Anesthetic management of the alcoholic patient

LOLA O’DANIEL, CRNA
Albuquerque, New Mexico

This article encompasses a brief background of the physical effects of alcohol—both in acute intoxication and in the chronic alcoholic. Also covered is an explanation of the process of intoxication, blood alcohol concentration and its pertinence in the anesthetic management of the acutely intoxicated. The author includes a section on the anesthetic management of both the chronic alcoholic and the patient who is acutely intoxicated.

Alcoholism occurs on all levels of society. The instance in recent times of two persons related to men serving in our nation’s highest office publicly admitting to having problems with alcoholism—Betty Ford and Billy Carter—serves to illustrate that the “skid row bum” holds no exclusivity on the condition.

- As a matter of record, one in nine persons in the United States is an alcoholic.
- More than 10,000,000 people in this country suffer from alcoholism.
- The life span of the alcoholic is 10 to 12 years shorter than the national average.
- There is a higher mortality rate found among alcoholics having other medical problems.
- In males between 45 to 65 years old, alcohol is the fourth leading cause of death, cirrhosis of the liver is fifth.
- The general personality characteristics of the alcoholic are: a depressive, high-pressure person, compulsive, impatient, and a heavy smoker.
- Thirty percent of hospitalized patients have a problem with alcohol abuse.¹

Effects of acute intoxication

The effects of acute intoxication are relative to the chronicity of the individual’s alcohol intake and resultant organ damage. You may be alerted to the degree of liver damage, cerebral damage or cardiomyopathy from the patient’s history, physical examination and laboratory studies. However, when you must give an anesthetic to a patient with a Blood Alcohol Concentration (BAC) of 300 mg%, the following are some of the main factors to consider.

Cerebrum. Alcohol is a ganglionic depressant rather than an excitant. The larger neurons are depressed first. As the BAC rises, the automatic functions of the brain are affected. Depending on the BAC, more and more of the inhibition originating in the frontal lobes is removed until the drinker reverts to an infantile state.² Though the patient may be happy due to his loss of inhibition, he may become enraged easily if he feels he is being harassed. All varieties of personalities can manifest in the patient with an elevated BAC. You must not only realize that that is not his normal behavior, but that he must be approached gingerly.

Post-binge headache. After an alcohol binge, four factors create the “hangover headache.”
1. Direct irritation of the meninges by the breakdown products released in the metabolism of alcohol.

2. Prolonged vasodilatation caused directly by the alcohol and directly related to the amount ingested.

3. Dehydration due to the diuretic effect of alcohol. This causes loss of fluid from the extracellular compartment and the spinal fluid system; in this aspect it is similar to the "spinal headache."

4. Edema of the brain relative to the amount of fluid ingested with the alcohol and the confusion of the fluid shifts in the compartments.

5. Head or spinal cord injury.

6. Drug overdose.

Fluid and Electrolyte Changes. In acute intoxication, the body undergoes the following: (1) the sodium level drops due to a large ingestion of fluid with alcohol; (2) diuresis due to alcohol takes place; (3) magnesium wasting results; (4) alterations in plasma osmolarity present; (5) metabolic acidosis occurs; and (6) there is a severe phosphate depletion syndrome.

Circulatory system. Alcohol causes vasodilation, and in turn, there is a decrease in blood pressure and an increase in pulse rate. Therefore, you see the "hallmark of the alcoholic"—the flushed face.

Process of alcohol intoxication
The actual process of alcohol intoxication consists of:

1. Tranquilization and sedation.
2. Mood changes.
3. Psychomotor retardation.
4. Reflex slowing.
5. Lethargy and sleep.
6. Coma and occasionally death.

Blood alcohol concentration (BAC) is measured in milligrams of alcohol per 100 cc of blood. For measurement purposes, one drink equals 12 oz beer or 4 oz table wine or 2.5 oz fortified wine or sherry or 1 oz whiskey, vodka or other distilled spirits.

If a 45 kg person consumes four drinks in one hour, his BAC will be 130 mg%. (As an example of what this means, in the State of New Mexico, driving while intoxicated (DWI) is any BAC greater than 100 mg%. A BAC of 80 mg% is DWI in many other states.)

