PATHOPHYSIOLOGY AND TREATMENT OF ALCOHOL WITHDRAWAL SYNDROME: A REVIEW

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Dana Bartlett is a professional nurse and author. His clinical experience includes 16 years of ICU and ER experience and over 20 years of as a poison control center information specialist. Dana has published numerous CE and journal articles, written NCLEX material and textbook chapters, and done editing and reviewing for publishers such as Elsevier, Lippincott, and Thieme. He has written widely about toxicology and was recently named a contributing editor, toxicology section, for Critical Care Nurse journal. He is currently employed at the Connecticut Poison Control Center and is actively involved in lecturing and mentoring nurses, emergency medical residents and pharmacy students.

ABSTRACT

Alcohol withdrawal can be mild and self-limiting but patients can also suffer serious complications and death. Providing care for a patient who is going through alcohol withdrawal is very challenging. Health clinicians with specialized knowledge, including a basic understanding of the pathophysiology of the syndrome, and an ability to make judicious decisions about medication administration are best able to support patients experiencing alcohol withdrawal. Clinicians knowledgeable about alcohol use and withdrawal need to show flexibility to change their approach to patient care as the clinical condition evolves.
Policy Statement
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Credit Designation
This educational activity is credited for 3 hours. Nurses may only claim credit commensurate with the credit awarded for completion of this course activity. Pharmacology content is 0.5 hours (30 minutes).

Statement of Learning Need
An ongoing learning need exists for health clinicians to provide care for patients who have alcohol withdrawal syndrome. Specifically, clinicians need to understand the pathophysiology and signs and symptoms of alcohol withdrawal, and the available treatment options. Importantly, clinicians need to be informed about the research on alcohol withdrawal, which is continuously evolving to support best practice diagnosis and treatment for improved outcomes.

Course Purpose
To provide health clinicians with knowledge of alcohol withdrawal syndrome recognition, severity level, treatment and recovery.
Target Audience

Advanced Practice Registered Nurses and Registered Nurses

(Interdisciplinary Health Team Members, including Vocational Nurses and Medical Assistants may obtain a Certificate of Completion)

Course Author & Planning Team Conflict of Interest Disclosures

Dana Bartlett, BSN, MSN, MA, CSPI, William S. Cook, PhD,
Douglas Lawrence, MA, Susan DePasquale, MSN, FPMHNP-BC – all have no disclosures

Acknowledgement of Commercial Support

There is no commercial support for this course.

Please take time to complete a self-assessment of knowledge, on page 4, sample questions before reading the article.

Opportunity to complete a self-assessment of knowledge learned will be provided at the end of the course.
1. **Alcohol withdrawal is caused by**
   a. sudden stopping or drastic reduction of drinking.
   b. long-term alcohol use.
   c. having more than five drinks a day.
   d. stopping drinking.

2. **The neurotransmitters primarily involved in alcohol withdrawal are**
   a. acetylcholine and dopamine.
   b. epinephrine and serotonin.
   c. GABA and glutamate.
   d. norepinephrine and adenosine.

3. **The onset of alcohol withdrawal usually begins within**
   a. 24 hours.
   b. 72 hours.
   c. 1 hour.
   d. 8 hours.

4. **True or False: Alcohol withdrawal can occur if the patient has a measurable ethanol level.**
   a. True
   b. False

5. **The risk of developing alcohol withdrawal increases with**
   a. delirium tremens (DTs).
   b. the amount and frequency of drinking.
   c. cognitive changes.
   d. long-term drinking and female gender.
Introduction

Alcohol withdrawal is a syndrome caused by a sudden cessation of alcohol intake or sudden reduction of alcohol intake in people who are chronic, excessive users of alcohol. The clinical presentation of alcohol withdrawal can be, and often is mild and often is, but serious complications and death are certainly possible. Providing care for a patient who is going through alcohol withdrawal is very challenging. It requires clinicians to have a basic understanding of the pathophysiology of the syndrome, the ability to make judicious decisions about medication administration, and the flexibility to change the approach to patient care as the clinical condition evolves. Alcohol is the term that is commonly used to refer to ethanol, which is the intoxicating component of alcoholic beverages. Other alcohols include ethylene glycol, isopropyl alcohol, and methanol.

Pathophysiology Of Alcohol Withdrawal

Chronic excessive use of alcohol disrupts the balance of activity of the neurotransmitters gamma-aminobutyric acid (GABA) and glutamate. Because of the importance of these neurotransmitters to the pathophysiology of alcohol withdrawal they are discussed here in detail. Additionally, in the following sections the terms alcohol and ethanol will be used synonymously.

Gamma-aminobutyric Acid

Gamma-aminobutyric acid (GABA) is one of the primary inhibitory neurotransmitter in the central nervous system. The binding of GABA to GABA receptors increases the flow of chloride ions into the cell,
hyperpolarizing the membrane and decreasing a cell’s responsiveness to stimulation.

Gamma aminobutyric acid receptor complexes have binding sites for GABA but also for drugs such as barbiturates and benzodiazepines and possibly for alcohol, as well. The interaction between GABA, GABA receptors, and alcohol is not completely understood. Acutely, alcohol increases the activity and transmission of GABA, enhancing its inhibitory effect and decreasing central nervous system activity and causing the well-known effects of alcohol intoxication such as decreased coordination and drowsiness. Chronic alcohol use decreases the sensitivity of GABA receptors to GABA, so more and more alcohol is required to achieve the same level of intoxication.¹

**Glutamate**

Glutamate is an excitatory neurotransmitter. The binding of glutamate to N-methyl-D-aspartate (NMDA) receptors increases the flow of calcium ions across cell membranes, causing depolarization and increasing the cell’s responsiveness to stimulation. Acutely, alcohol inhibits the activity of glutamate, and chronic alcohol consumption increases the number of glutamate receptors, an effect that is often referred to as upregulation of receptors.¹

The general state of arousal of the central nervous system is to a large degree determined by equilibrium of activity between GABA and glutamate. Chronic alcohol use creates an abnormal imbalance between inhibitory and excitatory central nervous system activity, as the glutamate receptors are upregulated and the sensitivity of the GABA receptors to GABA is decreased. When someone who chronically
uses alcohol to excess suddenly stops drinking or precipitously reduces consumption of alcohol, there are two important effects: 1) the inhibitory effect of alcohol on the GABA system is removed, and 2) there is increased activity of the upregulated glutamate receptors. The result is a hyper-excitable state that causes the signs and symptoms of alcohol withdrawal syndrome such as agitation, elevated blood pressure and heart rate, and seizures.²

The imbalance of GABA and glutamate activity is thought to be the primary mechanism of action of alcohol withdrawal. However, there is evidence that other neurotransmitters and neurotransmitter receptors are involved as well, and this may form the basis for the use of certain drugs for the treatment of alcohol withdrawal.

**The Physiological Effects Of Alcohol Use**

Ethanol is rapidly absorbed from the gastrointestinal (GI) tract. Approximately 20% of a dose is absorbed from the stomach and the remainder is absorbed in the small intestine. The absorption process is usually complete within 60 minutes but the absence or presence of food can increase or delay absorption.

