a reaction.²⁵ This reaction, known as the "alcohol hypersensitivity syndrome," may be due to abnormal enzymatic activity that raises acetaldehyde levels more than 10 times higher than normal after ethanol consumption.²⁶

There have been a few reports that the ingestion of H₂-blockers can significantly impair the metabolism of alcohol, resulting in higher blood levels for a given number of drinks.²⁷ More recent research demonstrates that use of cimetidine, ranitidine, or omeprazole does not significantly change ethanol elimination.²⁸

Differential Diagnosis

In essence, the diagnosis of alcohol intoxication is one of exclusion. (See **Table 1**.) Even if the patient is intoxicated, other pathology may be afoot. The emergency physician must be ever-vigilant for concomitant disease or occult trauma. The chronic user of alcohol, in particular, has an increased incidence of pneumonia, lung abscess, meningitis, cardiomyopathy, coagulopathy, and may be at higher risk for suicide.²⁹⁻³³ Alcohol increases the risk of injury, in part because alcohol correlates with other risk factors such as speeding and not wearing seatbelts.^{34,35} Whether alcohol adversely affects the severity and outcome of injury remains controversial.³⁶

Prehospital Care

The alcohol-intoxicated patient represents a large share of EMS transports. In a retrospective analysis of urban EMS transports, intoxicated individuals were transported more frequently than patients with seizures or respiratory

conditions. Of all patients who were transported five or more times during the study period, 71% were for alcohol.³⁷ Intoxicated patients also account for a significant fraction of municipal EMS budgets.³⁸ In addition, alcohol often plays a role in those who refuse treatment or transport by paramedics.³⁹ Surprisingly, many urban EMS policies do not address the issue of alcohol intoxication when deciding whether to allow patients to refuse care.⁴⁰

Prehospital care for the alcohol-intoxicated patient is largely supportive. Co-morbid conditions, particularly trauma, must be addressed as appropriate. All patients with an altered mental status require an assessment of their blood sugar. Of particular concern to prehospital management are issues related to the cervical spine and prevention of aspiration.

ED Evaluation

Stabilization And Initial Evaluation

Evaluation of the acutely intoxicated patient begins with the ABCs. When in doubt, a definitive airway should be secured.

In the absence of a clear mental status, the cervical spine should be considered injured until cleared both radiographically and clinically. In a prospective study of 974 trauma patients, 27 of whom had a cervical fracture, there was one patient with no clinical findings suggestive of a cervical injury other than a blood alcohol level of 200 mg/dL.⁴¹ In the more recent NEXUS trial, five patients with clinically significant cervical spine injuries—just less than 1% of all patients with clinically significant fractures—had intoxication as the sole indication for radiography (i.e., they had no spinal tenderness, neurological deficit, etc.).⁴²

Oxygen saturation via pulse oximetry is certainly indicated for unstable patients or those who have tachypnea or respiratory distress, but the role of routine pulse oximetry in the intoxicated patient remains unknown. An accurate set of vital signs including a temperature is critical for clinical decision-making. A rectal temperature may be more

accurate in patients with tachypnea or mouth breathing.^{43,44}

Blood glucose via bedside glucometry should be quickly obtained in patient with altered mental status. Up to 8.5% of ED patients with altered mental status are hypoglycemic.⁴⁵ While alcohol may cause hypoglycemia, a retrospective review and a prospective study of ethanol-positive adult ED patients failed to identify an increased frequency of hypoglycemia when compared with controls.^{46,47} Even if alcohol itself does not drop the blood sugar, a diabetic may get drunk and become hypoglycemic. Alcohol-induced hypoglycemia is more commonly reported in children than adults.^{48,49}

In a detailed review of the “coma cocktail,” Hoffman and Goldfrank advocate the use of dextrose (25 g of 50% dextrose in water; Abbot, \$21.26 per dose) in all patients with altered mental status when a bedside glucose determination is unavailable or when the determination is low.⁵⁰ (“Low,” in their view, is a perhaps overly conservative 120 mg/dL).

Thiamine (100 mg IV) is usually given at the time dextrose is administered in order to prevent inducing Wernicke’s encephalopathy,⁵⁰ although the empiric data for this routine practice is admittedly slim. (See the discussion in a subsequent section.) Never delay dextrose because thiamine is not readily available.

Naloxone is indicated only if there is suspicion of concomitant opiate use (i.e., miosis, hypoventilation [respiratory rate < 12], or evidence of recent opiate use).⁵¹ Flumazenil has no role in the acute management of alcohol intoxication and indeed will interfere with the effectiveness of benzodiazepines, the drugs of choice for the treatment of acute ethanol withdrawal.

History

History, while often the most important aspect of a patient evaluation, may be scant or unreliable in the intoxicated patient. Determine their bibulous habits, recognizing that reports regarding the amount of alcohol consumed may be apocryphal (“just two beers”). EMS may provide valuable information.

Useful factors may include the time of the last drink (or history of decreased intake), “usual” amount of alcohol consumed per day, any record of alcohol withdrawal, other drug use, and past medical history (e.g., diabetes and pancreatitis). Also inquire as to any medications the patient may be taking (e.g., disulfiram, warfarin, phenytoin, or agents that might depress mental status). For the patient with a presumed withdrawal seizure, whether or not they have had a prior withdrawal seizure becomes crucial. In the patient with abdominal pain or tachypnea, ask regarding alternative drinks such as wood or rubbing alcohol. (Screening questions for alcohol abuse or dependence are discussed in a later section.)

Physical Examination

Once the patient is stabilized, a careful head-to-toe physical examination (the “secondary survey”) must be performed. Of particular importance for the intoxicated patient is a thorough search for evidence of trauma, especially

Table 1. Differential Diagnosis Of The Patient Suspected Of Alcohol Intoxication.

Intracranial injury

- Hemorrhage
- Ischemia

Infective processes

- Intracranial
- Systemic

Metabolic abnormality

- Hypoglycemia
- Hyponatremia
- Hypoxemia
- Hypo- or hyperthermia
- Hepatic encephalopathy

Toxic exposure (polysubstance abuse)

Seizure disorder

- Postictal
- Nonconvulsive status

Withdrawal syndromes

head injury.

One of the most important aspects of the evaluation is a detailed neurological exam looking for evidence of focality.⁵² The examination should include evaluation of the level of consciousness, pupils, eye movements, cranial nerves, and motor and cerebellar function (including, at some point, gait testing). Horizontal nystagmus is frequent in intoxication but varies greatly among individuals and according to whether the subject's blood alcohol concentration is rising or falling.⁵³

Serial neurologic examinations—looking for improvement or deterioration over time—are perhaps more important than any single assessment. In a Norwegian study, a group of 429 intoxicated patients were evaluated by an ED protocol of repeated clinical examinations including a modified Glasgow Coma Scale (GCS). This approach, followed over several hours, was successful in sorting out the seriously ill from the “intoxicated only” patients.⁵⁴ Documenting serial GCS scores may be helpful, especially if multiple caretakers are involved during the patient's ED stay.

The patient clinically addicted to alcohol may show physical signs of alcohol dependence, such as rhinophyma, palmar erythema, spider angiomas, hepatomegaly, and/or testicular atrophy. Other findings may include “chipmunk cheeks” (parotid enlargement), gynecomastia, acne rosacea, and Dupuytren's contractures of the hands.

Sobriety Tests: Predicting Intoxication

The acutely intoxicated patient can vary in clinical appearance from lucid to comatose. It is extremely difficult to predict with any degree of confidence the alcohol level in a given patient. Accuracy of the standardized field sobriety test, long used by law enforcement, has been called into question.⁵⁵ A new boating sobriety test found horizontal gaze nystagmus, difficulty reciting the alphabet from A to Z, and difficulty with clapping hands while counting is associated with blood alcohol concentrations above 100 mg/dL, though the predictive value was no better than the standardized field sobriety test.⁵⁶ A recent trial investigating police officers' ability to detect breath odors from alcohol ingestion showed a significant rate of false-negatives.⁵⁷ It is worth pointing out, however, that most of the errors reported in this trial were related to *failing* to detect alcohol (false-negative), not in overestimating its presence (false-positive).⁵⁷

Most of the literature involves the ability of law enforcement personnel to detect intoxication. But can the emergency physician accurately detect the degree of intoxication based on the clinical examination (slurred speech, alcohol on breath, cerebellar function)? While many seasoned physicians have “won the pool” when predicting the blood alcohol level (BAL), studies on this are few. In one ED study, an Alcohol Symptom Checklist (a clinical scoring tool) did not correlate with the blood alcohol in intoxicated patients.⁵⁸

Diagnostic Studies

Blood Alcohol Levels

BALs correlate poorly with the degree of intoxication

observed clinically.²⁰ Despite this, some authors recommend a BAL on all patients who appear clinically intoxicated (an expensive and probably unnecessary approach),¹⁴ while others suggest a more selective (and rational) strategy.⁵⁹

While BALs may be useful in the prosecution of drunk drivers and possibly as part of alcoholism intervention programs, there are very few data demonstrating actual clinical utility. While it seems reasonable that they can be helpful when the diagnosis of intoxication is in doubt, this is not well-studied.

One possible benefit of BALs lies in the correlation between the BAL and the GCS score in patients with potential or known head injury. A prospective study of 918 head-injured patients showed that the GCS is not statistically affected by the presence of alcohol until the BAL is 200 mg/dL or more.⁶⁰ Coma was generally associated with an intracranial injury. The authors argue that knowledge of the BAL could be of “considerable diagnostic value,” especially if the mental status is depressed. This study would suggest that if the BAL is below 200 mg/dL and the patient with a possible head injury has a GCS of 14 or less, head CT would be indicated. Though prospective in design, this study is limited due to absence of routine correlation with neuroimaging and selection bias. Another study found that high BALs (>240 mg/dL) were associated with only a 2-3 point reduction in GCS in intoxicated assault victims in the absence of intracranial injury.⁶¹ This again implies that in an assault victim, a GCS of 13 or lower may be due to head trauma rather than intoxication.