If a 70 kg person consumes four drinks in one hour, his BAC will be 80 mg%.

In the 70 kg person, 120 cc of absolute alcohol given intravenously slowly will produce Stage III anesthesia. With this anesthesia, there is a narrow range before respiratory depression occurs. The calculated BAC here would be 320 mg% (equivalent to eight drinks in 30 minutes). Since the metabolism rate of alcohol is slow, it would take 17 hours for this person to eliminate the alcohol.

Treatment of acute intoxication
Stimulants have been used to treat acute intoxication, but have generally been abandoned because they may precipitate convulsions and because they have a transient effect.

Dialysis can eliminate alcohol from the body. This is useful in the treatment of children with an elevated BAC. Considering the small body weight of children, they can easily have a BAC at a toxic level.

In the non-surgical situation, time and supportive measures are the treatment of choice in acute intoxication.

In the surgical situation where the patient presents with an elevated BAC (especially in the case of the multiple trauma patient—usually the victim of a motor vehicle accident), more rapid changes occur. The necessary fluid and blood replacement needed throughout the procedure essentially dialyzes the patient. Quite likely, the patient will proceed to the recovery room with a greatly lowered BAC—a rather brutal way to achieve sobriety.

Complications of acute intoxication
Complications of acute intoxication are related to whether this inebriation is the first or one of multiple inebriations. The severity of the intoxication is measured by the BAC. The chronic alcoholic can tolerate a higher BAC than the first-time inebriate.

The complications of acute intoxication are more severe in children as their BAC will rise more sharply due to their smaller body weight. Death during acute intoxication occurs in a relatively large number of alcoholics.

Severe alcohol intoxication can mask other disease processes:

1. Head or spinal cord injury.
2. Drug overdose.
Hypoglycemia, especially with malnutrition.
4. Diabetic coma.
5. Hepatic coma.
8. Exposure hypothermia.
9. Overwhelming infection.
10. Cardiac arrhythmias.
11. Suicide attempts.
12. Epilepsy.

**Effects of chronic alcohol intake**

*Neurological complications.* These complications can be related to acute intoxication, withdrawal symptoms or nutritional deficiencies.

Epilepsy results when cerebral lesions have occurred due to chronic alcohol intake. There is an increase in the severity of the seizures with intoxication and when delirium tremens occur. Treatment of this epilepsy is alcohol abstinence which may improve the EEG reading and decrease the number of seizures.

Cerebellar cortical degeneration can result from chronic exposure of the nervous system to toxic levels of acetaldehyde which is a breakdown product in the metabolism of alcohol.

The Wernicke-Korsakoff syndrome can occur in the alcoholic who is in a depleted nutritional state. The symptoms are (1) mental confusion, (2) ataxia, (3) abnormal ocular motility, and (4) polyneuropathy. The treatment of Wernicke-Korsakoff syndrome is nutritional repletion, abstinence from alcohol, and administration of thiamine (Vitamin B1).

Amblyopia can be caused by alcoholism and nutritional depletion. This is worsened by the concomitant use of tobacco.

Memory loss is due to actual destruction of the cortical brain cells from a prolonged elevated BAC.

Delirium tremens (DTs) occur in alcohol withdrawal or reduction. Symptoms of impending DTs are tremor, tachycardia, clouding of sensation, nausea, and vomiting. Symptoms of frank DTs are delirium with clouding of sensation, visual hallucinations and moderate to marked signs and symptoms associated with alcohol withdrawal.

Acute alcoholic hallucinosis can occur in withdrawal. Symptoms are auditory hallucinations, and mild clouding of sensorium with few other symptoms of toxicity withdrawal.

Alcohols are at an increased risk of (1) stroke, (2) hypertensive encephalopathy, (3) Encephalitis, and (4) masked brain tumor or abscess.

*Hepatic complications.* Hepatic complications from chronic alcohol intake include alcoholic fatty liver, alcoholic hepatitis, and alcoholic cirrhosis. These complications can progress in that order; if the fatty liver and the alcoholic hepatitis have been neglected, cirrhosis occurs.