Alcohol dehydrogenase (ADH) is an enzyme found in the stomach and the liver and the first step in the metabolism of ethanol is ADH-induced conversion of ethanol to acetaldehyde. Acetaldehyde is converted by the mitochondrial enzyme acetaldehyde dehydrogenase to acetate and water and the acetate is converted to acetyl-CoA, which can be used for energy or to synthesize fatty acids.
The metabolism of alcohol is primarily dependent on the activity of ADH, and the average adult blood alcohol concentration decreases 15 - 20 mg/dL/hour. A drink of alcohol is defined as 14 grams of alcohol. Fourteen grams of alcohol are contained in 12 ounces of beer (5% alcohol content), 5 ounces of wine (12% alcohol content), and 1.5 ounces of whiskey or other distilled spirits (40% alcohol content).

Alcohol blood levels are reported in milligrams or grams of alcohol per deciliter. An example would be 250 mg/dL or .025 g/dL. An adult male weighing 165 pounds who drinks one 12-ounce container of beer would have a blood alcohol level of approximately 0.02 g/dL. The blood alcohol level that is the legal definition of intoxication is 0.08 g/dL or 80 mg/dL, so this individual would only need to drink slightly less than three, 12-ounce containers of beer to reach that level.

The primary neurological effect of acute alcohol use is central nervous system depression. Other common effects of acute alcohol consumption are impaired coordination and balance, and decreased inhibition. Moreover, acute alcohol ingestion decreases cardiac output and causes peripheral vasodilation, lowering blood pressure and increasing heart rate. Chronic alcohol use can cause peripheral neuropathy, cerebellar atrophy, and irreversible cognitive changes. Chronic alcohol ingestion increases the risk for hypertension and is a significant factor in the development of coronary artery disease. Damage to the liver and pancreas is a common effect of chronic alcohol consumption, and alcohol is an important cause of gastric bleeding. Excessive drinking significantly increases the risk of developing breast cancer, esophageal cancer, oral cancer, and rectal cancer.
**Alcohol Use Disorder**

The unhealthy use of alcohol by Americans is very common. The 2014 National Survey on Drug Use and Health noted that in 2014, 60.9 million Americans reported binge alcohol use in the past month and 16.3 million reported heavy drinking in the past month.³ Over 17 million American adults have an alcohol use disorder, and the twelve-month and lifetime prevalence of alcohol use disorder has been estimated to be 13.9% and 29.1%, respectively.⁴ The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) diagnostic criteria for alcohol use disorder are listed in Table 1; these have been changed slightly for the purposes of publication in this course.⁵

<table>
<thead>
<tr>
<th>Table 1: DSM-5 Diagnostic Criteria for Alcohol Use Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. A problematic pattern of alcohol use that causes clinically significant impairment and is accompanied by at least two of the following behaviors, occurring within a 12-month period:</td>
</tr>
<tr>
<td>1. More alcohol is used and alcohol use continues over a longer period than was intended.</td>
</tr>
<tr>
<td>2. The alcohol user has a persistent desire to decrease/control alcohol intake or he/she makes unsuccessful efforts to do so.</td>
</tr>
<tr>
<td>3. A great deal of time is spent in obtaining and using alcohol or recovering from the effects of drinking.</td>
</tr>
<tr>
<td>4. There is a craving, or a strong desire or urge to use alcohol.</td>
</tr>
<tr>
<td>5. Recurrent alcohol use that is the cause of failing to meet important obligations.</td>
</tr>
<tr>
<td>6. Continued alcohol use despite persistent/recurrent social or interpersonal problems caused or exacerbated by alcohol effects.</td>
</tr>
<tr>
<td>7. Social, occupational, or recreational activities are abandoned or curtailed because of alcohol use.</td>
</tr>
<tr>
<td>8. Recurrent alcohol use in situations that present physical danger.</td>
</tr>
<tr>
<td>9. Alcohol use is continued despite knowing that it is causing or exacerbating a physical or psychological problem.</td>
</tr>
<tr>
<td>Tolerance, defined as:</td>
</tr>
<tr>
<td>a. More alcohol is needed to become intoxicated or,</td>
</tr>
<tr>
<td>b. Same amount of alcohol produces a lower level of intoxication.</td>
</tr>
<tr>
<td>Withdrawal, evidenced by:</td>
</tr>
<tr>
<td>a. The withdrawal syndrome for alcohol as defined by the DSM-5 Criteria A and B of alcohol withdrawal or,</td>
</tr>
<tr>
<td>b. Alcohol or another sedative type substance such as a benzodiazepine is taken to relieve or avoid withdrawal symptoms.</td>
</tr>
</tbody>
</table>
**Alcohol Withdrawal: Basic Characteristics**

Over 17 million American adults have an alcohol use disorder and 50% of them will have signs and symptoms of alcohol withdrawal when they stop drinking or dramatically reduce how much they drink.\(^6\)\(^{-7}\) The signs and symptoms of alcohol withdrawal typically begin 8 hours after a change in alcohol intake pattern, although the onset may be earlier or later.\(^6\)\(^{-8}\) Most patients who have alcohol withdrawal have mild symptoms and out-patient care is sufficient for their management.\(^6\) Approximately 5% will develop severe alcohol withdrawal,\(^6\) putting them at risk for serious complications such as delirium tremens (DTs) and seizures.

Alcohol withdrawal syndrome is typically associated with people who are long-term, chronic users of alcohol. The risk of developing alcohol withdrawal increases with the amount of alcohol consumed and the frequency of drinking\(^6\) and although alcohol withdrawal is associated with chronic, heavy drinking, even a brief period of excess drinking followed by an abrupt cessation of drinking may cause alcohol withdrawal.\(^9\)

It is not clear why some people develop severe alcohol withdrawal.\(^2\) Factors that may increase a patient’s susceptibility to severe withdrawal include medical comorbidities, advanced age, previous episodes of alcohol withdrawal, development of DTs or seizures during a previous withdrawal, greater number of drinks during a 24 hour period, delayed recognition and delayed treatment of alcohol withdrawal, and genetic predisposition to severe withdrawal.\(^2,6,10,11\) The intensity of withdrawal symptoms appears to increase with each successive episode.\(^9\) The American Psychiatric Association’s diagnostic
criteria for alcohol withdrawal are listed in Table 2. The format has been modified slightly for this article.