The link between alcohol (ETOH) use and trauma is well-established; almost half of all trauma patients admitted to hospitals have detectable ethanol levels.^{62,63} The American College of Surgeons' Committee on Trauma recommends drug and alcohol screening as “essential” for level I and II and “desirable” for level III trauma centers.⁶⁴ However, this recommendation is not based on any convincing evidence of improved patient outcome. Indeed, a survey of trauma centers reveals that despite available resources and repeated ACS recommendations, measurements of blood alcohol levels and drug screens are routine in only 63.7% of level I and 40.0% of level II trauma centers.⁶⁵

An acceptable practice regarding BALs is to use clinical judgment. Obtain alcohol levels on patients in whom the diagnosis of intoxication is uncertain. Levels might also be useful in the evaluation of an intoxicated patient with clinical evidence of mild head injury. If the BAL is inconsistent with the patient's level of consciousness, it is imperative to search for the underlying disorder.

There are three methods for determining blood alcohol levels: a laboratory serum analysis, breath analysis, and saliva alcohol analysis. The breath analysis is dependent on patient cooperation, while a saliva analysis is dependent on obtaining an adequate specimen. When the tests are performed properly, all three methods are reliable.⁶⁶⁻⁶⁸

Laboratory Testing

Laboratory testing of blood is best guided by clinical circumstances. There is no evidence to support the “routine” ordering of blood tests for the uncomplicated ethanol-

intoxicated patient. All intoxicated patients with significant or persistent decrease in level of consciousness require bedside assessment of glucose.

Laboratory tests may be useful when the patient has other known or suspected medical problems (e.g., liver failure). Hypomagnesemia and hypokalemia can occur with chronic alcohol abuse, although the incidence and clinical relevance is not well-studied, thus making evidence-based recommendations impossible.

Alcoholism may also affect routine laboratory tests. Hyperamylasemia is frequent in alcohol intoxication even when there is no clinical evidence of pancreatitis.⁶⁹ However, lipase levels are not necessarily more accurate in establishing the diagnosis of pancreatitis in alcoholics, and decisions are best made on clinical findings.^{70,71}

Computed Tomography

The indications for a head CT scan in the alcohol-intoxicated patient have not been well-studied. In one small, prospective series of alcohol-intoxicated patients with minor head trauma, clinical parameters and neurologic exams were unable to predict intracranial injury.⁴⁸ Although no well-designed cohort study has shown improved outcome with non-selective neuroimaging of these patients, the alternative is admission for close observation or prolonged observation with frequent serial re-examination in the ED. However, in some busy EDs, frequent re-examination is not always possible. Consequently, a liberal policy for scanning the intoxicated patient with evidence of head trauma may be prudent, especially if serial re-examinations cannot be ensured.

Some intoxicated patients with evidence of recent head trauma need an emergent or urgent CT rather than serial examinations. While no clinical trials can tell us exactly when to order a CT scan, a few generalizations are supported by the literature. A head CT should be performed sooner, rather than later, in intoxicated patients who have any of the following:

- Clinical evidence of skull fracture
 - Basilar skull fracture: periorbital ecchymosis (racoon's eyes), mastoid ecchymosis (Battle's sign), CSF otorrhea or rhinorrhea, hemotympanum
 - Palpable skull fracture
- Major mechanism of injury and altered mental status
- Level of consciousness more depressed than expected compared to the serum alcohol level
- Significantly altered mental status (GCS $\leq$ 13) and evidence or suspicion of head trauma
- Falling GCS
- Focal neurologic deficit

Cervical Spine Radiography

The NEXUS study demonstrates that, on rare occasions, a traumatized, intoxicated patient may have a cervical spine injury despite lack of spinal tenderness or neurologic deficit. For this reason, trauma patients who are clinically intoxicated should have their cervical spines radiographically evaluated with a minimum of three views or be maintained in spinal precautions until the patient is sober enough to be

evaluated clinically.⁷³ (See the upcoming issue of *Emergency Medicine Practice* for more about cervical spine trauma.)

In practice, this is easier said than done. The intoxicated patient may be restless or combative and ill-disposed to rest quietly in spinal precautions. Always consider the risk-benefit ratio of pharmacologic paralysis in such a situation, taking the mechanism of injury into account. Succinylcholine and intubation may be appropriate for the uncooperative patient thrown through the windshield but not for the average "slip and fall" inebriate with a normal neurologic exam.

Chest And Abdominal Imaging

Chest radiographs are not routinely indicated but should be considered in the presence of chest trauma, pulmonary findings, hypoxia, or a clinical suspicion of aspiration.

Consider the need for CT of the abdomen in the alcoholic who suffers multiple trauma. Alcohol consumption increases the risk of abdominal trauma—a problem that is compounded by the alcoholic's potentially enlarged liver and congested spleen. Intoxicated patients are nearly five times more likely to have an unsuspected injury than non-alcoholics.⁷⁴ Serial examinations and the ED ultrasound of the abdomen play important roles in evaluation.

Management Of Ethanol-Related Conditions

All intoxicated patients with abnormal mental status warrant close observation, even in the presence of a "non-focal" neurological exam. The all-too-common practice of "parking" the patient in a distant corner, only to be forgotten over the next several hours, is to be avoided. These patients should remain visible to staff and require frequent mental status exams.

Patients who exhibit agitation, are disruptive to staff, or potentially threaten their own well-being cannot be allowed to harm themselves or others. The selective use of physical vs. chemical restraints varies among physicians and hospitals. Some believe physical restraints should be applied early when the patient appears combative and be maintained until it is absolutely clear that they are no longer necessary. Other physicians prefer sedation with judicious use of benzodiazepines or butyrophenones. The literature would tend to support the use of butyrophenones for behavioral emergencies due to intoxication and benzodiazepines in the case of a combative patient who is in alcohol withdrawal. In some cases, both physical and chemical interventions may be necessary. Of course, the use of restraints or sedation requires careful monitoring and adherence to mandated protocols.

Alcohol Hangover

*"Those dry Martinis did the work for me:
Last night at twelve I felt immense,
Today I feel like thirty cents.
My eyes are blurred, my coppers hot,
I'll try to eat, but I cannot.
It is not time for mirth and laughter,
The cold, gray dawn of the morning after."
—George Ade, The Sultan of Sulu, 1903*

As day follows night, so does the agony of a hangover chase the evening's intemperance. Annual economic loss in the United States alone, related to poor job performance and absenteeism associated with hangovers, is thought to be \$148 billion, with light-to-moderate drinkers contributing the major share.⁷⁵ Although a strict definition is lacking, headache, diarrhea, anorexia, vomiting, tremulousness, and fatigue are commonly recognized symptoms. It is not unexpected that many such patients seek emergency medical care; likewise, the history of recent alcohol use may not be readily offered. A recent comprehensive review containing little hard evidence suggested that rehydration, prostaglandin inhibitors (non-steroidal anti-inflammatory agents), and vitamin B₆ may represent effective therapy.⁷⁶

Ethanol Withdrawal

Alcohol withdrawal syndrome develops 6-24 hours after a decrease in ethanol intake and lasts from 2-7 days. Some patients experience mild symptoms of irritability and sleeplessness, while others suffer major withdrawal characterized by fever, diaphoresis, and hallucinations. Most patients experience autonomic hyperactivity with tremulousness, sweating, nausea, vomiting, and agitation.⁷⁷ Vital signs reflect an elevation in heart rate and blood pressure. Generalized seizures (alcohol withdrawal seizures) result from neuronal excitation and can occur within 12-24 hours of abstinence, as discussed in the next section.

The most dramatic presentation of alcohol withdrawal is *delirium tremens* (DTs), characterized by auditory and visual hallucinations, confusion, disorientation, and pronounced autonomic hyperactivity. It may be confused with metabolic disorders (such as hypoxia, hypoglycemia, hyperthyroidism, and hepatic encephalopathy) or with infectious disasters (such as sepsis, meningitis, or encephalitis). DTs usually occur on the third or fourth post-abstinence day and may be present in up to 5% of patients hospitalized for alcohol withdrawal.⁷⁸ The incidence of delirium tremens has markedly decreased with the use of benzodiazepines, though it continues to be a significant issue in some populations.⁷⁹ While early studies stated that the mortality rate for delirium tremens was nearly 20%,⁸⁰ modern reviews show a mortality rate of less than 15%.⁸¹

A benzodiazepine is the recommended pharmacologic intervention for managing all facets of alcohol withdrawal.⁸² Lorazepam (Baxter; \$1.56 per 2 mg dose), because of its ease of administration, rapid onset of action, non-hepatic metabolism, and lack of active metabolites, has emerged as the drug of choice in most hospitals. While it may be given by mouth, intramuscularly, or intravenously, the IV route is best suited for the patient in moderate to severe withdrawal. Begin with 2-5 mg lorazepam followed by repeat doses of 2-5 mg every 20 minutes as needed to control symptoms.

When using benzodiazepines, avoid switching agents but instead administer repeated doses of a single agent. β -blockers, clonidine, and carbamazepine may be considered for adjunctive therapy, although their benefit remains unproven.⁸² Phenothiazines are less effective than the

benzodiazepines in managing delirium.⁸² Butyrophenones such as haloperidol and droperidol are sometimes used as adjuncts in managing delirium and behavioral emergencies.

A majority of patients withdrawing from ethanol can be safely managed as outpatients,⁸³ and a significant number without pharmacological intervention.⁸⁴ However, no evidence-based guidelines exist to determine which individuals require aggressive medical intervention or hospitalization. The decision to admit may involve both medical factors (such as vital signs, neurologic examination, and response to therapy) as well as social considerations (including family support, a place to stay, money for medication, and the like). Alcohol detoxification units may be suitable for those who have stable vital signs and are not hallucinating or confused.

Withdrawal Seizures

Alcohol withdrawal seizures usually occur between 12 and 24 hours from the cessation of alcohol intake. While patients may have other signs of withdrawal, seizures may be an isolated phenomenon unaccompanied by a hyper-adrenergic state.

Do patients with alcohol withdrawal seizures need a CT scan of their head? They do if it is their first withdrawal seizure. In one retrospective case series of 259 patients, 6.2% of patients with a first-time seizure temporally related to alcohol use had intracranial lesions on CT.⁸⁵ Neuroimaging is unnecessary if patients have a history of withdrawal seizures and a normal neurological examination in the ED.