Cirrhosis is the fatty infiltration of the liver plus cellular necrosis and scarring. The scarring causes compression of the portal circulation. Complications of cirrhosis include:

1. Jaundice.
2. Ascites.
3. Edema.
5. Esophageal varices.
7. Anemia.

*Renal complications.* In the chronic alcoholic, there is an increased incidence of glomerulonephritis. This is the result of increased susceptibility to disease and the toxic effects of alcohol on the kidneys.

*Pancreatic complications.* Pancreatitis occurs in the chronic alcoholic because the elevated BAC simultaneously stimulates secretion by the pancreas while causing contraction of the sphincter of Oddi. This disease is difficult to distinguish clinically from alcoholic gastritis.

*Cardiac complications.* Alcoholic cardiomyopathy symptoms are inappropriate tachycardia, frequent extrasystoles, and angina-like chest pain. This disease can progress to congestive heart failure.

Alcoholic cardiomyopathy plus nutritional deficiency, especially lack of thiamine, will result in beriberi heart disease.

*Circulatory complications.* The alcoholic has an increased occurrence of hypertension, and may have decreased clotting ability due to malabsorption of Vitamin K and a decrease in factor VII. He may have alcohol-induced thrombocytopenia.

Lactacidemia occurs in the alcoholic. Lactate is a breakdown product in the metabolism of alcohol. Sustained high levels of lactate can progress to a severe refractory acidotic state. The lactacidemia causes secondary hyperuricemia which can cause gout. Treatment of this gout is alcohol withdrawal. Allopurinol will achieve no relief in alcoholic gout.

Hypoglycemia occurs in the chronic alcoholic with the intake of alcohol. Alcohol generally causes hyperglycemia; however, the alcoholic has a depletion of his glycogen stores. Therefore, with alcohol intake and stimulation of the carbohydrate metabolism mechanism, the chronic alcoholic proceeds into hypoglycemia.
The Rumpel-Leede sign may alert the anesthetist to a nutritional depletion state in the alcoholic. The Rumpel-Leede sign is the appearance of minute subcutaneous hemorrhages below a tourniquet within ten minutes.

Gastro-intestinal complications. Cancer of the esophagus occurs more often in whiskey drinkers than in wine drinkers due to the direct irritation. Esophageal varices as a complication are secondary to cirrhosis and portal hypertension.

In the stomach, gastric secretion is altered during alcohol intake. The secretions are stimulated similarly to histamine release; there is increased acidity with reduced pepsin secretion. The altered gastric secretion predisposes the alcoholic to gastritis. Direct irritation by the alcohol also contributes to gastritis. Alcoholics have an increased incidence of peptic ulcer disease.

Complications of the respiratory tract. Respiratory tract complications are related to the fact that the alcoholic has (1) a decreased resistance, (2) malnutrition, (3) an increased incidence of tobacco use, (4) depression of the cough reflex, and (5) an increased incidence of aspiration.

A husky voice in the alcoholic is due to direct irritation by alcohol and smoke. He is more prone to pneumonia, lung abscess, bronchiectasis, and tuberculosis. The alcoholic is more prone to pleural effusions secondary to congestive heart failure or rupture of the esophagus during vomiting.

Complications of pregnancy. Fetal alcohol syndrome is a set of anomalies found in children born of mothers who drank alcohol during pregnancy. There can be a variety within the range of anomalies in the fetal alcohol syndrome. The most striking is severe mental and growth retardation.

This syndrome can occur in the mother who is a "social drinker," but perhaps had one binge at the wrong time in the development of the fetus. It can occur if the mother is hospitalized for a threatened abortion and intravenous alcohol is used to halt the abortion. And, of course, it is most likely to occur in the mother who routinely drinks alcohol daily.

Psychiatric complications. Among alcoholics there is a high percentage of depressed people. They seem attracted to alcohol as a way to drug themselves, thereby creating a feeling of well-being.

Psychiatric complications in the "wet" alcoholic range from psychoses of jealousy, hallucinatory phases (the classic pink elephant), paranoia, depressive psychoses, and possible suicide in the confused and depressed state.

During withdrawal, unfortunately, the alcoholic's underlying depressed state is exposed. Confusion and marked deterioration of his mental state may be related to actual brain damage as well as withdrawal. Paranoia or a depressive reaction may occur during withdrawal. If the alcoholic is eligible for the Antabuse® (disulfiram) program, he may need accompanying psychotherapy. In withdrawal, again, he is a high risk candidate for suicide.