Table 2: DSM-5 Diagnostic Criteria for Alcohol Withdrawal

| A. Cessation or reduction of alcohol use in someone who has been a chronic, heavy drinker. |
| B. Two or more of the following signs/symptoms that develop within hours to days after cessation or reduction of drinking: |
| 1. Autonomic hyperactivity such as diaphoresis or tachycardia |
| 2. Hand tremor |
| 3. Nausea or vomiting |
| 4. Generalized tonic-clonic seizures |
| 5. Insomnia |
| 6. Auditory, tactile or visual hallucinations or illusions, transitory |
| 7. Psychomotor agitation |
| 8. Anxiety |
| C. The signs and symptoms in criterion B cause significant functional impairment. |
| D. The signs and symptoms in criterion B are not caused by another medical problem, by a mental disorder, or intoxication or withdrawal from another drug. |

Clinical Presentation Of Alcohol Withdrawal

The initial signs and symptoms of alcohol withdrawal are relatively minor. It is important to remember that alcohol withdrawal can be caused by a reduction in alcohol consumption, not just complete cessation, so alcohol withdrawal can happen even if the patient has a significant blood alcohol concentration.
Table 3: Initial Signs and Symptoms of Alcohol Withdrawal

<table>
<thead>
<tr>
<th>Agitation</th>
<th>Anorexia</th>
<th>Anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diaphoresis</td>
<td>Headache</td>
<td>Hypertension</td>
</tr>
<tr>
<td>Insomnia</td>
<td>Nausea and Vomiting</td>
<td></td>
</tr>
<tr>
<td>Palpitations and Tachycardia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Restlessness</td>
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</tr>
</tbody>
</table>

In most cases these signs and symptoms resolve in one to two days\(^9\) and minor cases may be handled in an outpatient setting. However, approximately 5% of patients who are in alcohol withdrawal will progress to severe withdrawal.\(^6\),\(^7\) The serious signs and symptoms of severe alcohol withdrawal may not develop for several days and are not always preceded by obvious evidence of withdrawal.\(^11\) They include the signs and symptoms listed in Table 3 (but of a more intense nature) as well as alcoholic hallucinosis, aspiration, decreased cerebral blood flow, dehydration, DTs, electrolyte disorders, hyperthermia, hyperventilation, respiratory alkalosis, and seizures.\(^2\)

Mild alcohol withdrawal that does not progress to moderate or severe withdrawal will usually resolve in one or two days. Moderate and severe alcohol withdrawal may last for a week or more. The fatality rate of severe alcohol withdrawal is approximately 2%,\(^11\) and severe alcohol withdrawal can be accompanied by many serious complications. The most prominent and well-known of these are DTs and seizures and these will be discussed below in detail.
**Delirium Tremens**

Delirium tremens (DTs), which is also called withdrawal delirium or colloquially as *rum fits*, is a complication of severe alcohol withdrawal that is characterized by a floridly altered mental state. Patients suffering from DTs are agitated, confused, disoriented, and delirious, and they frequently have vivid and frightening hallucinations. Other signs and symptoms of DTs include but are not limited to, diaphoresis, fever, hypertension, nausea, tachycardia, tremor, and vomiting.

The clinical presentation of DTs and alcohol withdrawal are almost identical, and the DSM-5 criteria for alcohol withdrawal are part of the DSM-5 criteria for withdrawal delirium. The difference between the two is that *a patient who has DTs has delirium*. The DSM-5 criteria for delirium are listed in Table 4. The format has been modified slightly for this article.

**Table 4: DSM-5 Criteria for Delirium**

<table>
<thead>
<tr>
<th>Disturbance in attention such as a reduced ability to focus and maintain attention or the ability to appropriately shift attention as needed.</th>
</tr>
</thead>
<tbody>
<tr>
<td>The disturbance in attention develops within a few hours or days and it is a significant change from baseline. In addition, the disturbance fluctuates throughout the day.</td>
</tr>
<tr>
<td>An additional cognitive disturbance such as a memory deficit is present.</td>
</tr>
<tr>
<td>These disturbances are not explained by a pre-existing or developing neurocognitive disorder and have not occurred because of severe change in consciousness.</td>
</tr>
<tr>
<td>The history, laboratory studies, and/or the physical examination suggest that the disturbance in attention has been caused by another medical condition, substance intoxication or withdrawal (<em>i.e.</em>, due to a drug of use or to a medication), or exposure to a toxin, or is due to multiple etiologies.</td>
</tr>
</tbody>
</table>
Delirium tremens usually begin three days or so after the beginning of alcohol withdrawal.\textsuperscript{7} The syndrome will usually resolve in anywhere from one to eight days\textsuperscript{7} but the signs and symptoms of alcohol withdrawal will usually continue for several days past that point.\textsuperscript{8}

Patients who are likely to develop DTs are those who 1) have previously had DTs; 2) are chronic, heavy drinkers; 3) develop alcohol withdrawal while still having an elevated alcohol level; 4) are over age 30; 5) have a CIWA-Ar score > 15 (especially if the systolic blood pressure is > 150 mm Hg or the pulse is > 100 bpm); 6) have had withdrawal seizures; and, 7) have a concurrent illness, particularly electrolyte abnormalities or cardiac, gastrointestinal, or respiratory diseases.\textsuperscript{2,7}

Delirium tremens is a very serious complication of alcohol withdrawal. The fatality rate of DTs has been estimated to be 1\%-4\%.\textsuperscript{7} Death is caused by aspiration, cardiac arrhythmias, electrolyte disorders, exacerbation of concomitant medical problems, hyperthermia, or seizures.\textsuperscript{7,9} Failure to recognize DTs also contributes to the fatality rate of DTs.\textsuperscript{15}

Identifying patients who are in alcohol withdrawal, have DTs, or are at risk for either, can be problematic. An evaluation should include screening for alcohol use disorder but this is not always done; however, even if screening is done, a patient may not be candid about his or her alcohol consumption patterns when answering the questions in the screening test. Additionally, a patient who consumes alcohol may be admitted to a hospital for a medical issue. This prevents the patient from drinking alcohol, which may lead to alcohol withdrawal or DTs. Diagnosis of alcohol withdrawal or DTs may be difficult when the
clinical presentation of the medical issue and the signs and symptoms of alcohol withdrawal or DTs are essentially the same.\textsuperscript{10,16}

Alcohol use disorder is very common in hospitalized patients.\textsuperscript{17,18} It has been identified in 16\%-31\% of ICU patients,\textsuperscript{11} and 50\%-60\% of trauma patients\textsuperscript{19} so alcohol withdrawal and DTs should be suspected if a patient’s mental condition changes in sudden, unexpected ways.

**Withdrawal Seizures**

Seizures occur in approximately 10\% of all patients who are in alcohol withdrawal.\textsuperscript{2,9} The seizures usually occur 12-48 hours after the patient last had a drink (but they can occur much sooner),\textsuperscript{2} and patients who have used alcohol for many years or who have had previous episodes of alcohol withdrawal seizures are more likely to have a seizure than those who are having a first episode of alcohol withdrawal.\textsuperscript{2,11}

Alcohol withdrawal seizures are typically generalized tonic-clonic seizures and they can be a single seizure or a brief series of seizures.\textsuperscript{2} Seizures caused by alcohol withdrawal are usually self-limiting. Prolonged or recurrent seizures or status epilepticus should prompt investigation for a medically-related cause.\textsuperscript{2} Alcohol withdrawal seizures can cause DTs if they are not treated.

