

Alcohol Withdrawal Syndrome

Overlooked and Mismanaged?

Mary G. McKinley, RN, MSN, CCRN

Patients are admitted to critical care with a myriad of problems. A problem easily overlooked is alcohol dependency and the potential development of alcohol withdrawal syndrome (AWS). Fourteen million persons in the United States abuse alcohol or are alcohol dependent. Nearly 27% of persons in the United

States between the ages of 18 and 64 years meet the diagnostic criteria for alcohol dependency, making it the most prevalent addictive disease in this country. According to estimates, 1 of every 5 patients admitted to a hospital is an alcohol abuser.¹

When patients are admitted for problems such as trauma, alcohol is often associated as a factor, and trauma patients are more likely than other patients to be assessed for AWS. Admissions to a critical care unit because of nontrauma conditions, however, are not always so clearly linked to alcohol abuse, and AWS can easily be overlooked. Overlooking AWS can have devastating consequences and complications. Persons who abuse alcohol have mortality and morbidity rates 2 to 4 times greater

than those of the general population.² The toxic effects of alcohol abuse on organs and tissues put these patients at increased risk for multiple system dysfunction. Early detection of and intervention for AWS can prevent, or at least minimize, the complications and consequences of the syndrome.

Terminology

Because confusion can exist about the different terms associated with AWS, clarification is necessary (see Sidebar). Alcohol abuse cannot be diagnosed on the basis of a set amount or frequency of alcohol consumption. Signs and symptoms of withdrawal rarely occur in persons who drink only occasionally. AWS usually occurs in persons who have been heavy drinkers for weeks or months who suddenly stop drinking. An example is a patient admitted to a critical care unit for treatment of an acute hypertensive episode who is a heavy drinker but because of the admission cannot continue the alcohol intake. An understanding of the signs and symptoms and behavioral indicators is key in recognizing patients who are at risk for AWS.



* This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. Describe pathophysiology involved between alcohol and alterations of neurotransmission in the brain
2. Define the relationship between alcohol and thiamine deficiency
3. Examine the management of patients undergoing alcohol withdrawal syndrome

Author

Mary G. McKinley is a graduate of Ohio Valley General Hospital Diploma School of Nursing, Wheeling College (now Wheeling Jesuit University), Wheeling, WV. She received her master's degree from West Virginia University, Morgantown, WV. She has maintained her CCRN certification since 1985. She is past president of the American Association of Critical-Care Nurses. She has published in the area of critical care and has spoken nationally on topics related to critical care and nursing.

To purchase reprints, contact The InnoVision Group, 101 Columbia, Aliso Viejo, CA 92656. Phone, (800) 809-2273 or (949) 362-2050 (ext 532); fax, (949) 362-2049; e-mail, reprints@aacn.org.

Definition of Terms

Alcohol abuse: Recurrent, harmful use of alcohol with failure to fulfill major work, school, or home responsibilities; drinking in potentially dangerous situations (eg, driving a car); having recurrent legal problems related to alcohol use; and continued drinking despite having ongoing relationship problems caused or worsened by drinking²

Alcohol dependence or alcoholism:

A disease that includes 4 symptoms: craving (compulsion to drink), loss of control (unable to limit drinking on any given occasion), physical dependence (signs and symptoms of withdrawal occur when alcohol use is stopped), and tolerance (need to drink greater amounts to “get high”)²

Alcohol withdrawal syndrome: A set of signs and symptoms, including hyperreactivity such as nausea, vomiting, sweating, shakiness, agitation, and anxiety, that develop when alcohol use is stopped after a period of heavy drinking¹

Alcohol withdrawal delirium or delirium tremens:

The worst form of alcohol withdrawal syndrome and a medical emergency with signs and symptoms of hallucinations, confusion, disorientation, generalized seizures, and pronounced autonomic hyperreactivity with hypertension, tachycardia, hyperthermia, tachypnea, and tremors¹

Pathophysiology

The complex mechanisms of alcohol intoxication, tolerance, dependence, and withdrawal are not completely understood, but a clear relationship exists between alcohol and alterations in neurotransmission in the brain. Neurotransmitters are chemical messengers that transmit information from one neuron to another across synapses. Neurotransmitters in the brain either promote (excitatory) or slow down (inhibitory) impulses along the neurons.