Basic metabolism of alcohol

Alcohol is absorbed through the stomach or by inhalation through the lungs. The rate of absorption is affected by the concentration of alcohol, the regional blood flow, and the absorbing surface.

Factors that slow absorption are: food in the stomach, the type of beverage consumed (the straight shot versus the Pina Colada), simultaneous effects of drugs, and any factor affecting gastrointestinal motility.

Once absorbed, 90-95% of the alcohol is oxidized in the liver. An adult can metabolize 7 gm/hr. Alcohol is oxidized in the liver by alcohol dehydrogenase. Native Americans have a genetic deficiency of alcohol dehydrogenase: therefore, this group has a greater problem with alcoholism than other ethnic groups. (This is especially pertinent to anesthetists in the Southwest area of the United States where one-third of the population is Native American.)

Ethanol is oxidized by alcohol dehydrogenase to acetaldehyde. The acetaldehyde is catalyzed by aldehyde dehydrogenase to acetate and acetyl coenzyme A.

Alcohol is eliminated by excretion via the breath, urine, and sweat. The longer the BAC remains elevated, the more likely some of the alcohol will be eliminated unchanged from the body. However that 90-95% of absorbed alcohol must be metabolized by the liver at the fixed rate of 7 gm/hr. Therefore, the effects of alcohol are prolonged and the return of the BAC to zero is slower.

It should be pointed out that methanol or wood alcohol ingestion is very toxic. Methanol ingestion can be treated by ethanol. Absolute alcohol given intravenously in doses to maintain a BAC of 100 mg% will occupy the alcohol dehydrogenase in metabolism. The methanol will then be excreted unchanged through the kidneys.

Enzyme induction and cross tolerance

Alcohol can cause enzyme induction but this...
is quite variable among individuals (apparently this enzyme induction is under genetic control). Where alcohol has produced enzyme induction there is an increase in the effectiveness of the detoxifying pathway; this can include the detoxifying of alcohol, sedatives, tranquilizers, hypnotics or narcotics. Therefore, you see a decrease in clinical response to such drugs in some alcoholics.

Although an alcoholic may have enzyme induction from alcohol, if he has liver damage as well, this will inhibit alcohol or drug detoxification. Consequently, you will see an increase in clinical response to these drugs.

The anesthetist should be wary of more than enzyme induction in the chronic alcoholic who requires surgery and anesthesia while he is still intoxicated. Since alcohol causes enzyme suppression, the alcoholic is more sensitive to anesthetics, narcotics, and tranquilizers.

_Cross tolerance_ is the result of alcohol, sedatives, tranquilizers, other abused drugs, hypnotics, and anesthetics following a similar metabolic pathway through the liver. Where an increased effectiveness of the detoxifying pathway has occurred, you will see a decreased response to these drugs.

**Blood volume and BAC**

The larger the blood volume of an individual, the more alcohol he can hold. The degree of inebriation as measured by the BAC is reviewed in Table I.

The lethal dose of alcohol in adults is 5-8 gm/kg. In children the lethal dose is 3 gm/kg. Without food slowing absorption death may occur with less than 5 gm/kg. in an adult. Post mortem BAC's have been found at levels from 450-700 mg%.

The chronic alcoholic can tolerate and survive a higher BAC than the occasional binge drinker.

**Drug and alcohol interactions**

_Antabuse®_ (disulfiram) is used for aversion therapy in the binge drinking alcohol abuser. Di-
sulfiram blocks the metabolism of acetaldehyde, therefore, the level of acetaldehyde in the blood rises with alcohol intake. When alcohol is ingested there is an "acetaldehyde reaction" with the undesirable symptoms of vasodilatation, flushing, nausea and vomiting, and hypotension.

Other drugs causing this acetaldehyde reaction include chloramphenicol (Chloromycetin®); furazolidonol (Furoxone®); griseofulvin (Fulvicin®); metronidazole (Flagyl®); and quinacrine (Atabrine®).