Lorazepam is the drug of choice for alcohol withdrawal seizures. It has a smaller volume of distribution than diazepam and thus a longer anticonvulsant duration of action (15 minutes for diazepam vs up to 12 hours for lorazepam).⁸⁶ The effectiveness of lorazepam in this setting was recently confirmed in a randomized, controlled trial.⁸⁷ This study involved patients with chronic alcohol abuse who presented to the ED after a witnessed, generalized seizure. They were randomly assigned to receive either 2 mg of lorazepam or normal saline intravenously and then observed for six hours. Only 3 of 100 patients (3%) who received lorazepam had a second seizure, as compared with 21 of 86 patients (24%) in the placebo group. In addition, admission to the hospital was almost doubled in the placebo arm.

While many alcoholics may list phenytoin as a medicine they take (or are supposed to take), well-designed prospective studies have demonstrated that phenytoin is *ineffective* in preventing alcohol withdrawal seizures.⁸⁸ The emergency physician must be sure, however, that the seizures are in fact related to alcohol withdrawal.

Disulfiram Reactions

Disulfiram (Antabuse®) is a pharmacologic agent that has been used with variable success to reduce the likelihood of relapse in patients with alcohol dependence.^{89,90} It is available in both oral and implantable form. To be effective, however, it must be used in combination with counseling and ongoing behavioral modifications.⁹⁰

Disulfiram functions as an "aversive drug" and serves

as a negative reinforcement to alcohol ingestion. When even small amounts of alcohol are consumed in the presence of disulfiram, an unpleasant reaction—consisting of flushing, headache, nausea, vomiting, vertigo, abdominal discomfort, palpitations, and diaphoresis—occurs. (See **Table 2** for other alcohol-drug reactions that can mimic the disulfiram-ethanol reaction.)

Disulfiram causes irreversible inhibition of aldehyde dehydrogenase. When ethanol is consumed and metabolized, the resultant acetaldehyde accumulates, causing the classic “disulfiram-alcohol reaction.”⁹¹ The symptoms begin as soon as five minutes after ingestion, peak at 15-20 minutes, and may last hours. The intensity of the reaction varies from person to person but in general is proportional to the dose of both alcohol and disulfiram. There is a striking “rash”—a moderate-to-intense erythema of the face, proximal upper extremities, and torso—that can be part of the disulfiram-ethanol reaction. Severe reactions, producing hypotension, dysrhythmias, severe respiratory depression, seizures, and even death, can occur when blood alcohol levels rise above 100 mg/dL. Reactions can also occur from inadvertent use of alcohol-containing products such as over-the-counter cough syrups.

Management of the disulfiram-related reaction/toxicity is largely supportive. Interventions include intravenous hydration, anti-emetics, and cardiac monitoring. Theoretically, metoclopramide should be avoided because of its antagonist properties, which may worsen existing hypotension. A reaction may occur for 1-2 weeks after the last dose; patients need to be informed of this in order to avoid an unintentional reaction.

Alcoholic Ketoacidosis

The patient is with alcoholic ketoacidosis (AKA) is usually a chronic alcoholic with poor nutritional status and recent binge drinking. It begins with a decrease in carbohydrate intake with resultant depletion of glycogen stores. The net effect is exhaustion of glycogen, reduced insulin production, and lipolysis. As free fatty acids are utilized, ketoacids are formed.

The initial symptoms are anorexia and nausea, followed by protracted vomiting and diffuse abdominal pain. These symptoms further reduce oral intake, and a downward spiral begins.

Table 2. Disulfiram-Like Reactions With Ethanol.

Antimicrobials

- Beta-lactams
- Cephalosporins
- Chloramphenicol
- Isoniazid
- Ketoconazole, griseofulvin
- Metronidazole
- Nitrofurantoin
- Sulfonamides

Sulfonylureas

- Chlorpropamide
- Glipizide, glyburide
- Tolbutamide, tolazamide

Clinical Evaluation

On examination, most patients show signs of volume depletion, including tachycardia and orthostatic hypotension. There may be the odor of ketones. Tachypnea and/or Kussmaul respirations (rapid and deep breathing), the body's attempt to compensate for the metabolic acidosis, may be present. Fever is generally absent. Unless there is a co-existing problem such as sepsis or hypoglycemia, mental status is usually normal. Abdominal examination often reveals diffuse tenderness; indeed, AKA can mimic pancreatitis and other causes of an acute abdomen.⁹² (See **Table 3.**)

Laboratory Testing: Acid-Base Determination

AKA is a clinical diagnosis based on history and supported by laboratory testing. The diagnosis is generally made with the help of an electrolyte panel, which will demonstrate a wide anion gap acidosis. A blood gas (either venous or arterial) is not absolutely necessary in classic presentations, but a serum pH can help determine the severity of the acidosis. The acid-base disturbance may be mixed and demonstrate a double or even “triple” disorder. A double disorder occurs when persistent vomiting adds a metabolic alkalosis to the underlying ketoacidosis. A triple disorder occurs when these two phenomena are compounded by a respiratory alkalosis associated with withdrawal. Other metabolic disturbances found in this setting include hyperchloremic acidosis, respiratory alkalosis, and lactic acidosis.⁹³

Serum electrolytes should be determined, as electrolyte disturbances including hypokalemia, hyponatremia, hypomagnesemia, and hypophosphatemia are common.⁹³ Blood glucose levels are variable though usually in the normal range. Alcohol levels are typically low or undetectable; however, AKA can occur with BALs consistent with intoxication.⁹³

In addition to an electrolyte panel, the second routine test for AKA involves measurement of serum and/or urinary ketones. Ketones are present in both the blood and urine of these patients, although not always in measurable form. Routine laboratory tests rely on the nitroprusside reaction, which is sensitive for acetoacetate and acetone but not for β -hydroxybutyrate. Since β -hydroxybutyrate is the major ketoacid in AKA, it is possible to underestimate the severity of the ketoacidosis if one relies on this test alone.^{94,95} Serial acetone determinations demonstrate a paradoxical worsening (becoming more positive) as the β -hydroxybutyrate is metabolized to acetoacetate.

Management

The cornerstones of therapy for AKA are volume repletion

Table 3. Differential Diagnosis In Patients With Suspected Alcoholic Ketoacidosis.

- Diabetic ketoacidosis
- Methanol or ethylene glycol ingestion
- Iron overdose
- Salicylate poisoning
- Severe pancreatitis
- Ischemic bowel

and glucose. (See also **Table 4.**) AKA is a reversible entity that responds rapidly to treatment. It has a low mortality rate, and when death does occur, it is generally attributed to coexisting disorders.^{93,95} Clinical response is the best way to follow the patient's response to intervention.

Give patients both dextrose and saline (D₅NS). Isotonic fluid resuscitation will normalize volume status and promotes renal excretion of both β -hydroxybutyrate and acetoacetate.⁹⁵ Glucose stimulates endogenous insulin and reduces further ketone production. As the ketones are converted back to bicarbonate in the liver, the acidosis resolves. When saline alone was compared to the combination of saline and glucose, the patients who received both therapies resolved their acidosis more quickly.⁹⁶

Potassium and magnesium stores should be repleted, and thiamine administration is advisable. Bicarbonate administration is not indicated in the management of AKA, because correction of the acidosis occurs rapidly once volume is restored and glucose is administered.^{94,95}

Disposition

Most patients will resolve their dehydration and acidosis within 8-12 hours. In one series, almost half of patients (46%) did not require admission.⁹³ Patients with AKA may be well-suited for management in short-term observation units in the ED. Criteria for discharge from the ED may include an anion gap that is headed toward normal (15 or so), resolution of the acidemia, normal hydration, and the ability to tolerate oral fluids. Criteria for admission include inability to tolerate oral intake, persistent metabolic abnormalities, or the existence of an underlying, coexisting, or precipitating illness such as pancreatitis or sepsis.

Wernicke's Encephalopathy And Korsakoff's Psychosis

Symptomatic thiamine deficiency (vitamin B₁) manifests as beriberi. Two entities exist: in one, cardiovascular dysfunction predominates ("wet beriberi"); in the other, neurological abnormalities predominate ("dry beriberi" or Wernicke-Korsakoff syndrome). Although one symptom complex tends to predominate in an individual, overlap may occur. It is believed that an abnormality of trans-ketolase activity (a thiamine-dependent enzyme), in combination with a low-carbohydrate diet, predisposes certain patients to develop neurological rather than cardiovascular complications.⁹⁷ Magnesium is a cofactor for this enzyme.

While the true incidence of Wernicke's encephalopathy is unknown, it is probably under-diagnosed.^{98,99} Many of the

recent reports involve non-alcohol-related conditions, including hyperemesis gravidarum, gastric reduction surgery, and chemotherapy.¹⁰⁰⁻¹⁰² Wernicke's encephalopathy is a true medical emergency, with a mortality rate ranging from 10%-20%. Of those who survive, recovery is often incomplete, with significant long-term morbidity.¹⁰³

ED Evaluation

The triad of Wernicke's encephalopathy consists of oculomotor disturbances (classically, ocular palsies and nystagmus), altered mental status, and ataxia. The full triad is often absent, which can delay the diagnosis and hence treatment. In 1997, new operational criteria for Wernicke's encephalopathy were proposed that specified that two of the following four signs must be present:¹⁰⁴

1. Dietary deficiencies judged by a body mass index less than two standard deviations below normal, a history of grossly impaired dietary intake, or an abnormal thiamine assay.
2. Oculomotor abnormalities including ophthalmoplegia, nystagmus, or a gaze palsy.
3. Cerebellar dysfunction, including ataxia, past pointing, dysidiadokinesia, or impaired heel-shin testing.
4. Altered mental status or mild memory impairment, judged as inability to recall two or more items in the four-item memory test or impairment on more elaborate neuropsychological tests of memory function.

In one study, the sensitivity for the diagnosis of Wernicke's encephalopathy was improved from 31% using the classic triad to 100% using the revised criteria.¹⁰⁴ Korsakoff psychosis is a disorder of memory, particularly recent memory in a setting of clear consciousness.^{105,106} Many, but not all, patients with this disorder have a preceding Wernicke's syndrome.¹⁰⁷

Treatment

Administration of parenteral thiamine hydrochloride (100 mg) is the cornerstone of therapy. Though a few isolated cases of anaphylactic reactions have been reported, IV administration is the preferred route of administration in the ED.¹⁰⁸ The ocular findings of Wernicke's syndrome may resolve within hours, but other deficits may require days, and recovery may be incomplete.