One of the main neurotransmitters in the brain affected by alcohol is the inhibitory neurotransmitter γ -aminobutyrate A (GABAA). GABAA allows more chloride to enter the neuron, making the neuronal cell membranes less likely to depolarize, thereby inhibiting the cell. A second neurotransmitter of importance in alcohol abuse is the excitatory neurotransmitter *N*-methyl-D-aspartate (NMDA). NMDA regulates neuron excitability, increasing depolarization of the neuronal membrane by regulating the flow of calcium into the neuron. Initially, in short-term use (1 or 2 drinks occasionally), alcohol enhances the GABAA receptors and inhibits the NMDA receptors.³ The result of short-term alcohol use therefore is depression of the behavioral inhibitory centers in the cerebral cortex and the reticular activating system, causing initial euphoria, exaggerated feelings of

well-being, and reduced self-control and then sedation and anesthesia (Figure 1).

The pathophysiological effects of long-term use of alcohol involve the same neurotransmitters but are complicated by tolerance and physical dependence as contributing factors to the problem of withdrawal of alcohol. Long-term alcohol intake (see Sidebar for indicators of alcohol dependence) results in a decrease in GABAA inhibitory function and an increase in NMDA excitatory function.³ Continued intake of alcohol causes adaptation of the central nervous system, reducing the initial, short-term effects. Abrupt cessation of intake of alcohol or withdrawal produces a rebound stimulatory effect, resulting in adrenergic hypersensitivity of the limbic system and brain stem, which can lead to irritability (manifested as aggressive behavior), tremors, and seizures.¹

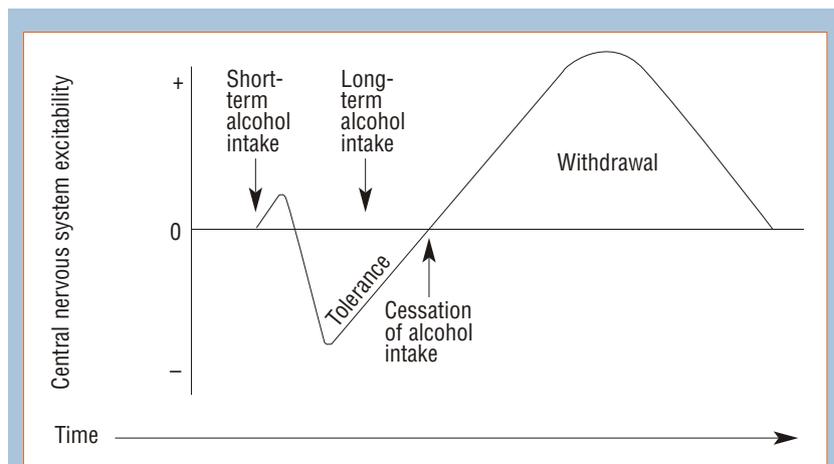


Figure 1 Schematic representation of the effects of alcohol exposure and withdrawal. The zero line represents the excitability of the brain. Short-term alcohol intake produces a depression of the inhibitory centers of the cerebral cortex, which results in the initial symptoms of intoxication (euphoria, exaggerated feelings of well-being, and loss of self-control followed by sedation). Long-term alcohol intake causes the initial decrease with tolerance that occurs during continued exposure to alcohol. Removal of alcohol causes a rebound stimulatory effect, increasing excitability in the nervous system.

Adapted from Finn and Crabbe.³

An additional problem that occurs with long-term use of alcohol is tolerance or a need to increase the amount of alcohol to obtain desired effects. Three types of tolerance develop.

The first type is metabolic tolerance or an increase in the rate of ethanol metabolism in the liver. As the ethanol is more rapidly metabolized, larger and still larger amounts of alcohol are needed to achieve the desired effects. The second type is cellular tolerance, which develops through the continued exposure of the cells in the brain to the neurochemical changes, the activities of GABAA, NMDA, and other neurotransmitters. Prolonged use of alcohol results in adaptation of the neurons to the use of alcohol, and larger doses of alcohol are needed to produce the same result. The third type is behavioral tolerance, learning to adapt behavior so the person can function under the influence of alcohol.¹

These 3 forms of tolerance can complicate the management of patients who have alcohol problems. For example, cellular tolerance may result in the need for increased dosages of medication to produce the desired results for a patient who has AWS.

Long-term exposure to alcohol also leads to dependence. In alcohol dependence, the neuronal adaptation to alcohol has become pronounced and the continued presence of alcohol is required for proper neuronal functioning. The metabolic and cellular changes caused by long-term use of alcohol do not resolve with cessation of drinking. The management of patients with AWS requires knowledge of the physical dependency caused by long-term use of alcohol.