The monamine oxidase (MAO) inhibitors are sometimes used in the treatment of depression. To review an old story: the depressed person on MAO inhibitors begins to feel better, decides to celebrate, buys a bottle of wine, some bread and cheese, and ends the evening in a hypertensive crisis. The MAO inhibitors block the metabolism of the catecholamines.

Tyramine, found in some wines and ripe cheeses, as well as ergot and decayed animal tissue, is the culprit when combined with MAO inhibitors. Tyramine is structurally related to epinephrine and norepinephrine, and when in combination with MAO inhibitors, precipitates a hypertensive crisis.

Alcohol in combination with any of the sedatives, hypnotics, tranquilizers, narcotics, and antihistamines will have an increased effect in depressing the central nervous system.

**Anesthetic management of the acutely intoxicated**

Factors to consider in the anesthetic management of the acutely intoxicated patient (besides the surgical condition) are:

1. A full stomach.
2. The patient's teeth—the anesthetist should personally inspect the patient's teeth, as a polite question as to their condition will not necessarily yield a reliable answer.

<table>
<thead>
<tr>
<th>BAC levels and their effects</th>
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<tbody>
<tr>
<td><strong>20 mg %</strong> Light drinkers begin to feel some effect. BAC after one drink.</td>
</tr>
<tr>
<td><strong>40 mg %</strong> Most people feel relaxed.</td>
</tr>
<tr>
<td><strong>60 mg %</strong> Judgment is somewhat impaired.</td>
</tr>
<tr>
<td><strong>80 mg %</strong> Impairment of motor skills. Drunk while driving (DWI) in some states.</td>
</tr>
<tr>
<td><strong>100 mg %</strong> Deterioration of reaction time. DWI in New Mexico.</td>
</tr>
<tr>
<td><strong>120 mg %</strong> Vomiting occurs unless this level is reached slowly.</td>
</tr>
<tr>
<td><strong>150 mg %</strong> Balance and movement is impaired. One-half pint of whiskey is in the blood.</td>
</tr>
<tr>
<td><strong>300 mg %</strong> Loss of consciousness in some.</td>
</tr>
<tr>
<td><strong>400 mg %</strong> Most lose consciousness—some die.</td>
</tr>
<tr>
<td><strong>450 mg %</strong> Breathing stops.</td>
</tr>
</tbody>
</table>
3. Decreased blood pressure and increased pulse rate due to the alcohol.
4. Diuresis.
5. The patient may be approaching metabolic acidosis.
6. Wide mood swings—ridiculously happy to combative.
7. The patient is probably a heavy smoker.
8. He or she may be a drug abuser.
9. Consider the BAC.

In approaching the anesthetic management of the acutely intoxicated, we should consider:

The rapid sequence induction versus the awake intubation: anesthetists know the awake intubation is the prime choice in this situation. However, most anesthetists lean toward the technique they find works best for them. The technique used depends on the skill of the anesthetist and the patient's degree of cooperation.

IV fluids: Dextrose 5% with Ringer's lactate plus plasmanate calculated hourly per the patient's needs will halt the fluid shifting that occurs due to the intoxication. This will hold the fluid in the extracellular compartment where it is more manageable and usable for our evaluation of kidney function and hourly fluid needs.

Balanced anesthesia: Here, it should be remembered that the patient has anesthetized himself with alcohol and possibly anything else he happened upon (Valium®, barbituates, or possibly street drugs). The anesthetic needs of such patients vary widely. Although the patient may have a cross tolerance to anesthetic agents, while he is intoxicated, he is in enzyme suppression. Blood gas readings will help the anesthetist evaluate whether or not the alcohol intake has progressed to metabolic acidosis.

Because alcohol and anesthesia can cause prolonged respiratory depression and because the patient has a full stomach, leave him intubated and support his respirations postoperatively.

Case report. A 55-year-old male, 87 kg, BAC 225 mg%, was involved in an automobile accident on a Friday night after participating in a "Happy Hour." His injuries included: a ruptured spleen, tear in the diaphragm, fractures of the left femur, left radius and ulna, and facial lacerations. Upon arrival to the operating room he was very talkative, cooperative and congenial.

Anesthesia management included a rapid sequence induction and, a light dose balanced anesthesia.