There have been concerns that the administration of hypertonic dextrose to patients without thiamine administration could precipitate Wernicke's encephalopathy.¹⁰⁹ For this reason, it has generally been recommended that thiamine be given before hypertonic dextrose. However, the evidence for this is weak, based on poorly described case reports where the patients involved either had prolonged dextrose without further nutritional support or had evidence of early Wernicke's encephalopathy before they received the dextrose.

Following the initial dose, thiamine should be continued on a daily basis along with additional supportive measures including IV fluids, repletion of potassium and magnesium, and maintaining adequate glucose levels.

Table 4. Management Pearls In Alcoholic Ketoacidosis.

- A negative nitroprusside reaction does not rule out ketoacidosis.
- An increasingly positive nitroprusside reaction is consistent with an improvement rather than a worsening of the ketoacidosis.
- Patients in AKA *do not* need insulin.
- Fluid resuscitation in AKA should include dextrose and saline.

Admission is indicated in order to provide ongoing care and aggressive nutrition support.

While patients with Wernicke's syndrome certainly need thiamine, the more germane question is: "Does every alcoholic need IV nutritional/metabolic therapy in the ED?" Anyone who works in an ED is familiar with the ubiquitous "yellow bag" (sometimes known as the "Rally Pack"). These bags of IV fluids, hanging solicitously over the alcoholic, generally contain a concoction of thiamine, folate, multivitamins, and magnesium. A recent review questions the value of this practice.¹¹⁰ The authors argue that single IV doses of these elements are not likely to reverse a long-standing deficiency and point out that there is no direct evidence linking hypomagnesemia to alcohol withdrawal, as is commonly believed. Perhaps more importantly, there is no evidence that this expensive practice does any good.

While routine administration of thiamine to all alcoholics may again be "overkill," toxicity is unlikely. Many argue that its widespread use is justified as the mortality and morbidity associated with Wernicke-Korsakoff syndrome is significant.^{50,103,108}

The "Other" Alcohols: Isopropanol, Methanol, and Ethylene Glycol

Isopropyl Alcohol

Isopropanol is a common household product, making it easily accessible for accidental and intentional ingestion. It is a colorless, bitter-tasting liquid. Some preparations contain other toxins (e.g., salicylates), and, as such, every effort should be made to identify the exact product involved. Desperate players may drink isopropanol when their preferred drink is unavailable. There have even been reports of intoxication from rubbing alcohol enemas.¹¹¹ (Whose idea was that?)

Isopropyl causes the same level of "drunkenness" at a serum level approximately half that of ethanol. It is rapidly absorbed, and approximately 80% is metabolized to acetone by alcohol dehydrogenase (ADH) following first order kinetics. The remaining 20% is excreted unchanged in the urine. (See **Figure 1**.)

Acetone is a ketone *but not a ketoacid*; thus, patients will have a normal pH and anion gap. Acetone is excreted primarily by the kidneys. If present in high enough concentrations, pulmonary excretion can become a significant mode of elimination, and the odor of acetone may be present.

Figure 1. Metabolism Of Isopropyl Alcohol.

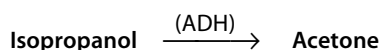


Figure 2. Metabolism Of Methanol.

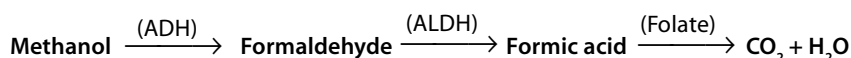


Figure 3. Metabolism Of Ethylene Glycol.



Clinical Signs And Symptoms

Because both isopropanol and acetone cause CNS depression, lethargy is a common presenting complaint. Other CNS effects include headache and ataxia. Gastrointestinal symptoms are prominent, including nausea, vomiting, hematemesis, and abdominal pain. Tachycardia is common, and hypotension may be seen in significant poisoning.

Laboratory Testing

Laboratory findings include acetonemia and acetonuria but not acidosis.¹¹² Both isopropanol and acetone contribute to increase osmolality and produce an osmolal gap. Blood levels of isopropanol do not correlate well with clinical symptoms and signs.¹¹³ Falsely elevated creatinine levels may be found following the ingestion of isopropanol.

Management

Supportive treatment, consisting of IV fluids, oxygen, and serial examinations, is sufficient in the majority of cases. Because isopropanol, like the other alcohols, is rapidly absorbed, charcoal does not have a role unless co-ingestants are suspected.^{91,114} Skin decontamination should be undertaken if there is reason to suspect dermal exposure. In the rare cases of persistent coma or shock refractory to fluid infusion, hemodialysis may be indicated.

Ethylene Glycol And Methanol

Ingestion of methanol or ethylene glycol is fortunately a rare occurrence. These products are widely available and accessible in various forms, including antifreeze, windshield-wiper fluid, and industrial solvents. It can occur as a suicide attempt, as an accidental ingestion (particularly in children), or as a substitution alcohol in an ethanol-dependent person. There are an estimated 5000 cases of ethylene glycol ingestion per year in the United States.¹¹⁵ Whatever the circumstances, such ingestions have a real potential for toxicity and fatality.

Mechanism Of Toxicity

While neither methanol nor ethylene glycol has significant endogenous toxicity, both produce highly toxic products when metabolized by hepatic alcohol dehydrogenase. Methanol is broken down to formaldehyde and formic acid (see **Figure 2**); ethylene glycol is metabolized to oxalate and glycolic acid (see **Figure 3**). These end products have multi-organ toxic effects and produce a characteristic high anion

gap metabolic acidosis.

Methanol is metabolized to formaldehyde by alcohol dehydrogenase (ADH) in a rate-limiting step. Formaldehyde is rapidly converted by aldehyde dehydrogenase (ALDH) to formic acid, the toxic metabolite responsible for most of the clinical effects. Folate is a cofactor for the conversion of formic acid to carbon dioxide and water.

Ethylene glycol is metabolized by alcohol dehydrogenase (ADH) to glycolaldehyde. This is converted to glycolic acid by aldehyde dehydrogenase (ALDH). This toxic metabolite is responsible for many of the clinical manifestations of ethylene glycol ingestion. Glycolic acid is metabolized to another toxic metabolite, oxalic acid, via an intermediate product, glyoxylic acid. The enzymes involved include lactic dehydrogenase (LaDH) and

glycolic acid oxidase (GAO).

Clinical Presentation And Evaluation

There is typically a latent period between ingestion of the toxic alcohol and onset of symptoms. It is during this time that the toxic metabolites are being formed. Particularly in the case of accidental ingestions, asymptomatic patients may present to the ED for evaluation.

Important Aspects Of The History

Many patients are reluctant to admit to the ingestion. Approach the patient in an open and nonjudgmental way and utilize family or friends when necessary. Attempt to establish what product was ingested, how much, and when.

Continued on page 14

Cost-Effective Strategies For Alcohol-Related Emergencies

While formal cost-benefit analysis is frequently lacking, the following strategies should be considered cost-effective. Individual practice should be tailored to the particular ED setting; what may be cost-effective in one department (for example, routine head CTs for all intoxicated patients with head injury) may not be cost-effective in another.

1. Perform a thorough and complete physical examination for all patients suspected to be intoxicated.

Early detection of an intracranial injury or systemic illness will clearly decrease morbidity and mortality and improve outcomes for both the patient and the physician.

2. Determine the blood glucose level immediately.

Albeit rare, hypoglycemia can accompany alcohol intake or can masquerade as an alcohol intoxication, which makes a rapid bedside glucose determination one of the best cost-effective strategies available.

3. Limit the number of blood alcohols drawn.

Not every patient needs a blood alcohol drawn. Perform serial physical examinations and document that the patient is clinically stable and is competent to leave at discharge. Saliva or breath tests are also cost-effective alternatives to standard BALs.

Risk-Management Caveat: Alcohol levels are occasionally helpful, such as when the diagnosis of intoxication is in doubt and when the patient has evidence of mild head trauma and slightly altered mental status. A level of less than 200 mg/dL should not cause a GCS of less than 15.

4. Perform serial examinations for intoxicated patients with evidence of head injury.

Not every intoxicated patient with a facial abrasion needs a CT. Perform serial examinations at regular intervals; documenting GCS scores may be of benefit in the low-risk patient.

Risk-Management Caveat: Serial examinations are an acceptable alternative to CT scanning in low-risk patients if they are actually done. If you cannot ensure serial examinations in your ED, CT scanning may be the most prudent strategy. Patients with clinical evidence of significant head trauma (focal neurologic exam, evidence of skull fracture, low GCS, etc.) require immediate CT scanning.

5. Use lorazepam to treat alcohol withdrawal.

Lorazepam has been clearly shown to decrease the incidence of withdrawal symptoms, which allows many alcohol abusers to be sent home rather than admitted to the hospital. It is especially valuable in those who have withdrawal seizures.

6. Use 4-methylpyrazole for toxic alcohol poisonings.

This drug may be cost-effective when used for the treatment of ethylene glycol or methanol poisoning. This is particularly true when it results in obviating the need for hemodialysis.