Long-term alcohol dependency also leads to thiamine deficiency, a contributing factor to the pathophysiology of AWS.⁴ Thiamine deficiency occurs for 2 reasons: inadequate intake due to a poor nutritional state and interruption of the metabolism of thiamine by alcohol. Thiamine plays a key role in glucose metabolism. Thiamine pyrophosphate (the active form of thiamine) acts as a coenzyme for 2 enzymes in the Krebs or tricarboxylic acid cycle: ketoglutarate dehydrogenase and pyruvate dehydrogenase. Most likely, a deficiency in thiamine pyrophosphate alters neuronal energy metabolism in the central nervous system and diminishes nerve transmission. The major organs affected by thiamine deficiency are those that depend on energy from the metabolism of carbohydrates: the peripheral nerves, heart, and brain.

Peripheral neuropathy with myelin degeneration is the result of thiamine deficiency in the peripheral nerves. Cardiomyopathy and hypertension can occur because of effects of thiamine deficiency on the heart.¹ Wernicke encephalopathy and Korsakoff syndrome are the result of chronic thiamine deficiency in the brain. Wernicke encephalopathy is characterized by ophthalmoplegia (paralysis of the eye muscles), nystagmus (horizontal and vertical involuntary, rapid, rhythmic movements of the eyeballs), and ataxia (slow, uncertain, short-stepped gait). Signs and symptoms of Korsakoff syndrome include retrograde amnesia (inability to recall information), anterograde amnesia (inability to assimilate new information), decreased spontaneity, decreased initiative, and confabulation (filling

in memory gaps with distorted facts).¹ Understanding the role of thiamine deficiency in long-term alcohol abuse is helpful in recognizing important clinical manifestations and in managing patients with AWS.

Clinical Features

Signs and symptoms of autonomic hyperreactivity and neuropsychiatric alterations characterize AWS. Autonomic hyperreactivity includes tremors, sweating, nausea, and vomiting. Neuropsychiatric alterations include agitation, anxiety, auditory disturbances, clouding of sensorium, and disturbances in visual or tactile senses. These signs and symptoms are somewhat nonspecific and therefore are easily confused with other problems common in critical care patients, such as electrolyte imbalances, pain, and infection.

The signs and symptoms of AWS usually occur within 24 hours after the last drink. They peak in 24 to 36 hours and end after 48 hours.⁵

Alcohol withdrawal delirium or delirium tremens can occur within 48 to 72 hours after the last drink. Delirium tremens is a serious and life-threatening complication of alcohol withdrawal and should be treated as a medical emergency. The peak time for occurrence of delirium is usually day 4 after cessation of alcohol use, and the delirium can persist for 2 to 3 days, or in severe cases, up to 2 weeks.¹ The pronounced autonomic hyperreactivity signs and symptoms of delirium include hypertension, tachycardia, tachypnea, and tremors. If untreated, these can lead to respiratory and cardiovascular collapse. Neuropsychiatric indications of delirium include hallucinations, confusion, disorientation, and impaired

Table 1 Clinical manifestations of alcohol withdrawal syndrome and delirium tremens

Manifestation	Alcohol withdrawal syndrome	Delirium tremens
Time of occurrence after last drink, hours	24	48-72
Autonomic hyperactivity	Tremors Diaphoresis Nausea Vomiting Headache	Hypertension Tachycardia Tachypnea Tremors
Neuropsychiatric alterations	Agitation Anxiety Auditory disturbances Clouding of sensorium Tactile disturbances Visual disturbances	Hallucinations Confusion Delirium Severe disorientation Impaired attention

Table 2 Laboratory values useful in recognizing alcohol withdrawal syndrome

Parameter	Normal value	Value in patients with chronic alcohol use
Mean corpuscular volume or mean cellular volume, μm^3	82-98	Increased
Serum level of γ -glutamyl transferase, U/L	Men 4-25 Women 7-40	Increased
Serum level of uric acid, $\mu\text{mol/L}$ (mg/dL)	0.24-0.51 (4.0-8.5)	Increased
Carbohydrate-deficient transferrin, g/L (mg/dL)	2.0-3.8 (200-300)	Increased

attention. (See Table 1 for a summary comparison of the manifestations of AWS and delirium tremens.)

