#### **Acute Alcohol Intoxication**

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When the learner has completed this self-study module, he/she will be able to:

- 1. Identify two substances other than alcoholic beverages that contain ethanol.
- 2. Identify two substances that contain dangerous alcohols and may be abused.
- 3. Identify the most common clinical effect of acute alcohol intoxication.
- 4. Identify three other signs and symptoms of acute alcohol intoxication.
- 5. Explain the most common cause of death from acute alcohol intoxication.
- 6. Identify the mechanism by which alcohol causes intoxication.
- 7. Identify the serum level of ethanol that defines intoxication.
- 8. Identify the average hourly rate of ethanol metabolism.
- 9. Identify the most important aspect of care of treating a patient with acute alcohol intoxication.
- 10. Identify three aspects of care that are often overlooked when caring for a patient with acute alcohol intoxication.

# What is Alcohol?

Alcohol is the common name for *ethyl alcohol* – commonly called *ethanol* – and ethyl alcohol is by far the most popular intoxicant in the United States. Ethyl alcohol is found in beverages such as beer, wine, and spirits like whisky, gin, etc. Ethyl alcohol is also found in high concentrations in hair sprays, perfumes and colognes, mouthwashes, food flavorings such as vanilla extract, and liquid hand sanitizers. *Note: In this module, the terms alcohol, ethyl alcohol, and ethanol will be used interchangeably.* 

**Key point:** Alcohols are common chemical compounds that are composed of a hydrocarbon and an attached hydroxyl group. Ethyl alcohol is the most familiar, but there are other alcohols that can be ingested and abused: **isopropyl alcohol** (rubbing alcohol), **methyl alcohol** (used in automobile windshield washer/deicer) and ethylene glycol (used as antifreeze in automobile radiators). *These are far more dangerous than ethyl alcohol and chronic alcoholics do ingest them.* 

The alcohol content of alcoholic beverages ranges from 4% - 6% for beer, 10% - 20% for wine, and 30% - 50% for spirits. Alcoholic beverages are also identified by the term *proof*, and proof is simply double the ethyl alcohol content of the beverage, e.g., 100 proof means 50% alcohol.

#### What are the Clinical Effects of Acute Alcohol Intoxication?

The most common clinical effect of acute alcohol is **central nervous system depression.** This can range from mild drowsiness to coma. Ataxia, dizziness, lack of coordination, and confusion are often seen. Nausea and vomiting are very common, respiratory depression is possible, and some patients may develop tachycardia and hypotension. Protective reflexes (e.g., the gag reflex) may be absent, and hypoglycemia, hypothermia, and rhabdomyolysis can develop if the intoxicated person is unconscious for a long period of time. **Death from ethanol intoxication is caused by respiratory depression and aspiration of stomach contents.** 

**Key Point:** Alcohol also causes a phenomenon called *holiday heart*. Holiday heart occurs when someone who has a healthy heart ingests a lot of alcohol in a short period of time (e.g., at a Christmas party). Some of these people will develop arrhythmias, and atrial fibrillation is the most common. The arrhythmia most often resolves spontaneously and no treatment is needed.

# How Does Alcohol Cause Intoxication?

The toxic effects of alcohol are probably caused by its interaction with certain neurotransmitters and/or their receptors:

- a) An interaction between ethyl alcohol and the neurotransmitter  $\gamma$ -aminobutryic acid (GABA)
- b) An interaction between ethyl alcohol and a specific receptor site for the neurotransmitters **glutamate** and **glycine**.

GABA is one of the two major *inhibitory* neurotransmitters. When GABA binds to GABA receptor sites on cell membranes, the cell becomes hyperpolarized and is unable to respond to stimulation – it is inhibited. Ethyl alcohol binds to a specific site associated with the GABA receptor and this increases the activity of GABA when it binds to the GABA receptor. When this occurs, the cells become less able to respond to stimuli and they become less active – the result is the CNS

depression, respiratory depression, diminished reflexes, etc. associated with alcohol intoxication.

Glutamate is one of the major excitatory neurotransmitters. When glutamate binds to receptors on cell membranes, the cells depolarize and act or respond. Glycine is a inhibitory neurotransmitter and works the same way as GABA. Both glutamate and glycine bind to a specific receptor site, the N-methyl-D-aspartate (NMDA) receptor. Ethyl alcohol interferes with the binding of glutamate and glycine to the NMDA receptor, so

the cell cannot respond to stimuli.



According to the Centers for Disease Control (CDC), more than 50% of the population drinks alcohol, there are more than 4 million visits to emergency rooms each year that are related to alcohol use, approximately 40% of automobile accident-related deaths involve alcohol intoxication, and excessive alcohol use is the 3<sup>rd</sup> leading lifestyle related cause of death. Acute alcohol intoxication is directly responsible for a large majority of assaults, sexual assaults, intimate partner violence, and cases of child abuse.

#### How is Alcohol Metabolized?

Ethanol is rapidly absorbed within 60 minutes. If there is food in the stomach, the alcohol is consumed slowly, or there is a high concentration of ethanol in the drink, absorption can take longer. Approximately 20% of an ingested amount is absorbed in the stomach and the examining 80% is absorbed by the small bowel.

Once ethanol is absorbed, it metabolized in the liver by an enzyme called **alcohol dehydrogenase** (ADH) to **acetaldehyde**. Acetaldehyde is metabolized by acetaldehyde dehydrogenase to **acetate** and **water.** 

**Key Point:** There is also ADH in the stomach and women have a lower concentration of gastric ADH. This is one reason why, given an equal amount of ethanol, most women will become more intoxicated than most men: a larger amount of ethanol reaches the brain instead of being broken down in the gut.