7. Hydration in patients with alcohol ketoacidosis can reduce the need for more intensive therapies.

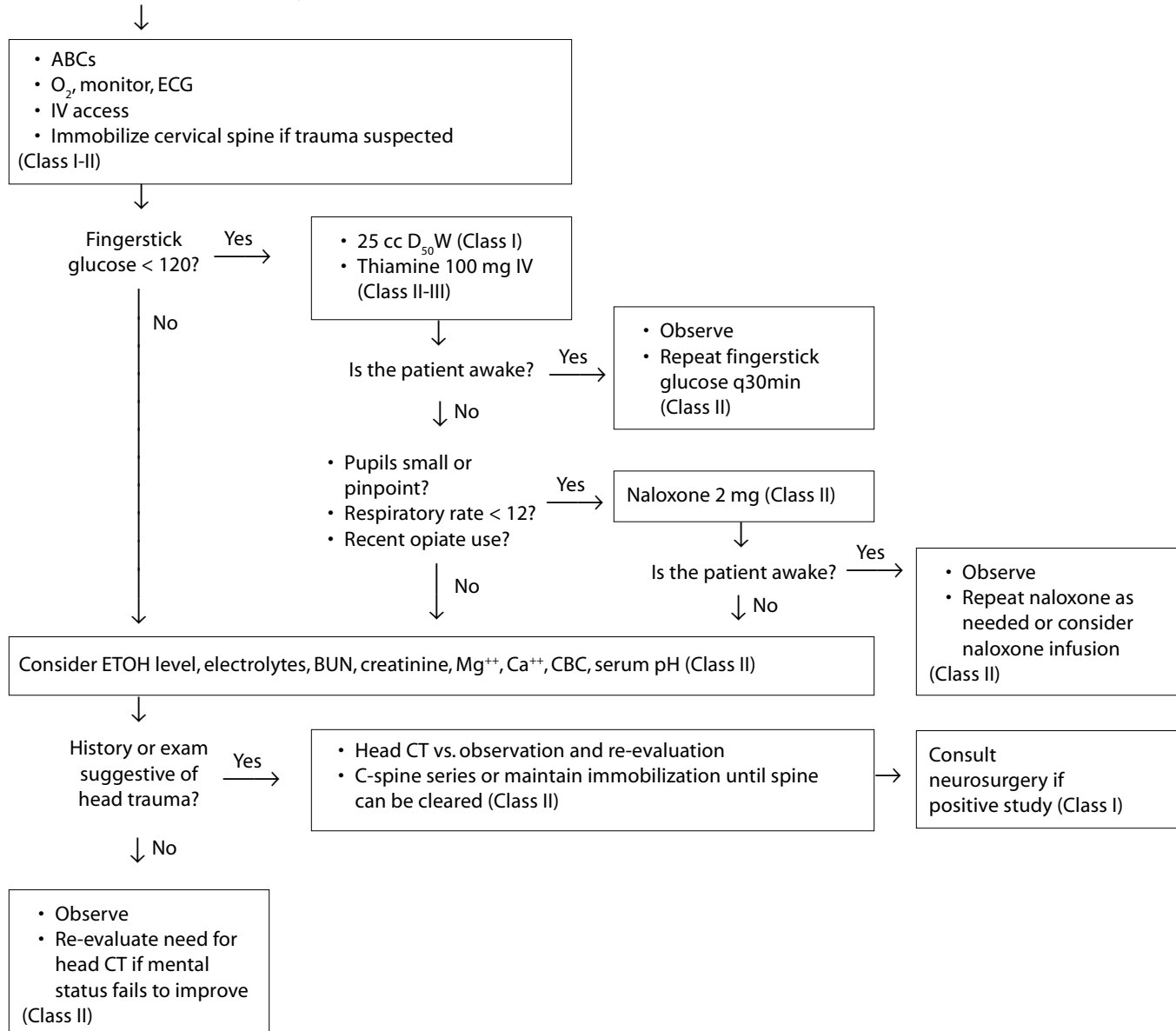
AKA generally responds rapidly to supportive care, which suggests the benefits of aggressive fluid resuscitation with an isotonic dextrose solution. Patients treated with a comprehensive rehydration strategy frequently do not need a hospital admission and can be safely discharged from the ED.

8. Alcohol screening and a "brief intervention" may reduce recidivism.

From a societal point of view, a "brief intervention" when the alcoholic is in the ED may be the most cost-effective strategy available for decreasing dollars spent on alcohol-related disease. These interventions occur when the patient is most vulnerable and maximizes the chances of altering behavior. The five minutes spent talking to the patient, especially when combined with coordinated discharge planning, may be lifesaving. ▲

Clinical Pathway: Managing The Unresponsive Intoxicated Patient

Unresponsive patient with history suggestive of alcohol intoxication

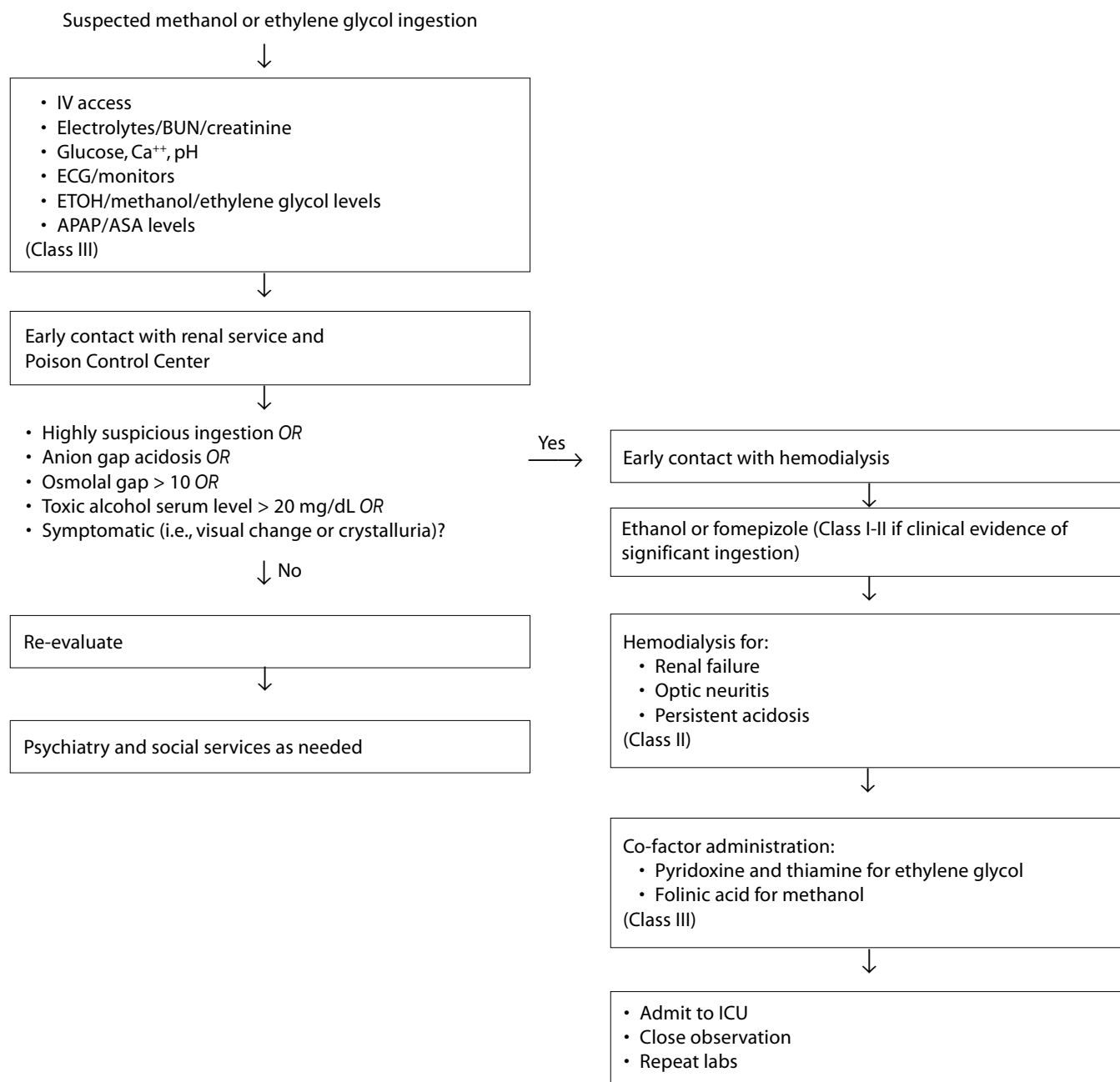


The **evidence for recommendations** is graded using the following scale. For complete definitions, see back page. **Class I:** Definitely recommended. Definitive, excellent evidence provides support. **Class II:** Acceptable and useful. Good evidence provides support. **Class III:** May be acceptable, possibly useful. Fair-to-good evidence provides support. **Indeterminate:** Continuing area of research.

This clinical pathway is intended to supplement, rather than substitute, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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Clinical Pathway: Management Of Toxic Alcohol Ingestion

