

ALCOHOL WITHDRAWAL MANAGEMENT IN THE ACUTE CARE SETTING

Clinical Policy and Procedure

Approved: July 2008

Next Review: July 2011

Go directly to:

[Clinical Institute of Withdrawal Assessment for Alcohol \(CIWA-A\)](#)
[Alcohol \(ETOH\) Withdrawal CIWA/Medication Administration Record](#)
[Acute Care Alcohol Withdrawal Orders](#)
[Alcohol Withdrawal Seizure Prophylaxis Orders](#)
[Signs/Symptoms](#)
[When to notify physician](#)

Purpose

To assist with early identification and management of the patient at risk for alcohol withdrawal syndrome (AWS) in the acute care setting and prevent negative physical and/or psychosocial consequences of the withdrawal experience.

Policy

Patients admitted to SMC, who exhibit signs/symptoms (S/S) of alcohol withdrawal or with known history of alcohol abuse, are evaluated for AWS. If AWS is confirmed, the patient receives prompt and appropriate medications to minimize the withdrawal symptoms.

PROCEDURE

1. Assess signs/symptoms.
 - a. **Early/Mild withdrawal signs of AWS:**
 - Anxiety
 - Mild diaphoresis
 - Hyperalert, may startle easily
 - Mild itching, vague pins and needles under skin
 - Shakes and jitters with movement
 - Nausea, anorexia
 - b. **Moderate withdrawal signs of AWS:**
 - Tremors at rest
 - Diaphoresis, especially face, palms
 - Tremors at rest, worsens with movement
 - Increased anxiety and emotional lability
 - Vomiting
 - Clouding of orientation, increased sensitivity to touch
 - Elevated heart rate and/or blood pressure (*Note:* If patient has known hypertension or is on BP meds, vital signs might not indicate AWS)

c. **Severe withdrawal signs/symptoms of AWS (delirium tremens)**

- Tachypnea, fever
- Total body tremor
- Profuse diaphoresis
- Extreme agitation, paranoia
- Complete disorientation
- Gastric pain
- Continuous hallucinations, inability to distinguish from reality

2. Rule out other physiologic conditions for patient's presenting symptoms (acute changes in sensorium-agitation, disorientation).

- Fingertick blood sugar looking for hyper/hypoglycemia
- Pulse oximetry looking for hypoxia
- BMP ordered by the licensed independent practitioner (LIP) looking for metabolic disturbances

3. Review patient history for any history of seizures. Collaborate with the LIP for seizure prophylaxis.

4. Notify the LIP of onset of signs and symptoms (S/S) and/or need to initiate the [Acute Care Alcohol Withdrawal Orders](#).

5. The RN implements the [Acute Care Alcohol Withdrawal Orders](#).

6. Patient/family education needs to include:

- Daily alcohol consumption causes physiologic dependence.
- If alcohol consumption is stopped, withdrawal signs/symptoms may occur.
- Inform patient and family of the S/S of alcohol withdrawal syndrome.
- Inform the patient that the signs and symptoms he or she is exhibiting resemble alcohol withdrawal.
- Medication is needed to relieve these symptoms and prevent acute withdrawal symptoms.
- Inform patient that assessment of CIWA will occur during sleep hours

8. Obtain vital signs and CIWA score according to the LIP order:

CIWA Score	VS and CIWA Score Frequency Wake patient if necessary
5 – 10	Every 4 hours
11 – 14	Every 2 hours
15 – 25	Every 1 hour
Greater than 25	Reassess in 15 minutes

9. Medicate patient based on CIWA score according to the LIP order.

10. Ensure patent IV site as ordered by the LIP.

11. If respiratory rate (RR) is 12 per minute or less, call the LIP.

12. Obtain pulse oximetry every shift and PRN. If SaO₂ is less than 92%, start oxygen at 2 L/min. Notify the LIP to obtain oxygen order.

13. If the patient has a seizure:

- Administer lorazepam ordered by LIP. Give lorazepam IM if no IV access.
- Pulse oxygen saturation, If SaO₂ is less than 92%, start oxygen at 2 L/min. Notify the LIP to obtain oxygen order.