**Key Point:** Approximately 40% of all Asians and 80% of all Native Americans have a low level of acetaldehyde dehydrogenase. When these people drink, they get high levels of acetaldehyde and get nauseated, flushed, and diaphoretic.

Most people metabolize ethanol at a rate of about 7 – 10 grams of ethanol an hour (this is slightly less than one can of beer, one glass of wine, or 1.5 ounces of hard liquor) and their blood alcohol level decreases by 12 mg/dL/h.

However, ethanol metabolism can vary significantly from person to person. People who drink large amounts of alcohol chronically may be able to metabolize ethanol at a faster rate than the "recreational" drinker. There are also individual variations in the activity of ADH.

Does The Chronic Drinker Metabolize Alcohol Differently Than The Non-Drinker?

It appears that they do. First, heavy drinkers metabolize alcohol at a faster rate. This happens because the ADH enzyme is more active in these people, and another alcohol metabolizing enzyme system is activated that increases the ability of the body to metabolize alcohol. Second, the chronic drinker develops a *functional tolerance*. As their drinking history gets longer and longer, a given amount of alcohol will cause less impairment. The alcohol-dependent person will need more and more alcohol to become intoxicated.



That depends. The amount of alcohol that will cause intoxication depends on age, gender, the person's normal pattern of drinking, his/her ethnicity, and his/her ability to metabolize alcohol. **The greater the amount ingested, the more intoxicated someone will become, but there are wide variations in individual response to any given dose.** For the *average* adult male who weighs 160 pounds/73 kg, two cans of beer with a 5% ethanol concentration will noticeably affect that person's behavior (he will act "tipsy"), he will not be able to concentrate very well, and his blood ethanol concentration will be approximately 50 mg/dL – 60 mg/dL. Three cans of beer and his blood ethanol concentration could reach the legal limit of 80 mg/dL. Two cans of beer consumed by an *average* adult female who weighs 120 pounds could result in a blood ethanol level of 60 mg/dL – 80 mg/dL.

**Key Point:** One "drink" has traditionally been defined as one 12 ounce serving of beer (5% alcohol), five ounces of wine (approximately 10% - 15% alcohol), and one "shot" of hard liquor ( $1\frac{1}{2}$  ounces, 50% alcohol).

#### What Level of Alcohol in the Blood Represents Intoxication?

This is a difficult question to answer. A level of alcohol in the blood that may cause significant impairment – even loss of consciousness – in one person may be tolerated by another. The level of intoxication following ingestion of an alcoholic beverage will depend on age, gender, the concentration of alcohol in each drink, how rapidly the alcohol is consumed, the presence or absence of food in the stomach, percentage of body fat, how quickly the individual metabolizes the alcohol, and how often and how much that person drinks, and the number of years they have been drinking.

At one time the legal definition of intoxication in terms of blood ethanol concentration varied from state to state, but the national legal limit is now 0.8. This is the level of alcohol that will produce an alteration in consciousness to the degree that the individual has lost the capacity to act with reason and caution.

**Key Point:** There are different ways that blood alcohol levels are reported and this can be confusing. The most common definition of legal intoxication – 0.8 or above – represents a measured serum ethanol level of 80 mg/dL.

For most people, a blood alcohol level ≥ 300 mg/dL will cause loss of consciousness, loss of the protective gag reflex, and serious respiratory depression. However, people who chronically abuse

alcohol can be awake, coherent, and ambulatory at *much* higher blood alcohol levels.

### TAKING CARE OF THE PATIENT WITH ACUTE ALCOHOL INTOXICATION

For most patients the intoxicated state may last up 8 hours. There are no specific treatments that are needed – *the care is symptomatic and supportive* – and the great majority of patients with acute alcohol intoxication will recover completely.

Start by assessing the patient's level of consciousness; assess his/her ability to protect the airway; assess oxygen saturation; and check temperature, pulse, and blood pressure.



Obtain a blood alcohol level, a serum glucose, serum electrolytes, and a 12-lead electrocardiogram. Gastric lavage would not be useful and ethanol does not bind to activated charcoal. Once the assessment has been made and the test obtained, focus on these aspects of care: ☐ **Airway protection/oxygenation:** Ethanol causes respiratory depression and loss of the protective gag reflex. These patients are at risk for hypoxemia and aspiration. Some will need endotracheal intubation and mechanical ventilation. Airway protection and oxygenation are the most important aspects of care for a patient with acute alcohol intoxication. ☐ **Watch the blood sugar:** People with acute ethanol intoxication cannot take in calories and they often have not been eating during a binge. If they are unconsciousness for a long period of time due to alcohol intoxication, liver stores of glycogen can get depleted, gluconeogenesis (synthesis of glucose from fats and proteins) is inhibited, and the patient can suffer severe hypoglycemia; this is often overlooked or forgotten. ☐ **Rhabdomyolysis:** If the patient is very intoxicated and has been unable to move for a long period of time, rhabdomyolysis is possible. ☐ Fluid replacement: Alcohol can act as a mild diuretic, it an cause hypotension, and the intoxicated patient cannot take oral fluids, so IV fluids are often needed ☐ **Trauma:** The patient may present with acute ethanol intoxication, but accidents and injuries are common in these situations. Examine the patient carefully for signs of trauma. This is often overlooked. ■ Other drugs/other alcohols: The story may be that the patient was drinking - but it is also possible he/she was using an illicit drug or abusing a prescription drug, or drinking something with ethylene glycol, methanol, etc. Take a careful history and obtain the appropriate laboratory tests to detect the presence of these substances; this is often overlooked or forgotten. ☐ **Alcoholic ketoacidosis:** Acute metabolic acidosis seen in binge drinkers with poor nutrition.

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