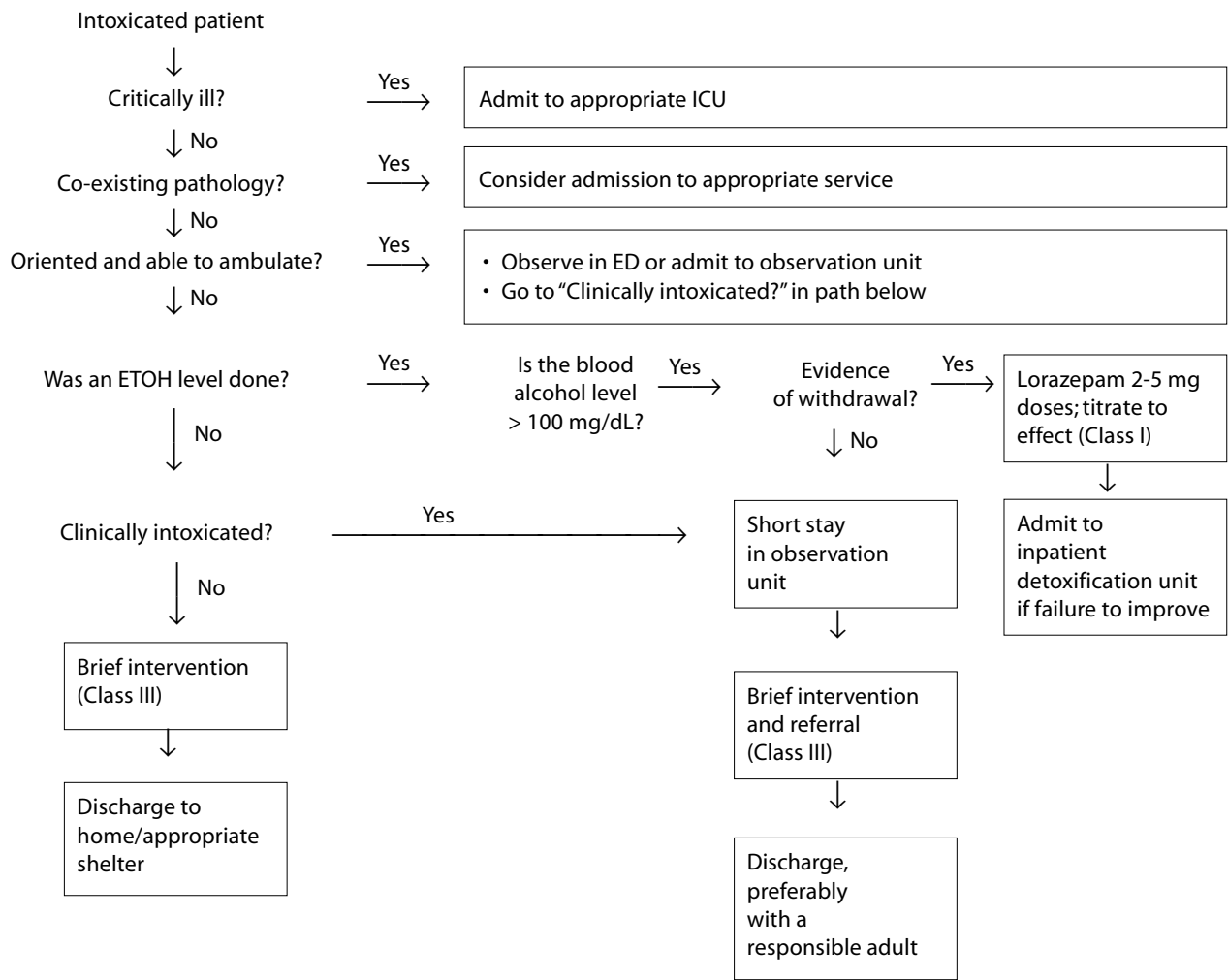


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Clinical Pathway: Alcohol Intoxication Discharge Strategy



The **evidence for recommendations** is graded using the following scale. For complete definitions, see back page. **Class I:** Definitely recommended. Definitive, excellent evidence provides support. **Class II:** Acceptable and useful. Good evidence provides support. **Class III:** May be acceptable, possibly useful. Fair-to-good evidence provides support. **Indeterminate:** Continuing area of research.

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Color, taste, and other descriptions of the product are unreliable and are no substitute for examining the product firsthand. If the container has not been brought to the ED with the patient, send a reliable person to the home or workplace to get it. If the product had been transferred from one container to another, instruct the person to search for the original container that contains the written product information.

Especially in the case of children, inquire about other potential “drinkers.” Children are often unwilling to admit to such ingestions for fear of repercussions from caretakers or peers.

Symptoms And Signs

Methanol: The hallmark of methanol toxicity is visual disturbance and abdominal pain in a patient with a metabolic acidosis. There is typically a latency period of 6-30 hours before symptoms develop. Early findings include confusion, inebriation, and ataxia. As metabolism continues and formic acid accumulates, malaise, severe headache, vomiting, vertigo, abdominal pain, and blurred vision become apparent. Examination often reveals sluggish, dilated pupils with retinal edema and hyperemic discs (i.e., pseudopapilledema). The toxic optic neuropathy may produce permanent blindness.

Ethylene Glycol: The hallmark of ethylene glycol

Table 5. The Three Phases Of Ethylene Glycol Toxicity.*

Phase 1 toxicity: CNS

- Neurological manifestations include nystagmus, hyporeflexia, and tetany from hypocalcemia.

Phase 2 toxicity: Cardio-respiratory

- Cardiovascular signs include episodes of hypertension or hypotension, as well as congestive heart failure with pulmonary edema.

Phase 3 toxicity: Renal

- Renal dysfunction is characteristic and results from direct effects of calcium oxalate crystals on renal tubules and interstitium. Acute tubular necrosis with resultant oliguric renal failure may occur. Significant, symptomatic hypocalcemia with prolonged QTc interval may develop.

*These phases are not always seen sequentially.

Table 6. Useful Formulas In Suspected Toxic Ingestions.

Anion gap = ([Na] – [Cl + HCO₃])

- Normal range = 12 (± 2) mEq/L
- Elevation usually represents unmeasured anions in the serum

Osmolal gap =
(measured osmolality – calculated osmolality)

Calculated osmolality = ([2Na] + [BUN/3] + [glucose/18])

- Normal range = 280 to 300 mOsm/kg H₂O
- Gap ≥ 10 mOsm/kg H₂O suggests exogenous osmoles

toxicity is renal failure in a patient with a metabolic acidosis. The latent period ranges from 1-12 hours. The characteristic symptoms theoretically develop in a triphasic manner, involving the CNS, cardiopulmonary, and renal system. (See **Table 5.**) Initial symptoms include altered mental status with inebriation, nausea, vomiting and hematemesis.

Diagnostic Studies

In patients with suspected toxic alcohol ingestions, draw blood for electrolytes, BUN, creatinine, calcium, pH, ethanol level, and osmolality. Methanol and ethylene glycol levels should also be sent but usually are not readily available. In the absence of an ingestion history, the diagnosis of toxic alcohol toxicity is strongly suggested by the characteristic metabolic disturbances found.

An anion gap acidosis along with an osmolal gap means toxic alcohol poisoning until proven otherwise. (See also **Table 6.**)

*A normal osmolal gap does not exclude the presence of the toxic alcohols.*¹¹⁶⁻¹¹⁸ When the alcohol is fully metabolized, the osmolal gap will return to normal, but the life-threatening acidosis and toxic metabolites will still be present. If an osmolal gap is present and not explained by ethanol (serum ethanol level divided by 4.3), an estimate of the serum methanol or ethylene glycol concentration can be made by multiplying the gap by 2.6 for methanol or 5 for ethylene glycol.¹¹⁹

Urinalysis: In the past, some literature suggested using a Wood’s lamp to detect urine fluorescence following possible ethylene glycol ingestion. However, a more recent trial suggests that this technique be abandoned because of the numerous false-positive results.¹²⁰

A more important intervention is a microscopic urinalysis, looking for crystalluria (monohydrate and dihydrate forms of calcium oxalate). This is frequently seen in ethylene glycol poisoning.¹²¹

Treatment And Intervention

Treatment is initiated based on suspicion and the characteristic metabolic derangement. (See also **Table 7.**) The first step in management of toxicity from methanol and ethylene glycol lies in inhibiting the formation of toxic metabolites.

Table 7. The Fundamentals Of Managing A Toxic Alcohol Ingestion.

General and supportive care:

- IV access, oxygen, cardiac monitor and pulse oximetry
- No value in ipecac, lavage, or charcoal
- Involve the local Poison Control Center and nephrology service early

Labs

- Electrolytes, ABG, calcium, renal function, osmolality, and serum ETOH and other alcohol levels
- Calculate anion and osmolar gap.

Therapy

- Consider sodium bicarbonate infusion if pH is less than 7.3
- Fomepizole or ethanol infusion
- Hemodialysis for severe cases

The two modalities available include ethanol infusion and 4-methylpyrazole (fomepizole). Hemodialysis has a central role when significant toxicity exists.

Ethanol Infusion: Ethanol is a preferential substrate for hepatic alcohol dehydrogenase and competes for the enzyme to prevent metabolism of other toxic alcohols. This blocks the formation of toxic metabolites and allows time for removal of the parent compound by other means (e.g., hemodialysis).¹²²⁻¹²⁴ The accepted target plasma ethanol concentration is 100-125 mg/dL.¹²⁵ An IV loading dose of 10 mL/kg of 10% ethanol followed by a maintenance infusion of 1.5 mL/kg/h will usually maintain therapeutic levels. During hemodialysis, increase the ethanol dose (≈ 3 mL/kg/h) to maintain this therapeutic level. Ethanol infusions should be continued until the serum levels of methanol and ethylene glycol are below 20 mg/dL.

The therapeutic use of ethanol is not without problems. IV preparations are not readily available in some hospitals; plasma ethanol levels need to be monitored frequently to ensure adequate levels; and prolonged use may produce hypoglycemia and hepatotoxicity.

Fomepizole: Recently, fomepizole (4-methylpyrazole) has been approved in the United States as an alternative means of inhibiting hepatic alcohol dehydrogenase (Antizol® by Orphan Medical; supplied as 1g/1mL at cost of \$1150 per 1.5 mL). (See **Table 8**.) It is currently approved for use in ethylene glycol poisoning and appears to be effective and well-tolerated in this setting.^{126,127} Although not yet FDA approved, recent studies show that it is also safe and effective in the treatment of methanol toxicity.¹²³ A loading dose of 15 mg/kg intravenously is administered followed by doses of 10 mg/kg every 12 hours until serum levels fall below 20 mg/kg.

Fomepizole does not promote renal excretion of the parent compound or toxic metabolites; therefore, hemodi-

alysis is still indicated in toxic patients, especially those with methanol or ethylene glycol levels above 50 mg/dL.

Hemodialysis: Hemodialysis is effective in removing the toxic alcohols from the blood and thus inhibiting production of toxic metabolites. In addition to the parent compounds, formaldehyde, formate, oxalate, and glycolate are also cleared during hemodialysis.^{128,129} **Table 9** lists the indications for hemodialysis.^{124,130} Once initiated, hemodialysis is continued until the acidosis is corrected and the levels of the toxic alcohol are 20 mg/dL or less. Peritoneal dialysis can be used but is significantly less effective than hemodialysis.¹³¹

Co-factor Administration: Supplemental thiamine (100 mg a day) and pyridoxine (100 mg a day) should be given as they are co-factors in ethylene glycol metabolism and may be metabolically consumed. Folinic acid (leucovorin, the co-factor involved in formate metabolism) is similarly recommended in cases of methanol poisoning.^{125,132}

Disposition

Patients with methanol or ethylene glycol toxicity require an intensive-care setting in a facility that can provide emergency hemodialysis. (See also **Table 10**.)

Special Populations

*"You can't be a Real Country unless
you have a beer and an airline—
it helps if you have some kind of a football team,
or some nuclear weapons,
but at the very least you need a beer."
—Frank Zappa (1940–1993), U.S. rock musician¹⁶⁸*

The Drunk Teenager

Underage drinking is a common occurrence. Many trauma-related visits, including fatalities, in this age group are directly related to alcohol use. A 1997 study at the Children's Hospital of Wisconsin found that 39% of injured adolescents seen during the eight-month study period were "alcohol-positive."¹³³ Equally alarming are the findings of Johnston and colleagues in their survey of drinking habits among high-school children; 25% of eighth-graders and 50% of 12th-graders reported alcohol use in the preceding month, and 15% of eighth-graders and 28% of 12th-graders

Table 8. Fomepizole Use In The ED.