14. Initiate patient safety measures to prevent self-injury.

- Elevate HOB 30 degrees if the patient is somnolent, to prevent aspiration.
- Implement fall precautions. See [Fall Prevention](#).

15. Notify the LIP if:
- Patient exhibits acute changes in sensorium.
 - CIWA score continues to increase or does not decrease after medication administration.
 - Patient has seizure, difficult to arouse, or RR below or equal to 12 per minute.
 - Patient's pulse greater than 120, less than 50.
 - Patient's systolic pressure above 180 mmHg, less than 90 mmHg.
 - Patient's temperature above 101.5° F (38.5° C).
 - Patient's requesting AMA discharge.
 - Patient's SaO₂ stays less than 92% on 2 L/min or airway compromise.
16. Request that the LIP evaluate the patient within one hour, and consider ICU transfer if:
- Patient has seizure.
 - Patient's level of sedation needs closer monitoring.
 - CIWA score is above 15 for more than four hours.
 - Patient has received 6 mg of lorazepam in one hour, or a total of 15 mg of lorazepam in four hours, or a total over 200 mg of chlordiazepoxide in four hours has been administered.
- **If the LIP is unable to see the patient within one hour, contact the rapid response team (RRT).**
17. Consult with Care Coordination for discharge planning.
18. Collaborate with the LIP regarding physician-to-physician consult with addiction medical physician.
19. If AWS assessment is below 5 and no medication has been administered in 48 hours, consult with LIP about discontinuing the alcohol withdrawal orders.
-

Definitions

Front-loading regimen. Scheduled doses of long-acting benzodiazepine (chlordiazepoxide) to provide smoother, less symptomatic withdrawal experience.

Wernicke's encephalopathy is a syndrome characterized by ataxia, ophthalmoplegia, confusion, and impairment of short-term memory. It is often a result of inadequate intake or absorption of thiamine (vitamin B1), especially in conjunction with carbohydrate ingestion. Its most common correlate is prolonged alcohol consumption resulting in thiamine deficiency. Alcoholics are therefore particularly at risk, but it may also occur with thiamine deficiency states arising from other causes, particularly in patients with such gastric disorders as carcinoma, chronic gastritis, and repetitive vomiting.

Korsakoff's syndrome is a condition that mainly affects chronic alcoholics. It is due to the direct effects of alcohol or to the severe nutritional deficiencies that are associated with chronic alcoholism, and has been seen in association with vitamin B deficiency. The syndrome also occurs in other severe brain disturbances (e.g., paralysis, dementia, brain damage, infections and poisonings). In chronic alcoholism the condition usually occurs following delirium tremens.

The syndrome is characterized by a severe memory defect, especially for recent event, for which the patient compensates by confabulation, the reciting of imaginary experiences. Psychologists working with these patients often have great problems sorting out what is truth and what are lies of what patients say; this is story-telling on a high level. Other symptoms may include delirium, anxiety, fear, depression, confusion, delusions and insomnia, painful extremities, sometimes bilateral wrist drop, more frequent bilateral foot drop with pain or pressure over the long nerves. Confabulation, a requisite for the classic syndrome, is not always present in mild conditions. Polyneuropathy may or may not be present.

Propylene glycol (PG) toxicity. Propylene glycol is a colorless, odorless liquid frequently used as a vehicle for IV medications, such as lorazepam, or other drugs, such as diazepam, digoxin (Lanoxin), or phenytoin, multivitamins, and even cosmetics. Of medications containing PG, lorazepam has the highest proportion at 80%. Approximately 45% of PG is eliminated by the kidneys, while the remaining 55% is metabolized to lactic acid, pyruvic acid, or acetone by hepatic alcohol dehydrogenase. PG is osmotically active and can cause an elevation in serum osmolality. Although PG toxicity has not been well studied, case reports have described CNS depression, cardiac arrhythmias, respiratory depression, hemolytic anemia, hyperosmolality, lactic acidosis, increased anion gap, and renal dysfunction in patients receiving agents that use PG as a vehicle for drug delivery. Patients on IV lorazepam in large doses (greater than 0.1 mg/kg/hr) for extended periods of time (greater than 48 hours) are at risk for developing propylene glycol toxicity.