Laboratory values that may assist in recognition of AWS include mean corpuscular volume and serum levels of γ -glutamyl transferase, uric acid, and carbohydrate-deficient transferrin⁶ (Table 2). The values are abnormally increased with regular consumption of 180 to 240 mL (6-8 oz) of alcohol per day.⁵

Assessment Tools

As a part of assessment at the time of admission to the critical care unit, all patients should be screened for alcohol abuse. The usual assessment includes an alco-

hol use history that includes the frequency and quantity of regular alcohol consumption, how many years the use has been occurring, and the most recent intake.

One tool that has been suggested as an assessment questionnaire for alcohol use is the CAGE tool⁷ (Table 3).

Guidelines suggest that patients with a CAGE score greater than 2 or a score greater than 1 and at least 1 positive laboratory result (Table 2)

should be considered alcohol dependent and at risk for AWS.² The CAGE tool has been used in multiple studies and has documented reliability and validity in clinical settings. The advantage to using CAGE is that it is quick, easy to use, and easy to score.⁷

The Clinical Institute Withdrawal Assessment of Alcohol Scale, revised (CIWA-Ar; Figure 2) is a tool that can be used to objectively assess patients for the development of AWS. This scale has well-documented reliability and validity.⁸ It is a refined tool with a list of 10 signs and symptoms that is quick, easy to use, and useful in a variety of hospital settings, including psychiatric and general medical surgical units. The categories assessed in the CIWA-Ar include agitation, anxiety, auditory disturbances, orientation and clouding of sensorium, headache, nausea and vomiting, paroxysmal sweats, tactile disturbances, tremor, and visual disturbances.

The CIWA-Ar is used not only for diagnosis and monitoring but also as a basis for pharmacological management of patients with AWS. Scores of less than 8 to 10 on the CIWA-Ar indicate minimal to mild withdrawal.⁸ Scores of 8 to 15 indicate moderate withdrawal, and scores of 15 or more indicate severe withdrawal and impending delirium.⁸ By using the CIWA-Ar to assess patients, nurses

Table 3 The CAGE questionnaire⁷

- More than 2 positive responses to the questions suggest alcohol dependence.
1. Have you ever felt you ought to Cut down on drinking?
 2. Have people Annoyed you by criticizing your drinking?
 3. Have you ever felt Guilty about your drinking?
 4. Have you ever had a drink first thing in the morning (Eye opener) to steady your nerves or to get rid of a hangover?

CLINICAL INSTITUTE WITHDRAWAL ASSESSMENT OF ALCOHOL SCALE, REVISED (CIWA-AR)

Patient : _____ Date : _____ Time : _____ (24 hour clock, midnight = 00:00)

Pulse or heart rate, taken for one minute: _____ Blood pressure: _____

NAUSEA AND VOMITING - Ask "Do you feel sick to your stomach? Have you vomited?" Observation.

0 no nausea and no vomiting
1 mild nausea with no vomiting
2
3
4 intermittent nausea with dry heaves
5
6
7 constant nausea, frequent dry heaves and vomiting.

TREMOR - Arms extended and fingers spread apart. Observation.

0 no tremor
1 not visible, but can be felt fingertip to fingertip
2
3
4 moderate, with patient's arms extended
5
6
7 severe, even with arms not extended

PAROXYSMAL SWEATS - Observation.

0 no sweat visible
1 barely perceptible sweating, palms moist
2
3
4 beads of sweat obvious on forehead
5
6
7 drenching sweats

ANXIETY - Ask "Do you feel nervous?" Observation.

0 no anxiety, at ease
1 mildly anxious
2
3
4 moderately anxious, or guarded, so anxiety is inferred
5
6
7 equivalent to acute panic states as seen in severe delirium or acute schizophrenic reactions

AGITATION - Observation.

0 normal activity
1 somewhat more than normal activity
2
3
4 moderately fidgety and restless
5
6
7 paces back and forth during most of the interview, or constantly thrashes about.

TACTILE DISTURBANCES - Ask "Have you any itching, pins and needles sensations, any burning, any numbness, or do you feel bugs crawling on or under your skin?" Observation.

0 none
1 very mild itching, pins and needles, burning or numbness
2 mild itching, pins and needles, burning or numbness
3 moderate itching, pins and needles, burning or numbness
4 moderately severe hallucinations
5 severe hallucinations
6 extremely severe hallucinations
7 continuous hallucinations

AUDITORY DISTURBANCES - Ask "Are you more aware of sounds around you? Are they harsh? Do they frighten you? Are you hearing anything that is disturbing to you? Are you hearing things that you know aren't there?" Observation.