Advantages

- Safe
- Effective
- Non-sedating (it does not produce changes in mental status)
- Therapeutic plasma levels are reliably achieved

Limitations

- Expensive
- Not widely available
- Ability to monitor levels limited in many laboratories
- Not yet approved for use in methanol poisoning

Table 9. Indications For Hemodialysis In Methanol Poisoning.

- Renal failure: serum creatinine > 3
- Visual impairment following methanol ingestion
- Persistent acidosis, pH < 7.3, despite HCO₃ and ethanol or fomepizole use
- Toxic alcohol plasma levels of ≥ 20 -50 mg/dL

Table 10. Pitfalls In The Management Of Toxic Alcohol Ingestions.

- Failing to identify potential victims, especially children
- Mistaking the latent phase of toxicity for a minor, nontoxic ingestion
- Waiting for methanol or ethylene glycol levels to initiate therapy
- Failing to recognize hypoglycemia
- Forgetting to increase the ethanol infusion during hemodialysis
- Failing to recognize that metabolized methanol or ethylene glycol can result in a non-osmolal gap metabolic acidosis

reported binge drinking (5 or more drinks on one occasion) in the preceding two weeks.¹³⁴

Although many of these patients are still “minors,” treatment for urgent and emergency problems should be initiated in the absence of parental consent. Attempts should be made to contact the parents or legal guardians as soon as possible. Management of the intoxicated teenager is similar to that outlined for the intoxicated adult patient. A few different aspects of care in this setting are worth mentioning:

- The intoxicating effects of alcohol may be more pronounced in younger drinkers, and hypoglycemia more common.
- Metabolism of ethanol may be slower, necessitating longer observation periods.
- Use of additional recreational drugs needs to

be considered.

- The parents or legal guardians will need to be located before discharge. If this is not an isolated occurrence, referral for appropriate intervention should be offered.

The Elderly

One of the most common pitfalls regarding alcoholism in the elderly is failure to recognize it. Some elderly may be particularly adept at hiding their alcohol use from physicians and family. The symptom patterns and presentations vary from their younger counterpart.^{135,136} An ED-based study found the prevalence of lifetime alcohol abuse to be 24% and the prevalence of current alcohol abuse was found to be 14%. The CAGE and MAST questionnaires have been shown to be useful for screening elderly populations.^{135,137}

Ten Excuses That Don't Work In Court

1. “He was a frequent flier.”

Physicians may develop a “false sense of security” when dealing with patients who frequent the ED on a regular basis. A prior visit to the ED does not immunize the patient against future disease. Each visit must be considered just that—a “new” visit—rather than “just another” visit.

2. “She never said she had neck pain.”

The combination of intoxication and trauma is a risk factor for occult cervical spine injury. Physicians must have a low threshold for suspecting injuries in this group of patients and take the necessary steps to detect and manage them.

3. “He said he was cold, so we didn’t undress him.”

Medical staff are often reluctant to undress intoxicated patients for full assessment. Many are uncooperative, while others may have wet or soiled clothing. Important findings will be overlooked and injuries will certainly be missed in intoxicated patients unless the patient is fully undressed and examined.

4. “She doesn’t have diabetes, so we didn’t check a glucose.”

Hypoglycemia may coexist with alcohol intoxication. Unless this is specifically sought, it will be missed. Untreated hypoglycemia can result in permanent neurological disability.

5. “He seemed like he just needed to sleep it off.”

Unfortunately, in this patient’s case, he never woke up. The subdural hematoma continued to expand until the patient herniated. Patients who appear intoxicated require a full evaluation with frequent reassessments.

6. “She didn’t look like a drinker.”

Alcohol abuse knows no boundaries. To believe that one can recognize an “alcoholic” by how a person “looks” is

naïve and potentially dangerous.

7. “He only had some black eyes. I never do a CT for black eyes.”

This gentleman was not punched in the eyes—he fell and had a basilar skull fracture (with an associated subdural). Be liberal in scanning the intoxicated patient with head trauma. Indications for prompt head CT include severe mechanism of injury and altered mental status, focal neurologic examination, evidence of a skull fracture, a low GCS, or a GCS inappropriately low compared to the blood alcohol level. In other patients, serial examinations may be appropriate if the ED has the resources and commitment to provide these examinations. If you cannot provide regular examinations, get a head CT!

8. “She didn’t need a neuro exam because she didn’t have any neurological complaints.”

“Impairment” while intoxicated by alcohol is a neurological complaint! Specific neurological complications ranging from seizures to subdural hematomas can occur from alcohol abuse. All intoxicated patients require a focused neurological examination to identify coexisting abnormalities or complications of alcohol abuse.

9. “The nurse told me he was ready to leave.”

Before the patient is discharged, reassessment is indicated. The decision of when “he is ready to go” is the responsibility of the treating physician.

10. “I didn’t know she was going to drive home.”

When a patient is “ready for discharge” from the ED, it should be stated clearly to the patient that he or she should not drive. This advice should also be noted in the written discharge instructions. ▲

The problem is more common among elderly men than women.¹³⁸ Alcohol abuse among the elderly is associated with cognitive impairment and, independently, with short-term mortality.¹³⁸

Pregnancy

Recent literature suggests that approximately 20% of pregnant women drink alcohol.¹³⁹ The American Academy of Pediatrics recommends “total abstinence from alcohol in pregnancy or for women who are planning a pregnancy.”¹⁴⁰ Ethanol readily crosses the placenta and is distributed to all fetal tissue. The multiple detrimental effects of prenatal alcohol exposure on the developing fetus have been recognized since the 1960s.¹⁴¹ The extreme effect of alcohol on the fetus is the Fetal Alcohol Syndrome (FAS), which is characterized by the triad of CNS defects (mental retardation), dysmorphic features (particularly craniofacial), and growth retardation.¹⁴² Even in the absence of the classic features of FAS, children exposed to chronic alcohol intake in pregnancy appear to be at a higher risk of attention deficit disorders, speech disorders, and delayed development or impairment of fine motor skills.¹⁴³

Although there is a legal requirement for healthcare workers to report suspicions of child abuse to authorities at a state level, this does not apply to an intoxicated expectant mother. When caring for patients under these circumstances in the ED, the emergency physician should offer brief counseling and education and refer the patient to appropriate services for follow-up.

Alcohol And The Driver

Blood alcohol testing for the presumptively intoxicated driver remains a contentious issue, and laws vary considerably from state to state. One recurring theme is that drunk drivers who are transported to an ED are rarely prosecuted. A retrospective review of the trauma registry at a major Midwestern trauma center revealed that fewer than one-third of legally intoxicated drivers involved in a motor vehicle crash (MVC) were charged with driving while impaired; the conviction rate was lower still.¹⁴⁴ A recent prospective study found that only 22% of intoxicated drivers involved in MVCs were charged with driving while intoxicated, even under conditions considered ideal by the authors (police at scene, inebriation of driver clinically evident to out-of-hospital personnel, confirmatory ethanol level ≥ 100 mg/dL).¹⁴⁵ While levels obtained from blood routinely drawn for medical evaluation are sometimes not admissible as evidence, this varies considerably by state.

Reporting requirements also differ dramatically. While some states have legally sanctioned physicians who reported drivers who were driving while intoxicated, other states permit emergency physicians to contact police regarding intoxicated patients involved in an MVC. That is, the emergency physician can breach the usual standards of patient confidentiality in order to protect society.

In many states, there is an implied consent for chemical testing unless the driver expressly refuses the test. Refusal usually results in automatic suspension of the driver's license. What happens when a patient refuses to submit to

testing depends on local state law. Refusal to consent should be well-documented in the medical record. In many states, legal blood alcohol levels should *not* be drawn against a patient's wishes without a court order. However, in other states, blood may be drawn against the patient's will if the officer signs a document certifying that there is probable cause for obtaining the blood sample and that obtaining a search warrant would cause unreasonable delay. Such a certificate is usually employed when a third party is killed or seriously injured in an accident involving alcohol.

Skin preparation may be an issue as well. In the past, some defense lawyers have argued that use of isopropanol skin antiseptics falsely elevated the blood alcohol level. (“If isopropyl was it, then you must acquit!”) Although experiments have shown this claim to be false,¹⁴⁶ iodine-based disinfectants (not tincture) are recommended to avoid confusion in court. A well-defined ED protocol will help to clarify the relationship between the legal and the medical system, but such a protocol must be consistent with local law.

The “Impaired Physician”

Physicians, like any other members of society, may become dependent on alcohol. If faced with the difficult situation of an impaired colleague and friend, one needs to take the necessary—though difficult—step of getting appropriate help.

The American College of Emergency Physicians (ACEP) policy statement on the “Impaired Physician” (policy number 400116) states that physician impairment exists “when a physician's professional performance is adversely affected because of illness, including mental or physical, aging, alcoholism, or chemical dependence.” The American Medical Association (AMA) is clear about the ethical responsibilities of physicians who are aware of colleagues with such problems; Policy 275.952 (*AMA Policy Compendium*) states: “Physicians have an ethical obligation to report impaired, incompetent, or unethical colleagues. Physicians should be familiar with the reporting requirements of their own state and comply accordingly.” Both ACEP and the AMA express support for physicians who are impaired, and both organizations support the return of the physician, without bias, once appropriate interventions have proven successful.

Alcohol Abuse Screening And Brief Interventions

Several standardized questionnaires have been developed to help healthcare workers identify individuals at high risk for problem drinking and alcohol dependence.¹⁴⁷⁻¹⁴⁹ These simple screening tools have been shown to be effective and practical^{150,151} and require little time to use in the ED.