The patient was left intubated postoperatively, with respiratory support provided by a ventilator. His only preoperative recall was lighting his pipe just before the accident and then later that of his wife removing his wedding ring in the emergency room. His first recall postoperatively was awakening Saturday afternoon. The only alcohol related pathology he had was increase in liver size.

Anesthetic management of the chronic alcoholic

Besides his surgical condition, the chronic alcoholic facing surgery generally has the following complications.

1. Malnutrition, possibly accompanied by overhydration, is the case if the patient had been drinking up until hospital admission. He has a decreased resistance to infection and will have delayed postoperative healing.
2. Liver damage: The extent of the liver damage should be documented by enzyme studies preoperatively in the elective surgery case.
3. Because most alcoholics have a history of tobacco use, preoperative pulmonary teaching may help avoid postoperative pulmonary complications.
4. Cardiomyopathy: The patient may have a history of chest pain. You can expect that he will have arrhythmias on the ECG monitor during surgery. Quite likely he has cardiac arrhythmias constantly.
5. Renal disease: Be aware that the patient is a good candidate for glomerulonephritis.
6. Neurological complications: These may be related to intoxication, withdrawal or neuropathy. Impending DTs should be treated. Frank DTs should be avoided; they may undo the surgery, as in fracture stabilization for example.
7. Cross tolerance may be present unless there is liver damage to the extent that the patient is sensitive to the anesthetic agents.

Regional anesthesia may not be the best anesthetic choice for the alcoholic. The personality of the alcoholic during the stress of facing surgery may lead the anesthetic choice away from regional anesthesia to general anesthesia. Psychologically, the patient may not be able to cope with the idea of "being awake" as well as facing surgery. Also, general anesthesia can postpone DTs for 24 hours at least.8

The alcoholic is more prone to traumatic injury and subsequent surgery.

Intraoperative management. Valium® or Librium® is indicated, pre-, intra- and post-operatively to allay apprehension and prevent DTs.

If DTs are as imminent as surgery is necessary, an intravenous infusion of alcohol by continuous administration may be indicated—administering
the anesthetic agent in addition to the intravenous alcohol.³

The chronic alcoholic patient has stressed himself over a long period of time and now he faces the additional stress of surgery. If anesthetic management becomes difficult the anesthetist should perhaps think in terms of adrenal-cortical support or augmentation.

A new facet in the medical-legal trends today. If an alcoholic is admitted for surgery and his alcoholism is not recognized, whereupon he proceeds through surgery and develops DTs—his surgery is undone. This patient can sue the medical personnel involved in his case for negligence: alcoholism being categorized as a disease.¹

Case report. A 50-year-old female, 55 kg in weight, was admitted for an elective abdominal hysterectomy. She had a long history of alcohol and polydrug abuse. She had cardiomyopathy and frequent hospital admissions for chest pain during alcohol binges.

She had been on Antabuse® for two months prior to admission. She seemed to be in optimal health as she faced this surgery.

Anesthesia management consisted of very light dose NLA; she apparently had no cross tolerance to the anesthetic. Postoperatively she had no recall of the procedure and progressed smoothly through the hospitalization to return home.

Alcoholism: A disease, not a moral issue

With alcoholism, we have a situation in dealing with a group of patients where almost anything could go wrong. These patients we should "dust with a feather" as nurse anesthetist educator Helen Vos taught us, then experience tells us we had better "have a sledgehammer on the cart as well."

Even though our dealings with alcoholics are generally very short term, we can still be responsible for recognizing a possible drinking problem and communicating this suspicion or knowledge to the persons responsible for the patient's medical management. Sometimes the anesthesiology interviews with the patient do cover areas that are missed in a general workup.

Through communication with the patient and those responsible for his medical management, we can perhaps influence some of these alcoholic patients in the direction of treatment on their primary disease—alcoholism.

REFERENCES

AUTHOR

Lola O'Daniel, CRNA, is a graduate of the Johns Hopkins Hospital School of Nursing, Baltimore, Maryland, and the Barnes Hospital School of Anesthesia, St. Louis, Missouri. She is now a staff anesthetist at the Bernalillo County Medical Center, Albuquerque, New Mexico.

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