0 not present
1 very mild harshness or ability to frighten
2 mild harshness or ability to frighten
3 moderate harshness or ability to frighten
4 moderately severe hallucinations
5 severe hallucinations
6 extremely severe hallucinations
7 continuous hallucinations

VISUAL DISTURBANCES - Ask "Does the light appear to be too bright? Is its color different? Does it hurt your eyes? Are you seeing anything that is disturbing you? Are you seeing things that you know aren't there?" Observation.

0 not present
1 very mild sensitivity
2 mild sensitivity
3 moderate sensitivity
4 moderately severe hallucinations
5 severe hallucinations
6 extremely severe hallucinations
7 continuous hallucinations

HEADACHE FULLNESS IN HEAD - Ask "Does your head feel different? Does it feel like there is a band around your head?" Do not rate dizziness or lightheadedness. Otherwise, rate severity.

0 not present
1 very mild
2 mild
3 moderate
4 moderately severe
5 severe
6 very severe
7 extremely severe

ORIENTATION AND CLOUDING OF SENSORIUM - Ask "What day is this? Where are you? Who am I?"

0 oriented and can do serial additions
1 cannot do serial additions or is certain about date
2 disoriented for date by no more than two calendar days
3 disoriented for date by more than two calendar days
4 disoriented for place and/or person

The CIWA-Ar is *not* copyrighted and may be reproduced freely.
Sullivan, J.T.; Sykora, K.; Schneiderman, J.; Naranjo, C.A.; and Sellers, E.M.
Assessment of alcohol withdrawal: The revised Clinical Institute Withdrawal
Assessment for Alcohol scale (CIWA-Ar). *British Journal of Addiction* 84:1353-1357, 1989

Patients scoring less than 10 do not usually
need additional medication for withdrawal

Total CIWA-Ar Score _____

Rater's Initials _____

Maximum Possible Score 67

Figure 2 The Clinical Institute Withdrawal Assessment of Alcohol Scale, revised.

Available at: www.erowid.org/chemicals/alcohol/alcohol_ARF_withdrawal_scale.shtml. Accessed March 7, 2005.

can quantify the potential for the development of AWS and therefore initiate treatment for patients who require therapy. The CIWA-Ar is not copyrighted and can be downloaded from the following Web site: www.erowid.org/chemicals/alcohol/alcohol_ARF_withdrawal_scale.shtml.

Management of Patients With AWS

The treatment plan for patients with AWS includes pharmacological management and supportive care.

Pharmacological Management

Most signs and symptoms of alcohol withdrawal are caused by the rapid removal of the depressant effects of alcohol in the central nervous system. Although many pharmacological agents have depressant effects on the central nervous system, the cornerstone of pharmacological management for AWS patients is benzodiazepines. These drugs are the only agents that have been proved in placebo-controlled trials to reduce the severity of the effects of alcohol withdrawal and prevent progression to the serious complications of AWS.⁹ Benzodiazepines work by potentiating the responses of the GABAA receptors, thereby enhancing neuronal inhibition, or putting on the brakes. At this time, no specific benzodiazepine is recommended for use. The choice of agent to be used should be guided by the agent's duration of action, potential for abuse, and cost.⁹

Benzodiazepines with a short-half life (eg, oxazepam, with a half-life of 4-14 hours, or lorazepam, with a half-life of 10-20 hours) result in rapid changes of drug levels in the blood and may have to be given at an inter-

val of every 4 hours to avoid abrupt fluctuations of the drug levels, which may precipitate seizures. These short-duration agents may be particularly useful in patients with serious liver impairment or encephalopathy or when rapid control of signs and symptoms is needed. Agents with a longer duration of action (eg, diazepam, with a half-life of 20-90 hours, or chlordiazepoxide, with a half life of 24-48 hours) may be used because they maintain the therapeutic levels more evenly and consistently and so provide more effective control of signs and symptoms.

Another consideration in selecting an agent for treatment is the potential for abuse of the drug. The results of some clinical trials indicate that certain agents are preferred by patients with addictive disorders.⁸ Agents with a rapid onset of action, such as diazepam or lorazepam, have a higher abuse potential than do agents with a slower onset, such as chlordiazepoxide or oxazepam.