CAGE

The CAGE questionnaire is a rapid screening tool for identification of problem drinking¹⁴⁸ and is recommended by the Society for Academic Emergency Medicine (SAEM) Substance Abuse Task Force.¹⁵² CAGE is a mnemonic for the

four questions involved. (See **Table 11.**)

When two or more questions are answered “yes,” the sensitivity of the CAGE instrument for alcohol dependence is 76%, with a specificity of 90%.¹⁵³

MAST And AUDIT

The Michigan Alcoholism Screening Test (MAST) questionnaire, although effective, is substantially longer than the CAGE. A modified short-form MAST (B-MAST) consists of 10 questions.¹⁴⁷ Although the Alcohol Use Disorder Identification Test (AUDIT) was not developed for use in the ED,^{149,154} it has been used with success in this setting.^{153,155} In one large study, comparison of these screening tools showed that the sensitivity of CAGE and B-MAST for alcohol dependence varied among ethnic groups, and all of the instruments were less sensitive in women than men.¹⁵⁶

TWEAK

The CAGE screening questionnaire is considered less reliable in women than in men, and its sensitivity in pregnancy has been questioned. As a result, the “TWEAK” questionnaire was developed and has proven helpful to identify “at risk” pregnant drinkers.¹⁵⁷ (See **Table 12.**)

TWEAK has a sensitivity of 87% for harmful drinking, with a specificity of 86%.¹⁵³ There is evidence to suggest that a brief intervention for prenatal alcohol use can be effective,^{139,158} and referral to appropriate treatment and support systems should be offered to the patient.

Brief Intervention In The ED

Although screening allows identification of people with drinking problems, screening serves little purpose without intervention. While most practitioners agree that referral to treatment programs can be of value for properly motivated people, many physicians are unaware or underestimate the value of giving advice to the patient at the time of the ED visit. A 1996 multi-national World Health Organization study showed that five minutes of “simple advice” was as effective as 20 minutes of brief counseling.¹⁵⁹ Nine months later, male patients advised to stop drinking

Table 11. The CAGE Questionnaire.

• Have you ever felt the need to C ut down on your drinking?
• Have you ever felt A nnoyed by criticism of your drinking?
• Have you ever felt G uilty about your drinking?
• Have you ever felt the need to drink a morning E ye-opener?

Table 12. The TWEAK Questionnaire.

• Can you hold six or more drinks? (T olerance)
• Are your friends or relatives W orried about your drinking?
• Have you ever had an E ye-opener (morning drinking to get going)?
• Have you ever had blackouts (A mnnesia)?
• Have you ever felt the need to “ K ut” down on your drinking?

reported approximately 17% lower average daily alcohol consumption than those in the control group (who had no alcohol counseling).

The immediate aim of the brief intervention is to facilitate recognition by the patient that a problem exists and to motivate (not force or persuade) the patient to take the next step: a change in behavior. To have any chance of success, targets need to be set by the patient, not the physician.

Disposition

Critically ill patients—whether from trauma or severe manifestations of associated medical conditions—require admission to an appropriate intensive care unit. Patients otherwise too ill to be discharged from the ED, with a reasonably clear mental status, and with well-defined diagnoses may be admitted to non-monitored, non-intensive care settings.

Heavily intoxicated patients who have no associated acute medical problems frequent many EDs. Alcohol levels of 400-500 mg/dL and higher are not uncommon in chronically dependent patients; patients may not be able to safely ambulate on their own for eight or more hours. Hospital admission for these patients may be of limited medical value and is not cost-effective. Ideally, patients who fall into this category should be cared for in observation units or hospital “short-stay” units. Inpatient admission to an alcohol detoxification unit, for properly motivated patients, may be of benefit and financially justifiable.¹⁶⁰

All intoxicated patients who are discharged should be advised to not drink and drive, and this advice should be documented in the patient record. It is also preferred, but rarely practical, to discharge them in the care of a non-intoxicated adult. Offering all patients detox services (and documenting any refusals) is a nice touch. Providing contact numbers for treatment centers may be useful.

Blood Alcohol Levels And Discharge

Patients who arrived intoxicated, *did not* have a blood alcohol level determination, and are clinically sober can be discharged, after documenting that they are safe to leave the ED. The disposition of intoxicated patients who are clinically sober but who had a blood alcohol determination performed is slightly more complex. Although the patient’s clinical condition is more relevant than the alcohol level, the legal systems in all states view patients with a BAL above 80-100 mg/dL as impaired. On the other hand, allowing these patients’ BALs to drop to 100 mg/dL or lower almost guarantees that they will go into withdrawal. It could be argued that should a patient with an elevated BAL be involved in an accident outside the ED, the patient was discharged prematurely and the physician may be liable for damages. However, a recent review failed to reveal any cases in which a physician or hospital was sued under such circumstances.¹⁶¹

In general, when a patient has an initial blood ethanol level above 100 mg/dL, it is critical to document on discharge that the patient is “clinically sober.” This may

involve an assessment of mental status, speech, gait, and coordination. It is *not* necessary to document a second BAL lower than 100 mg/dL prior to discharge. Patients who are not clinically sober cannot be allowed to sign out of the ED "against medical advice."

One interesting study surveyed plaintiff attorneys regarding how serum alcohol determinations influence malpractice suits. Overall, 63.9% of the attorneys surveyed would advise patients to sue if they were involved in a traffic crash after ED evaluation for intoxication. Over 40% of attorneys would advise clients that they received potentially negligent care when impairment was documented by a serum alcohol concentration and no advice was given regarding drunk driving. Fewer than 20% advised a suit when impairment was not documented by a test for serum alcohol concentration and no advice was given regarding drunk driving. Only 3.5% of attorneys would suggest negligence when impairment was documented by a test for serum alcohol concentration *and* advice was given not to drive.¹⁶² (This study seems to show that some plaintiff attorneys will sue no matter *what* the physician does.)

Summary

Alcohol-related problems have been ailing mankind and challenging physicians since time immemorial. As with many other debilitating and chronic illnesses, medical progress has been slow. Nevertheless, our understanding of alcohol and its effects on our patients continues to improve. Emergency medicine, in particular, has contributed to better therapies for the toxic alcohols, improved strategies in screening and brief intervention, as well as improved management of alcohol withdrawal.

Several studies suggest that brief intervention by a physician is effective in reduction in alcohol use in problem drinkers.^{159,163,164} Such interventions are also cost-effective.¹⁶⁵ Although these larger studies were conducted in the primary care setting, there is literature to suggest that a single, brief intervention in the ED is worthwhile.^{152,166,167}

Alcohol intoxication, while easy to diagnose clinically and easy to confirm with the use of the laboratory, is often accompanied by more subtle but potentially life-threatening conditions. Just because the patient smells of alcohol does not mean that he or she is not seriously ill. The emergency physician must maintain vigilance for co-existing pathology. ▲

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Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited

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(Prospective, randomized, controlled; 723 patients)

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Physician CME Questions

33. All of the following are routinely indicated in the intoxicated patient with altered mental status *except*:
- thiamine 100 mg IV.
 - monitoring the glucose level.
 - naloxone 0.2 mg IV.
 - frequent neurological exams.
34. Blood alcohol levels should be measured in which of the following situations?
- Routinely, to assist in discharge planning
 - If indicated clinically or if the diagnosis is uncertain
 - At the request of a law enforcement officer, even if the patient refuses the test
 - If "alcohol on breath" has been documented in the triage assessment
35. Alcohol withdrawal seizures are best managed by which of the following?
- Phenobarbital
 - Lorazepam
 - Phenytoin
 - Naltrexone
36. Which of the following toxic alcohols is associated with calcium oxalate crystals in the urine?
- Isopropanol
 - Ethylene glycol
 - Ethanol
 - Methanol
37. Ocular toxicity is characteristic of which of the following ingestions?
- Isopropanol
 - Methanol
 - Ethylene glycol
 - Isopropyl alcohol
38. Which of the following is the first to improve in Wernicke's encephalopathy following thiamine administration?
- Unsteady gait
 - Memory impairment
 - Altered mental status
 - Oculomotor findings

39. Which of the following is *not* seen with isopropanol ingestion?

- Metabolic acidosis
- Osmolal gap
- Ketonemia
- Ketonuria

40. Which of the following properties makes 4-methylpyrazole useful in treating methanol and ethylene glycol toxicity?

- It binds to the parent compound, rendering it non-toxic.
- It binds to the toxic metabolites, rendering them non-toxic.
- It binds to alcohol dehydrogenase, preventing metabolism of the parent compound.
- It increases renal excretion of the alcohols.

41. Which of the following is the cornerstone of alcoholic ketoacidosis treatment?

- Bicarbonate infusion
- Administration of insulin
- Dextrose in water infusion
- Normal saline with dextrose infusion

42. Which of the following is *not* typically seen in Wernicke's encephalopathy?

- Cerebellar disturbance
- Seizures
- Oculomotor disturbance
- Confusional state

43. All of the following are true about disulfiram *except*:

- It causes irreversible inhibition of aldehyde dehydrogenase.
- Symptoms occur in 15-20 minutes following alcohol ingestion.
- Tolerance occurs with prolonged use.
- Disulfiram does not affect the rate of alcohol elimination from the body.

44. Which of the following conditions account for the most ambulance runs per patient?

- Seizure disorder
- Acute alcohol intoxication
- Respiratory conditions
- Pregnancy-related emergencies

45. A patient with a blood alcohol (ethanol) level of 180 mg/dL may be:

- alert, oriented, and ambulatory.
- confused.
- unresponsive.
- any of the above.

46. Which of the following definitively excludes the diagnosis of alcoholic ketoacidosis?

- Normal glucose level
- Negative nitroprusside test for ketones
- Alcohol (ethanol) level consistent with intoxication
- None of the above

47. **The intoxicated driver involved in a motor vehicle crash:**
- is automatically charged with DWI.
 - is charged with DWI in the majority of cases.
 - is charged with DWI in fewer than half of all cases.
 - will be charged with DWI if a police report is filed by the emergency physician.
48. **Which of the following is true concerning the treatment of Wernicke's encephalopathy?**
- Thiamine should only be given after dextrose has been administered.
 - Dextrose should only be given after thiamine has been administered.
 - IM administration of thiamine is contraindicated due to poor absorption.
 - IV administration of thiamine is the preferred route of administration.

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives an alpha-numerical score based on the following definitions.

Class I

- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:

- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II

- Safe, acceptable
- Probably useful

Level of Evidence:

- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case-control studies
- Less robust RCTs
- Results consistently positive

Class III

- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:

- Generally lower or intermediate levels of evidence

- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate

- Continuing area of research
- No recommendations until further research

Level of Evidence:

- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

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