Clinicians should be aware that the relationship between alcohol and seizures is complex. Alcohol is well-known to precipitate seizures in patients who have epilepsy. Moreover, alcohol use is a common disorder in people who have epilepsy: the higher the consumption of alcohol the greater the risk for developing epilepsy. Diseases such as
cerebral infections, stroke, and trauma, which are commonly associated with alcohol use, are common causes of seizures.\textsuperscript{15}

**Alcoholic Hallucinosis**

Alcoholic hallucinosis is a well-described but relatively uncommon complication of alcohol withdrawal.\textsuperscript{2,9,20-22} Alcoholic hallucinosis is characterized by vivid, auditory hallucinations (and occasionally visual and tactile symptoms) that begin soon after cessation or reduction of alcohol intake and that resolve within 24-48 hours.\textsuperscript{2} The time of resolution of the hallucinations differentiates alcoholic hallucinosis from DTs.

This disorder appears to occur most often in patients who started drinking at an early age and who have a long history of heavy drinking, accompanied by illicit drug use.\textsuperscript{22} Taylor, \textit{et al.}, and de Millas, \textit{et al.}, note that some patients who have alcoholic hallucinosis go on to develop chronic hallucinations and chronic psychosis.\textsuperscript{22,23}

**Treatment Of Alcohol Withdrawal**

Alcohol withdrawal is a clinical diagnosis. It cannot be confirmed with laboratory studies or other objective testing. Alcohol withdrawal can be mistaken for many medical problems such as drug overdose, infection, and certain types of trauma and it can co-exist with them; so a careful assessment for alternate causes of a patient’s condition is necessary. Assessment for newly-developed medical problems must be done as well if it is not clear that alcohol withdrawal is present.
The basic treatments for alcohol withdrawal are: 1) Evaluation to identify medical problems caused by alcohol withdrawal such as aspiration, bleeding, dehydration, electrolyte disorders, and metabolic derangements; 2) Treating signs and symptoms; and, 3) Preventing progression of the syndrome. Providing care for a patient who has alcohol withdrawal is not complicated; however, it should be remembered that alcohol withdrawal often occurs in patients who have a poor underlying state of health. It can cause serious complications such as DTs, seizures, and death. These patients need constant attention and care because it may be a week or more before alcohol withdrawal resolves.

**Initial Evaluation**

After the initial physical assessment and history taking, the following diagnostic tests should be done. These recommendations assume that the patient will be admitted to a hospital and not treated as an outpatient. These tests should be repeated as necessary.

<table>
<thead>
<tr>
<th>Arterial or venous blood gas</th>
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</thead>
<tbody>
<tr>
<td>BUN and creatinine</td>
</tr>
<tr>
<td>Chest X-ray (if indicated by physical examination findings)</td>
</tr>
<tr>
<td>Complete blood count</td>
</tr>
<tr>
<td>Coagulation studies</td>
</tr>
<tr>
<td>Creatine kinase level</td>
</tr>
<tr>
<td>CT scan of the head</td>
</tr>
<tr>
<td>12-lead ECG</td>
</tr>
<tr>
<td>Electrolytes</td>
</tr>
<tr>
<td>Ethanol level</td>
</tr>
<tr>
<td>Liver function studies</td>
</tr>
<tr>
<td>Magnesium level</td>
</tr>
<tr>
<td>Pancreatic enzymes</td>
</tr>
<tr>
<td>Phosphate level</td>
</tr>
<tr>
<td>Serum glucose</td>
</tr>
</tbody>
</table>
The above recommendations may seem excessive; however, chronic alcohol use may cause anemia, cardiac disease, electrolyte disturbances, liver damage, and pancreatic damage. In addition, alcohol withdrawal often causes agitation, muscle damage and rhabdomyolysis, dehydration, and respiratory alkalosis.

**Symptomatic and Supportive Care**

Patients who have alcohol withdrawal are almost always dehydrated and hypomagnesemia, hypokalemia, and hypophosphatemia are also common. Initial care should include intravenous (IV) hydration, accurate monitoring of intake and output, and IV supplementation of magnesium, phosphate, and potassium, if needed. Thiamine and folate supplementation is often given empirically as deficiency in these vitamins is relatively common in this patient population.

It may or may not be safe for these patients to have oral intake but because of the increased metabolic activity caused by alcohol withdrawal and the possibility of malnutrition in patients who have alcohol use disorder, intravenous glucose should be started to provide a source of calories. If it appears that the clinical course will be prolonged, other forms of nutrition should be considered.

As mentioned above, alcohol withdrawal often causes agitation so the patient should be in a quiet atmosphere. Mechanical restraints may be needed if the patient is severely agitated and poses a danger to himself or herself or to the staff. Mechanical restraints should be used according to policy and removed as soon as possible.
Patient Care And Monitoring: Use Of The CIWA-Ar Scale

The cornerstone of treatment for a patient who is undergoing alcohol withdrawal is sedation. Sedation alleviates and controls the signs and symptoms of alcohol withdrawal and helps prevent complications. Determining how much sedation a patient needs is a clinical judgment made at the bedside by the nurse, and a symptom-triggered approach using the Clinical Institute Withdrawal Assessment for Alcohol, revised (CIWA-Ar) is recommended.²

The CIWA-Ar scale, commonly referred to as the CIWA Scale, is an assessment tool that is used to determine the severity of alcohol withdrawal.²,²⁴ There are ten items in the CIWA-Ar assessment tool. The responses or results of items number one through nine are given a score of 0 to 7, with 7 representing the severe manifestation. Item number 10 is given a score of 1 to 4.

Example 1

Visual disturbances, a score of seven would be given if a patient is highly sensitive to light and continually hallucinating.

Example 2

Orientation and clouding of sensorium, a score of 3 would be given if the patient is disoriented by more than two calendar days. The scores of the ten components are added and the patient’s condition is categorized a mild, moderate, or severe. Severe indicates that the patient may develop DTs, seizures, or both.⁷
Table 5: CIWA-Ar Components

<table>
<thead>
<tr>
<th>Agitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety</td>
</tr>
<tr>
<td>Auditory disturbances (hearing things that are not there, sensitivity to loud noise)</td>
</tr>
<tr>
<td>Headache</td>
</tr>
<tr>
<td>Nausea or vomiting</td>
</tr>
<tr>
<td>Orientation and clouding of sensorium</td>
</tr>
<tr>
<td>Paroxysmal sweating</td>
</tr>
<tr>
<td>Tactile disturbances: itching, the sensation of bugs crawling on the skin</td>
</tr>
<tr>
<td>Tremor</td>
</tr>
<tr>
<td>Visual disturbances such as hallucinations, sensitivity to light</td>
</tr>
</tbody>
</table>

The CIWA-Ar is used to evaluate the level of severity of alcohol withdrawal and it also provides nurses and other healthcare professionals with a tool that gives them objective data they can use to determine how much sedation a patient needs and how often sedation should be given.\(^2\) Using the CIWA-Ar in this way is called *symptom-triggered therapy*, and this approach has been shown to be superior to a fixed-dose approach to administering sedation.\(^1^6\) As Hoffman and Weinhouse write, symptom-triggered therapy is "*giving the patient the therapy they need, only when they need it ... (and) achieves equivalent or superior clinical endpoints while requiring lower doses of sedatives and shorter periods of hospitalization.*"\(^2\)

The CIWA-Ar scale can be used as often as every 15-30 minutes during periods of acute, severe withdrawal. If the patient’s condition improves and he or she responds to therapy, the interval between CIWA-Ar scale evaluations can be extended.
The CIWA-Ar scale is widely used but it does have limitations. For example, it cannot be used in patients who are unable to communicate verbally; moreover, it has not been extensively tested and validated; it has not been proven to be useful for critically ill patients admitted to intensive care; using the CIWA-Ar scale may cause a clinician to mistake a medical illness for alcohol withdrawal;\textsuperscript{16} and, completing a CIWA-Ar assessment can take up to 15 minutes.\textsuperscript{24} Nonetheless, the CIWA-Ar scale is commonly accepted and used, and it does provide a framework for a structured approach to symptomatic care of a patient going through alcohol withdrawal, particularly in the use of sedation.