In the current healthcare environment, cost of pharmacological management is also a consideration. Table 4 includes the approximate average wholesale costs of some of the benzodiazepines.⁹

Because the goal of therapy is to alleviate the signs and symptoms of withdrawal, providing a fixed stan-

dardized dose of the selected benzodiazepine is not effective in treating alcohol withdrawal.⁸ Individualizing therapy according to the signs and symptoms (symptom-triggered therapy) of each patient results in administration of less medication and shorter treatment.⁸

The CIWA-Ar can be used to provide guidance and to monitor effective treatment with benzodiazepines. Monitoring should be completed by using the CIWA-Ar every 4 hours, and if the score is greater than 8 to 10, the selected benzodiazepine should be administered (Table 4). One hour after administration of the drug, the CIWA-Ar should be used again to determine if further medication is needed. Monitoring should continue until the CIWA-Ar score is less than 8 to 10 for 24 hours.⁸

Comorbid conditions and history of previous withdrawal seizures should also be considered when dosages of benzodiazepines are determined. The goal is to administer enough of the drug to alleviate the signs and symptoms and then to decrease the dose as the signs and symptoms abate.

Thiamine is another pharmacological agent recommended in the treatment of patients with AWS. The purpose of thiamine replacement is

Table 4 Common benzodiazepines used to treat patients with alcohol withdrawal syndrome

Drug	Standard dose		Recommended dose range for patients with alcohol withdrawal syndrome, mg
	Amount, mg	Cost, cents	
Chlordiazepoxide	25	3	50-100
Diazepam	5	7	10-20
Lorazepam	1	11	2-4
Oxazepam	15	25	10-30

Table 5 Effects of alcohol on the body and supportive care for patients with alcohol withdrawal syndrome

System affected/ cancer risk	Effect	Mechanism	Nursing interventions
Central nervous system	Impaired judgment and memory Impaired balance and motor coordination Sleep disturbances Peripheral neuropathy Alcohol-related psychiatric disorders Dementia Depression Hallucinations Wernicke encephalopathy Korsakoff syndrome	Effects are due to the combination of the direct toxic effects of alcohol on neural tissue, thiamine deficiency, and nutritional deficits	Maintain patient's safety Provide quiet environment Have patient avoid television and other activities that may contribute to hallucinations Initiate interventions to prevent falls
Cardiovascular	Beneficial effects Possible reduction in risk for death due to cardiovascular conditions (1-2 drinks per day over long periods) Deleterious effects Decreased myocardial contractility Cardiomyopathy Dysrhythmias Mild to moderate hypertension	Beneficial effects occur because alcohol increases levels of high-density lipoprotein and decreases platelet aggregation Deleterious effects are due to the direct toxic effects of alcohol on cardiac muscle and the vasopressor effects of ethanol	Monitoring patients for dysrhythmias Provide rest to reduce fatigue Monitor for hypertension
Hematopoietic	Increased size of red blood cells (mean corpuscular volume) Decreased production of white blood cells Thrombocytopenia	Effects are due to the direct toxic effects of alcohol on bone marrow that cause suppression and malnutrition-related folate deficiency	Monitor blood values and bleeding Do a complete nutritional assessment Encourage patient to eat nutritiously Maintain a safe environment and initiate interventions to avoid injury Prevent infection
Gastrointestinal	Esophageal inflammation Mallory-Weiss lesions Esophageal varices Acute pancreatitis Alcohol-induced hepatitis Cirrhosis	Effects occur because alcohol stimulates an increase in acid production and causes direct damage of the gastric mucosal barrier; cirrhosis occurs as a result of fatty buildup in the liver	Monitor patient for gastrointestinal bleeding Monitor for alterations in liver function
Musculoskeletal	Alcoholic myopathy Lower bone density Increased risk of fractures	Effects on muscle tissue are due to the direct toxic effects of alcohol on muscle tissue. Skeletal effects are due to the direct toxic effects of alcohol on osteoblasts	Maintain a safe environment Initiate interventions to prevent falls
Cancer risk	Increased risk for cancer of the Breast Esophagus Oral cavity Overall risk of cancer is 10 times greater in persons who are alcoholic than in the general population ⁶	Alcohol is not a direct-acting carcinogen but one of its metabolites, acetaldehyde, may act as a tumor promoter. Chronic alcohol use also causes increased degradation of retinol by the liver and results in vitamin A deficiency, which is associated with an increased incidence of cancer	Explain effects of chronic alcohol use Offer information on treatment options and sources of support

to avoid the severe and irreversible complications (as discussed earlier) that can occur as a result of thiamine

deficiency. A dose of 50 to 100 mg of thiamine is given parenterally or orally daily for 3 days or more.⁵