Forms

- ◆ [Acute Care Alcohol Withdrawal Orders](#)
- ◆ [Alcohol Withdrawal Seizure Prophylaxis Orders](#)
- ◆ [Alcohol \(ETOH\) Withdrawal CIWA/Medication Administration Record](#)

Supplemental Information

The brain maintains neurochemical balance through inhibitory and excitatory neurotransmitters. The main inhibitory neurotransmitter is gamma aminobutyric acid (GABA) which acts through GABA-alpha (GABA-A) neuroreceptor. One of major excitatory neurotransmitters is glutamate, which acts through N-methyl-D-aspartate (NMDA) neuroreceptor.

Alcohol enhances the effects of GABA, leading to decreased overall brain excitability. Chronic exposure to alcohol results in the compensatory decrease of GABA-A neuroreceptor response to GABA, evidenced by increasing tolerance to the effects of alcohol

Abrupt cessation of alcohol exposure results in brain hyperexcitability because receptors previously inhibited by alcohol are no longer inhibited. Brain hyperexcitability manifests clinically as agitation, anxiety, irritability, and tremors. Severe manifestations include seizures and delirium tremens.

Minor withdrawal symptoms can occur while the patient still has a measurable blood alcohol level.

Withdrawal **seizures** are more common in patients who have a history of multiple episodes of detoxification.

Patients at high risk of experiencing alcohol withdrawal syndrome (AWS) include:

- Patients admitted to hospital with elevated blood alcohol level
- Patients with known history of AWS/delirium tremens (DTs)/seizures
- Patients who admit to daily intake of alcohol

Signs/symptoms begin 4-8 hours after last alcoholic drink and peak within 48-72 hours. Symptom management is the most important strategy in the management of the patient experiencing alcohol withdrawal. Episodes of delirium tremens have a mortality rate of 1 to 5 percent.

Long-term alcohol dependency leads to thiamine deficiency. Thiamine plays a key role in glucose metabolism. The major organs affected by thiamine deficiency are the peripheral nerves, heart and brain. Peripheral neuropathy with myelin degeneration, cardiomyopathy, hypertension, Wernicke encephalopathy, and Korsakoff syndrome are results of chronic thiamine deficiency.

Patients requiring parenteral fluids should be given thiamine (100 mg) before glucose is administered to prevent precipitation of Wernicke encephalopathy.

Patients at high risk of experiencing seizures during AWS may be started on seizure prophylaxis medications. (See [Alcohol Withdrawal Seizure Prophylaxis Orders](#)).

Using the CIWA-A scale, the clinical picture should be considered because medical and psychiatric conditions may mimic alcohol withdrawal symptoms. In addition, certain medications (e.g., beta blockers) may blunt the presentation of withdrawal symptoms.

Expert Consultants

Jim Walsh, MD
Addiction Recovery Service (ARS) nursing staff

Author

Ann McElroy, RN, MSN, AOCN, BC

Regulatory Requirement

Not applicable.

References

- Mayo-Smith, M.F., Beecher, L.H., Fischer, T.L., et al. (2004). Management of alcohol withdrawal delirium. Archives of Internal Medicine, 164, pp. 1405-1412.
- McKinley, M.G. (2005). Alcohol withdrawal syndrome. Critical Care Nurse 25(3), pp. 40-49.
- Becker, K., & Semrow, S. Standardizing the care of detox patients to achieve quality outcomes. Journal of Psychosocial Nursing, 44(3), pp. 33-38.
- deCarolis, D.D., Rice, K.L., Ho, L., et al. (2007). Symptom-driven lorazepam protocol for treatment of severe alcohol withdrawal delirium in the intensive care unit. Pharmacotherapy, 27(4), pp. 510-518.
- Phillips, S., Haycock, C., & Boyle, D. (2006). Development of an alcohol withdrawal protocol. Clinical Nurse Specialist, 20(4), pp. 190-197.
- Wojtecki, C.A., Marron, J., Allison, E.J., et al. (2004). Systematic ED assessment and treatment of alcohol withdrawal syndromes: A pilot project at a Veteran Affairs Medical Center. Journal of Emergency Nursing, 30(2), pp. 134-140.

Addenda

1. [Clinical Institute of Withdrawal Assessment for Alcohol \(CIWA-A\)](#)