The CIWA-Ar scale depends in part on the patient being able to answer questions. If this is not possible, the Richmond agitation-sedation scale (RASS) may be a viable alternative\textsuperscript{2,26} although it has not been studied extensively.\textsuperscript{27} The RASS has been formatted differently here for this article.

\begin{table}[h]
\centering
\begin{tabular}{|c|}
\hline
+4 - Combative and violent  
+3 - Aggressive towards the staff, uncooperative  
+2 - Agitated  
+1 - Restless  
0 - Alert and calm  
-1 - Drowsy  
-2 - Somewhat sedated  
-3 - Moderately sedated  
-4 - Deeply sedated  
-5 - Unresponsive  
\hline
\end{tabular}
\caption{Richmond Agitation-Sedation Scale (RASS)}
\end{table}
Pharmacological Treatment

Sedation by pharmacological treatment, especially the use of benzodiazepines, is a mainstay of treating patients undergoing alcohol withdrawal. The psychomotor agitation and the sympathetic hyperactivity that characterizes alcohol withdrawal can be quite uncomfortable and potentially dangerous and sedation is the most effective therapy for treatment of these signs and symptoms. Supportive care is also important for these patients but it does not address psychomotor agitation and sympathetic hyperactivity. A wide variety of drugs have been tried as treatments for alcohol withdrawal. The most commonly used drugs are benzodiazepines, barbiturates, dexmedetomidine, and Propofol.

After basic supportive care, sedation is the primary treatment for alcohol withdrawal. Many years of experience and a considerable amount of research support the use of benzodiazepines as the first-line agent for the treatment of alcohol withdrawal and when used with the CIWA-Ar scale, benzodiazepines are very effective. Very often benzodiazepines and supportive care are all that is needed but patients who have severe withdrawal or are having DTs often need additional pharmacologic intervention. If benzodiazepines are insufficient then dexmedetomidine, phenobarbital, or propofol can be added to the regimen.

Because there is no consensus for criteria or parameters for deciding when benzodiazepines are not adequate treatment, the choice of which medication to use and in what order to use them depends on the clinical situation and the familiarity of the providers with the drugs. There are many other drugs that can be used to treat alcohol
withdrawal that is unresponsive to benzodiazepines. However, the clinical evidence for their effectiveness is scant. There is even less evidence that can help clinicians decide which of these many medications may be helpful for a particular patient and how to add them to the treatment regimen. Ferreira, et al., stated that many therapies have been used “... including continuous infusion of GABA agonists, ethanol, dexmedetomidine, antiepileptics, and antipsychotics, introducing a significant amount of variability into clinical practice. This variability in treatment approaches highlights the lack of uniformity and recommendations available for the treatment of severe alcohol withdrawal.” A brief discussion of medications that have been or can be used for alcohol withdrawal is discussed here.

Benzodiazepines

Benzodiazepines are the most well-studied and commonly used drugs for the treatment of alcohol withdrawal.⁶,⁹,¹¹ The benzodiazepines are very effective at treating the psychomotor signs and symptoms of alcohol withdrawal such as anxiety, agitation, and tremor. They can prevent the progression of the syndrome from mild to major.

The benzodiazepines bind to specific areas on the GABA receptors. This binding increases the frequency of chloride ion channels opening, which in turn lead to hyperpolarization of cells. This makes these cells less able to respond to stimulation, causing central nervous system depression. Benzodiazepines that are commonly used to treat alcohol withdrawal are chlordiazepoxide, diazepam, lorazepam, midazolam, and oxazepam. As far as choosing a benzodiazepine, there is “... little empirical data to support the use of one benzodiazepine over another.”⁹
The benzodiazepines differ in their onset of action, duration of action, dosing, metabolism, and available forms, and these differences should be considered when using these drugs. For example, the rapid onset of the effects of diazepam using IV administration of the drug make it useful in controlling DTs and the initial, severe signs and symptoms of alcohol withdrawal. However, diazepam has a relatively long duration of action and it has pharmacologically active metabolites. These characteristics can cause prolonged over-sedation in certain patients.

The benzodiazepines can be given orally, intramuscularly (IM), intravenously (IV), or as a continuous infusion. Intramuscular administration is the least favorable route because of erratic absorption. The amount of a benzodiazepine that is needed is determined by using the CIWA-Ar scale and by using prescribing data for the drug. In addition, the patient’s age and ability to metabolize and excrete drugs and the presence of medical co-morbidities must be considered.

However, the doses that are used for patients who are in alcohol withdrawal will often far exceed the normal dosing guidelines: up to 50 mg of diazepam in one hour, a 24-hour total dose of lorazepam of 484 mg, and a 24-hour total dose of diazepam of 2160 mg have been used. These amounts are far more than the labeled, approved doses and although physicians do have the discretion to off-label dosing, it gives a sense of perspective to know that the maximum recommended 24-hour dose of lorazepam is 10 mg.

Common side effects of the benzodiazepines are drowsiness, hypotension, and respiratory depression. It is important for clinicians
to consider how sedated the patient should be. The answer will differ with each patient care situation, patient comfort and safety. Avoiding complications are the primary considerations for using sedation, not the convenience of the staff. Although benzodiazepines are very effective for treating alcohol withdrawal, when high doses of these drugs are administered for a prolonged period, severe withdrawal signs and symptoms can occur if the benzodiazepines are not slowly tapered.

**Dexmedetomidine**

Dexmedetomidine is classified as an alpha-adrenergic agonist and a sedative, and its labeled uses are: 1) sedation of patients who are admitted to intensive care, intubated and mechanically ventilated; and, 2) as a sedative prior to performance of certain procedures.

The sedative of action of dexmedetomidine is thought to be due, in part, by increasing the activity of alpha<sub>2</sub>a-adrenoceptors in the brainstem resulting in inhibition of norepinephrine release, and by increasing the activity of peripheral alpha<sub>2</sub>b receptors. Dexmedetomidine is given as an IV bolus; the onset of action is 5-10 minutes, the peak occurs within 15-30 minutes, and the duration of action is 60-120 minutes, depending on the dose. The IV bolus is followed by a continuous IV maintenance infusion.