Supportive Care

The effects of alcohol on the body should be taken into consideration

in supportive care (Table 5). Reducing environmental stimuli and providing uninterrupted periods of rest can help minimize the severity of signs and symptoms. Use of restraints should be avoided because they may worsen the neuropsychiatric alterations. Orientation should be provided as necessary with clocks and calendars. Use of television may contribute to a patient's confusion and hallucinations, particularly if the program is unfamiliar to the patient. Because malnutrition is a concern with patients with AWS, providing adequate nutritional support is important. Monitoring fluid balance is important because fluid retention occurs as a result of the inhibition of vasopressin secretion, which occurs with increases in blood alcohol levels.¹ In order to help reduce the stress of critical illness, which can exacerbate AWS, the concomitant medical disorder that brought the patient to the critical care unit should be aggressively treated.

Discharge planning should include encouragement to seek treatment for addictive disease. A nonjudgmental explanation of the effects of alcohol abuse and the possible complications can help patients make appropriate behavioral changes. Patients who abuse alcohol may be noncompliant with self-care management and are at risk for chronic AWS complications and increased likelihood of experiencing injury or trauma. Psychiatric support services and referrals are appropriate for follow-up.

Summary

Because alcohol abuse and alcohol dependency are widespread, many critical care patients may have AWS.

AWS has marked consequences that affect the care of these patients, and critical care nurses must actively assess and intervene to prevent or at least minimize the complications of AWS. By increasing awareness, using reliable assessment tools, and knowing guidelines for the management of these patients, nurses can reduce the consequences of overlooking AWS and mismanaging patients who have the syndrome.

Acknowledgment

The author wishes to thank Kathy Kaminski, RN, for her encouragement and assistance in identifying the focus for this article.

References

1. Schuckit M. Alcohol and alcoholism. In: Brunwald E, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL. *Harrison's Principles of Internal Medicine*. Vol. 2. 15th ed. New York, NY: McGraw-Hill Professional Publishing; 2001:2561-2566.
2. *Alcohol: What You Don't Know Can Harm You*. Bethesda, Md: US Dept of Health and Human Services, National Institute on Alcohol Abuse and Alcoholism; 2001. NIH publication 96 4153.
3. Finn DA, Crabbe JC. Exploring alcohol withdrawal syndrome. *Alcohol Health Res World*. 1997;21:149-156.
4. Bridges KJ, Trujillo EB, Jacobs DO. Alcohol-related thiamine deficiency and malnutrition. *Crit Care Nurse*. December 1999;19:80-85.
5. Tierney LM, McPhee SJ, Papadakis MA, eds. *Current Medical Diagnosis and Treatment 2003*. 42nd ed. New York, NY: Appleton & Lange; 2003.
6. Chernecky CC, Berger BJ, eds. *Laboratory Tests and Diagnostic Procedures*. 3rd ed. Philadelphia, Pa: WB Saunders Co; 2001.
7. Bisson J, Nadeau L, Demers A. The validity of the CAGE scale to screen for heavy drinking and drinking problems in a general population survey. *Addiction*. 1999;94:715-722.
8. Jaeger T, Lohr R, Pankratz S. Symptom-triggered therapy for alcohol withdrawal syndrome in medical inpatients. *Mayo Clin Proc*. 2001;76:695-701.
9. Mayo-Smith MF. Pharmacological management of alcohol withdrawal: a meta-analysis and evidence-based practice guideline. American Society of Addiction Medicine Working Group on Pharmacological Management of Alcohol Withdrawal. *JAMA*. 1997;278:144-151.

CE Test Test ID C0532: Alcohol Withdrawal Syndrome: Overlooked and Mismanaged?

Learning objectives: 1. Describe pathophysiology involved between alcohol and alterations of neurotransmission in the brain 2. Define the relationship between alcohol and thiamine deficiency 3. Examine the management of patients undergoing alcohol withdrawal syndrome

1. What percentage of the population between the ages of 18 and 64 years meet the diagnostic criteria for alcohol dependency?

- a. 11%
- b. 18%
- c. 27%
- d. 34%

2. What are the mortality and morbidity rates of people who abuse alcohol compared to the general population?

- a. 1 to 3 times greater
- b. 2 to 4 times greater
- c. 2 to 5 times greater
- d. 1 to 2 times greater

3. What is the function of the neurotransmitter γ -aminobutyrate A?

- a. Allows more chloride to inhibit the cell
- b. Regulates calcium to inhibit the cell
- c. Allows less chloride to excite the cell
- d. Regulates calcium to excite the cell

4. What is the function of the neurotransmitter N-methyl-D-aspartate?

- a. Allows more chloride to inhibit the cell
- b. Regulates calcium to inhibit the cell
- c. Allows less chloride to excite the cell
- d. Regulates calcium to excite the cell

5. What is the term used to describe the effect resulting in hypersensitivity of the limbic system and brain stem when abrupt cessation of alcohol occurs?

- a. Rebound stimulatory
- b. Rebound depression
- c. Rebound hypersensitivity
- d. Rebound irritability

6. What are the 3 types of tolerances that develop with long-term alcohol use?

- a. Metabolic, cellular, and behavioral
- b. Metabolic, cognitive, and social
- c. Cognitive, cellular, and behavioral
- d. Behavioral, cognitive, and metabolic

7. Which vitamin deficiency plays a key role in long-term alcohol dependency?

- a. Vitamin A deficiency
- b. Vitamin B₁ deficiency
- c. Vitamin B₆ deficiency
- d. Vitamin C deficiency

8. In what metabolism does thiamine play a key role?

- a. Fat
- b. Protein
- c. Glucose
- d. Amino acid

9. What is the effect of thiamine deficiency on peripheral nerves?

- a. Myelin degeneration
- b. Myelin regeneration
- c. Excitation
- d. Depression

10. What are the 2 syndromes related to thiamine deficiency on the heart?

- a. Wernicke and Korsakoff
- b. Cardiomyopathy and hypertension
- c. Wernicke and cardiomyopathy
- d. Korsakoff and hypertension

11. What are the 2 syndromes related to thiamine deficiency on the brain?

- a. Wernicke and Korsakoff
- b. Cardiomyopathy and hypertension
- c. Wernicke and cardiomyopathy
- d. Korsakoff and hypertension

12. What is the cornerstone for pharmacological management of patients with alcohol withdrawal syndrome?

- a. Amphetamines
- b. Benzodiazepines
- c. Opiates
- d. Placebo

Test answers: Mark only one box for your answer to each question. You may photocopy this form.

- | | | | | | | | | | | | |
|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|--------------------------------|--------------------------------|--------------------------------|
| 1. <input type="checkbox"/> a | 2. <input type="checkbox"/> a | 3. <input type="checkbox"/> a | 4. <input type="checkbox"/> a | 5. <input type="checkbox"/> a | 6. <input type="checkbox"/> a | 7. <input type="checkbox"/> a | 8. <input type="checkbox"/> a | 9. <input type="checkbox"/> a | 10. <input type="checkbox"/> a | 11. <input type="checkbox"/> a | 12. <input type="checkbox"/> a |
| <input type="checkbox"/> b | <input type="checkbox"/> b | <input type="checkbox"/> b |
| <input type="checkbox"/> c | <input type="checkbox"/> c | <input type="checkbox"/> c |
| <input type="checkbox"/> d | <input type="checkbox"/> d | <input type="checkbox"/> d |

Test ID: C0532 Form expires: June 1, 2007. Contact hours: 1.5 Fee: \$11 Passing score: 9 correct (75%) Category: A Test writer: Jane Baron, RN, CS, ACNP

AMERICAN ASSOCIATION of CRITICAL-CARE NURSES

Mail this entire page to:
AACN
101 Columbia
Aliso Viejo, CA 92656
(800) 899-2226

Program evaluation

	Yes	No
Objective 1 was met	<input type="checkbox"/>	<input type="checkbox"/>
Objective 2 was met	<input type="checkbox"/>	<input type="checkbox"/>
Objective 3 was met	<input type="checkbox"/>	<input type="checkbox"/>
Content was relevant to my nursing practice	<input type="checkbox"/>	<input type="checkbox"/>
My expectations were met	<input type="checkbox"/>	<input type="checkbox"/>
This method of CE is effective for this content	<input type="checkbox"/>	<input type="checkbox"/>
The level of difficulty of this test was:		
<input type="checkbox"/> easy <input type="checkbox"/> medium <input type="checkbox"/> difficult		
To complete this program, it took me _____ hours/minutes.		

Name _____ Member # _____

Address _____

City _____ State _____ ZIP _____

Country _____ Phone _____ E-mail address _____

RN License #1 _____ State _____

RN License #2 _____ State _____

Payment by: Visa M/C AMEX Check

Card # _____ Expiration Date _____

Signature _____