Dexmedetomidine has proven to be a useful adjunct in treating moderate to severe alcohol withdrawal but not all clinicians are enthusiastic about its use for this purpose. Several studies have shown that the use of dexmedetomidine can reduce patients’ needs for benzodiazepines and antipsychotics and unlike the
Benzodiazepines, dexmedetomidine does not cause respiratory depression. However, this same research did not find that adding dexmedetomidine to the medication protocol reduced the length of hospital stay or ICU stay. Dexmedetomidine can cause bradycardia and hypotension (well-known side effects of the drug), and there are no controlled trials that show dexmedetomidine reduces the risk of DTs or seizures. The conclusion is that it should not be used as the primary drug for this clinical situation.

**Barbiturates**

The barbiturates, specifically phenobarbital, have long been used to treat alcohol withdrawal. Phenobarbital increases the binding of GABA to GABA receptors and it increases the duration of opening of chloride ions channels. The net result is similar to that of benzodiazepines - hyperpolarization of the cells and a decrease in the responsiveness of the cells to stimulation; however, because the mechanism of action of the barbiturates is slightly different from that of benzodiazepines, using them together produces a synergistic effect and the combination of a barbiturate and a benzodiazepine can be useful for treating alcohol withdrawal, especially if the patient is not responding to benzodiazepines. When it is given intravenously the onset of action of phenobarbital is five minutes and the duration of action is six hours or longer. Sedation and respiratory depression are common side effects of barbiturates.

Phenobarbital has been shown to be an effective adjunct for treatment of alcohol withdrawal and has been used as an initial emergent therapy before starting benzodiazepines or when patients are refractory to therapy with benzodiazepines, but there is little
evidence that provides clear guidance to clinicians regarding when and for whom the drug should be used.\textsuperscript{2,6,11,35}

**Propofol**

Propofol is a short-acting, IV general anesthetic that is used for the induction and maintenance of anesthesia and for sedating intubated, mechanically-ventilated ICU patients. The mechanism of action of propofol is thought to be by agonism of GABA\textsubscript{A} receptors and (possibly) by reducing glutamatergic activity through NMDA receptor blockade. The onset of action can be from 9 to 51 seconds, the average being 30 seconds. The duration of action depends on how much is given, the rate of administration, and the duration of administration. Three to 10 minutes is the average duration of action but this can be prolonged 10-15 minutes after extended use.\textsuperscript{36}

Propofol has been recommended as a treatment for severe alcohol withdrawal and/or DTs.\textsuperscript{2} It can be effective for this purpose, and it can reduce the need for benzodiazepines and antipsychotics.\textsuperscript{37} For patients who are not responding to benzodiazepines, the use of propofol should be considered.\textsuperscript{37} When using propofol for treating alcohol withdrawal, keep the following points in mind: 1) hypotension is a common side effect of propofol; 2) patients receiving propofol often require mechanical ventilation; 3) patients undergoing alcohol withdrawal who are given propofol and are intubated will need intubation for a relatively long time; and, 4) as noted by Brotherton, et al., in their 2016 review, “... *data assessing the utility of propofol for AWS exhibited significant heterogeneity.*”\textsuperscript{37}
Prolonged administration of high doses of propofol can cause a rare but potentially fatal complication called *propofol infusion syndrome* (PRIS). Signs and symptoms of PRIS include (but are not limited to) arrhythmias, hypotension, and metabolic acidosis. The incidence of PRIS is very low, approximately 1%. Propofol infusion syndrome can also happen when relatively low doses of the drug are given.

**Antipsychotics**

Antipsychotics such as haloperidol can be used for patients who need more sedation than the benzodiazepines can provide, but there are significant risks associated with these drugs when used for this purpose; *i.e.*, hypotension, lowering the seizure threshold, and interference with heat dissipation. Hoffman, *et al.*, wrote: “Treatment with antipsychotics would only be appropriate when a decompensated thought disorder (such as schizophrenia) coexists with ethanol withdrawal and any symptoms associated with ethanol withdrawal have been definitively treated with benzodiazepines.” Prior to administration of an antipsychotic, a 12-lead ECG should be done to be sure the QTc is not prolonged and serum magnesium and potassium should be measured.

**Anticonvulsants**

The anticonvulsants carbamazepine, gabapentin, and levetiracetam have been used to treat alcohol withdrawal. These drugs may be useful as adjunctive therapies or in certain clinical situations, but there is little experience with their use and very few clinical trials that have closely examined their benefits and risks. Alcohol withdrawal seizures are almost always self-limiting and, if status epilepticus occurs, phenobarbital and propofol can be used.
**Muscle Relaxant**

Baclofen is a muscle relaxant, antispasmodic, that has been used to treat alcohol withdrawal but there is insufficient evidence for its effectiveness.\(^2,39\)

**Beta-blockers**

Beta-blockers may alleviate some of the signs and symptoms of alcohol withdrawal but there is no evidence that they are superior to the benzodiazepines.\(^2\)

**Ethanol**

Ethanol has often been used to treat patients who are going through alcohol withdrawal and there is evidence that it may be effective for this\(^39\) but current research and expert opinion recommends against using ethanol as a treatment for alcohol withdrawal.\(^2,21,31\)

**Clinical Care Of Alcohol Withdrawal**

Clinical care of a patient who is going through alcohol withdrawal can be very time consuming. These patients are often very agitated and confused and they may have significant changes in their vital signs. Many have comorbidities that are the precipitating cause of withdrawal or made worse by withdrawal. They require constant attention, and assessment, and reassessment. Clinical care, however, is not complicated. The specific primary clinical actions that are important in these situations are highlighted in the Table below.
Careful use of the CIWA-Ar scale.
Assessment of the effectiveness of sedation.
Awareness of and observation for the side effects of sedation.
Frequent measuring of vital signs.
Monitoring of intake and output.
Frequent assessment of the patient’s neurological status.
Fluid replacement as needed.
Observing for and treating complications such as aspiration, hyperthermia, and seizures.
Maintaining a quiet and calm atmosphere.
Helping the patient to be oriented to time, place, and person.

Summary

Alcohol withdrawal is a syndrome caused by a sudden cessation of alcohol intake or sudden reduction of alcohol intake in people who are chronic, excessive users of alcohol. The signs and symptoms of alcohol withdrawal typically begin approximately 8 hours after the characteristic change in alcohol intake, although the onset may be earlier or later. The clinical presentation of alcohol withdrawal can often be mild, however serious complications and death are certainly possible.

Chronic excessive use of alcohol disrupts the balance of activity of the neurotransmitters gamma-aminobutyric acid (GABA) and glutamate, and when drinking stops the inhibitory effect of alcohol on the GABA system is removed and there is increased activity of the up-regulated glutamate receptors. The result is a hyper-excitatable state that causes the signs and symptoms of alcohol withdrawal syndrome.

The CIWA-Ar scale is an assessment tool that is used to determine the level of alcohol withdrawal and to guide the need for, and effectiveness
Treatment of alcohol withdrawal involves basic supportive care and sedation. The most commonly used drug to provide sedation for patients in alcohol withdrawal are the benzodiazepines. In severe cases, if the patient has DTs, or if benzodiazepines are not providing sufficient sedation dexmedetomidine, phenobarbital, or propofol can be added to the medication regimen.

Alcohol withdrawal syndrome is typically associated with people who chronically use alcohol, and the risk of developing alcohol withdrawal increases with the amount and frequency of drinking. The signs and symptoms of alcohol withdrawal have been reviewed here, including severe withdrawal where there is the risk for alcoholic hallucinosis, aspiration, dehydration, DTs, seizures among other symptoms. It is necessary for health clinicians to be able to recognize levels of alcohol withdrawal. If quickly recognized and appropriately treated, for patients in mild withdrawal, the survival rate is high.

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Completing the study questions is optional and is NOT a course requirement.
1. Alcohol withdrawal is caused by
   a. sudden stopping or drastic reduction of drinking.
   b. long-term alcohol abuse.
   c. having more than five drinks a day.
   d. stopping drinking.

2. The neurotransmitters primarily involved in alcohol withdrawal are
   a. acetylcholine and dopamine.
   b. epinephrine and serotonin.
   c. GABA and glutamate.
   d. norepinephrine and adenosine.

3. The onset of alcohol withdrawal usually begins within
   a. 24 hours.
   b. 72 hours.
   c. 1 hour.
   d. 8 hours.

4. True or False: Alcohol withdrawal can occur if the patient has a measurable ethanol level.
   a. True
   b. False

5. The risk of developing alcohol withdrawal increases with
   a. delirium tremens (DTs).
   b. the amount and frequency of drinking.
   c. cognitive changes.
   d. long-term drinking and female gender.

6. Gamma-aminobutyric acid (GABA) is a neurotransmitter that is best described as
   a. inhibitory.
   b. depolarizing.
   c. excitatory.
   d. an up-regulator.
7. Most cases of alcohol withdrawal are
   a. mild.
   b. severe.
   c. fatal.
   d. accompanied by seizures.

8. Severe alcohol withdrawal usually begins
   a. in 1-2 hours.
   b. after seizures have occurred.
   c. in several days.
   d. when delirium tremens (DTs) have started.

9. Severe alcohol withdrawal typically causes
   a. bradycardia and respiratory depression.
   b. drowsiness and status epilepticus.
   c. agitation and tachycardia.
   d. hypertension and hypothermia

10. Complications of serious alcohol withdrawal include
    a. delirium tremens and seizures.
    b. renal failure and pulmonary edema.
    c. arrhythmias and deep vein thrombosis.
    d. congestive heart failure and cerebrovascular accident.

11. Seizures caused by alcohol withdrawal are usually
    a. multiple.
    b. prolonged.
    c. status epilepticus.
    d. self-limiting.

12. Patient who are going through alcohol withdrawal often
    a. are well-nourished.
    b. are dehydrated.
    c. not in need of nutritional support.
    d. are in fluid overload.
13. The primary drug used to treat alcohol withdrawal is
   a. ethanol.
   b. the benzodiazepines.
   c. baclofen.
   d. dexmedetomidine.

14. Which of these is used to treat severe alcohol withdrawal?
   a. morphine.
   b. aspirin.
   c. phenobarbital.
   d. heparin.

15. A common side effect of propofol is
   a. hypotension.
   b. hyperthermia.
   c. fever.
   d. hypothermia.

16. The ______ scale is commonly used to assess and treat patients in alcohol withdrawal.
   a. MAST
   b. CIWA-Ar
   c. Glasgow
   d. APACHE

17. Chronic alcohol use can cause peripheral neuropathy, cerebellar atrophy, and
   a. temporary depression.
   b. decreased inhibition.
   c. irreversible cognitive changes.
   d. alcohol withdrawal syndrome.

18. True or False: The DSM-5 criteria for alcohol withdrawal are part of the DSM-5 criteria for withdrawal delirium.
   a. True
   b. False
19. The signs and symptoms of alcohol withdrawal typically begin approximately ______ after a reduction in alcohol intake.
   a. 8 hours
   b. one week
   c. 24 hours
   d. two days

20. The most prominent, well-known, and serious complications of alcohol withdrawal are
   a. alcoholic hallucinosis and headache.
   b. dehydration and restlessness.
   c. anxiety and dehydration.
   d. delirium tremens (DTs) and seizures.

21. Alcohol withdrawal seizures are usually
   a. not self-limiting.
   b. generalized tonic-clonic seizures.
   c. absence seizures.
   d. limited to people with pre-existing epilepsy.

22. Alcoholic hallucinosis appears to occur most often in patients who, among other things,
   a. already suffered from a psychosis.
   b. started drinking at an early age.
   c. were homeless.
   d. had pre-existing epilepsy.

23. The cornerstone of treatment for a patient who is undergoing alcohol withdrawal is
   a. abstinence.
   b. rehydration.
   c. sedation.
   d. vitamin supplementation.
24. True or False: Patients who have alcohol withdrawal are almost always dehydrated.
   
   a. True
   b. False

25. The Clinical Institute Withdrawal Assessment for Alcohol, revised (CIWA-Ar) is used to evaluate
   
   a. the level of severity of alcohol withdrawal.
   b. how much sedation a patient needs.
   c. how often a patient needs sedation.
   d. All of the above

26. ____________ is/are the most well-studied and most commonly used drug(s) for the treatment of alcohol withdrawal.
   
   a. Barbiturates
   b. Benzodiazepines
   c. Dexmedetomidine
   d. Propofol

27. True or False: Baclofen has been used to treat alcohol withdrawal because of its documented effectiveness.
   
   a. True
   b. False

28. Alcohol withdrawal seizures are almost always self-limiting and if status epilepticus occurs, ____________ can be used to treat it.
   
   a. baclofen
   b. ethanol
   c. phenobarbital and propofol
   d. haloperidol
29. With chronic alcohol use glutamate receptors are ________ and the sensitivity of the GABA receptors to GABA is decreased.

a. reversed  
b. inhibited  
c. blocked  
d. upregulated

30. True or False: Beta-blockers may alleviate some of the signs and symptoms of alcohol withdrawal but there is no evidence that they are superior to the benzodiazepines.

a. True  
b. False
CORRECT ANSWERS:

1. Alcohol withdrawal is caused by

   a. sudden stopping or drastic reduction of drinking.

   "Alcohol withdrawal is a syndrome caused by a sudden cessation of alcohol intake or sudden reduction of alcohol intake in people who are chronic, excessive users of alcohol."

2. The neurotransmitters primarily involved in alcohol withdrawal are

   c. GABA and glutamate.

   "Chronic excessive use of alcohol disrupts the balance of activity of the neurotransmitters gamma-aminobutyric acid (GABA) and glutamate, and when drinking stops the inhibitory effect of alcohol on the GABA system is removed and there is increased activity of the up-regulated glutamate receptors."

3. The onset of alcohol withdrawal usually begins within

   d. 8 hours.

   "The signs and symptoms of alcohol withdrawal typically begin approximately 8 hours after the characteristic change in alcohol intake, although the onset may be earlier or later."

4. True or False: Alcohol withdrawal can occur if the patient has a measurable ethanol level.

   a. True

   "It is important to remember that alcohol withdrawal can be caused by a reduction in alcohol consumption, not just complete cessation, so alcohol withdrawal can happen even if the patient has a significant blood alcohol concentration."
5. The risk of developing alcohol withdrawal increases with
   b. the amount and frequency of drinking.
   "The risk of developing alcohol withdrawal increases with the amount and frequency of drinking..."

6. Gamma-aminobutyric acid (GABA) is a neurotransmitter that is best described as
   a. inhibitory.
   "Gamma-aminobutyric acid (GABA) is one of the primary inhibitory neurotransmitter in the central nervous system."

7. Most cases of alcohol withdrawal are
   a. mild.
   "Most patients who have alcohol withdrawal have mild symptoms, but approximately 5% develop severe alcohol withdrawal and are at risk for serious complications, such as delirium tremens (DTs) and seizures."

8. Severe alcohol withdrawal usually begins
   c. in several days.
   "The serious signs and symptoms of severe alcohol withdrawal may not be manifested for several days and are not always preceded by obvious evidence of withdrawal."

9. Severe alcohol withdrawal typically causes
   c. agitation and tachycardia.
   "When someone who chronically uses alcohol to excess suddenly stops drinking or precipitously reduces consumption of alcohol, ... the result is a hyper-excitable state that causes the signs and symptoms of alcohol withdrawal syndrome such as agitation, elevated blood pressure and heart rate, and seizures."
10. Complications of serious alcohol withdrawal include
   a. delirium tremens and seizures.

   "Most patients who have alcohol withdrawal have mild symptoms, but approximately 5% develop severe alcohol withdrawal and are at risk for serious complications, such as delirium tremens (DTs) and seizures."

11. Seizures caused by alcohol withdrawal are usually
   d. self-limiting.

   "Alcohol withdrawal seizures are almost always self-limiting and if status epilepticus occurs phenobarbital and propofol can be used."

12. Patient who are going through alcohol withdrawal often
   b. are dehydrated.

   "Patients who have alcohol withdrawal are almost always dehydrated and hypomagnesemia, hypokalemia, and hypophosphatemia are common, as well."

13. The primary drug used to treat alcohol withdrawal is
   b. the benzodiazepines.

   "Many years of experience and a considerable amount of research support the use of benzodiazepines as the first-line agent for the treatment of alcohol withdrawal and when used with the CIWA-Ar scale these drugs are very effective."

14. Which of these is used to treat severe alcohol withdrawal?
   c. phenobarbital.

   "Alcohol withdrawal seizures are almost always self-limiting and if status epilepticus occurs phenobarbital and propofol can be used."
15. A common side effect of propofol is
   a. hypotension.
   "Hypotension is a common side effect of propofol…"

16. The _____ scale is commonly used to assess and treat patients in alcohol withdrawal.
   b. CIWA-Ar
   "The CIWA-Ar scale is an assessment tool that is used to determine the level of alcohol withdrawal and also to guide the need for, and effectiveness of sedation."

17. Chronic alcohol use can cause peripheral neuropathy, cerebellar atrophy, and
   c. irreversible cognitive changes.
   "Chronic alcohol use can cause peripheral neuropathy, cerebellar atrophy, and irreversible cognitive changes."

18. True or False: The DSM-5 criteria for alcohol withdrawal are part of the DSM-5 criteria for withdrawal delirium.
   a. True
   "The clinical presentation of DTs and alcohol withdrawal are almost identical, and the DSM-5 criteria for alcohol withdrawal are part of the DSM-5 criteria for withdrawal delirium."

19. The signs and symptoms of alcohol withdrawal typically begin approximately ______ after a reduction in alcohol intake.
   a. 8 hours
   "The signs and symptoms of alcohol withdrawal typically begin approximately 8 hours after the characteristic change in alcohol intake, although the onset may be earlier or later."
20. The most prominent, well-known, and serious complications of alcohol withdrawal are
d. delirium tremens and seizures.

"The fatality rate of severe alcohol withdrawal is approximately 2%, and severe alcohol withdrawal can be accompanied by many serious complications. The most prominent and well known of these are DTs and seizures and these will be discussed below in detail."

21. Alcohol withdrawal seizures are usually
b. generalized tonic-clonic seizures.

"Alcohol withdrawal seizures are usually generalized tonic-clonic seizures and they can be a single seizure or a brief series of seizures."

22. Alcoholic hallucinosis appears to occur most often in patients who, among other things,
b. started drinking at a young age.

"Alcoholic hallucinosis is a well described but rare complication of alcohol withdrawal.... This disorder appears to occur most often in patients with a long history of heavy drinking, who had started drinking at a young age, and have used illicit drugs."

23. The cornerstone of treatment for a patient who is undergoing alcohol withdrawal is
c. sedation.

"The cornerstone of treatment for a patient who is undergoing alcohol withdrawal is sedation."
24. True or False: Patients who have alcohol withdrawal are almost always dehydrated.

a. True

"Patients who have alcohol withdrawal are almost always dehydrated and hypomagnesemia, hypokalemia, and hypophosphatemia are common, as well."

25. The Clinical Institute Withdrawal Assessment for Alcohol, revised (CIWA-Ar) is used to evaluate

a. the level of severity of alcohol withdrawal.
b. how much sedation a patient needs.
c. how often a patient needs sedation.
d. All of the above [correct answer]

"The CIWA-Ar is used to evaluate the level of severity of alcohol withdrawal and it also provides nurses and other healthcare professionals with a tool that gives them objective data they can use to determine how much sedation a patient needs and how often he/she needs sedation."

26. _____________ is/are the most well-studied and most commonly used drug(s) for the treatment of alcohol withdrawal.

b. Benzodiazepines

"Many years of experience and a considerable amount of research support the use of benzodiazepines as the first-line agent for the treatment of alcohol withdrawal and when used with the CIWA-Ar scale these drugs are very effective."

27. True or False: Baclofen has been used to treat alcohol withdrawal because of its documented effectiveness.

b. False

"Baclofen has been used to treat alcohol withdrawal but there is insufficient evidence for its effectiveness."
28. Alcohol withdrawal seizures are almost always self-limiting and if status epilepticus occurs, ____________ can be used to treat it.

c. phenobarbital and propofol.

"Alcohol withdrawal seizures are almost always self-limiting and if status epilepticus occurs phenobarbital and propofol can be used."

29. With chronic alcohol use glutamate receptors are _______ and the sensitivity of the GABA receptors to GABA is decreased.

d. upregulated

"Chronic alcohol use creates an abnormal imbalance between inhibitory and excitatory central nervous system activity, as the glutamate receptors are upregulated and the sensitivity of the GABA receptors to GABA is decreased."

30. True or False: Beta-blockers may alleviate some of the signs and symptoms of alcohol withdrawal but there is no evidence that they are superior to the benzodiazepines.

a. True

"Beta-blockers may alleviate some of the signs and symptoms of alcohol withdrawal but there is no evidence that they are superior to the benzodiazepines."
Reference Section

The References below include published works and in-text citations of published works that are intended as helpful material for your further reading.


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