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Ethanol Emergencies and Their Treatment

The acutely intoxicated patient is a frequent and high-risk visitor to the emergency department. Such patients may present with challenging complications, such as ketoacidosis or encephalopathy, as well as in various stages of alcohol withdrawal. The clinical presentations and treatment for these common, yet frequently challenging, conditions will be discussed. This course will be presented in a case-based format, and 15 to 20 unknowns will provide ample opportunity for audience interaction.

- Discuss the complications of alcohol abuse.
- Differentiate the clinical presentations of alcohol-related emergencies and discuss their management.
- Recognize the spectrum of alcohol withdrawal and determine the appropriate management.

MO-37
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3:00 PM - 3:55 PM
Room # N242
Las Vegas Convention Center

FACULTY

Corey M Slovis, MD, FACEP

Professor, Emergency Medicine and Medicine; Chairman, Department of Emergency Medicine, Vanderbilt University Medical Center, Nashville, Tennessee

Overview

This handout is an overview of the most important acute emergencies that alcohol abusers may experience. I have tried to summarize the key facts, concepts and controversies. Please remember some general concepts when dealing with alcohol abusers.

Secure the ABC's and follow all 5 vital signs closely.
Always consider toxic-metabolic imbalance.
R/O infection.
Always consider a CNS etiology.
Never assume AMS in an alcoholic is just due to ETOH

I will return to these 5 concepts in more depth beginning in the section concerning **Altered Mental Status in the Alcoholic.**

General Facts on Blood Alcohol Levels

- **Each ounce of whiskey, glass of wine, or can of beer raises blood ETOH by 25 mg %**
- **One ml/kg of pure ethanol increases blood ETOH by 100 mg% at 2 hours.**
- **ETOH level of 100 - 150 mg% = intoxication.**
- **LD„ = 500 mg% (in untrained drinkers only)**
- **Blood ETOH falls at about 25 mg% per hour.**

Lesser Known Facts

- One ounce (30 ml) of 100 proof ETOH may raise a I year old (15 kg) child's serum level to 100 mg%.

- Novice Drinkers may be intoxicated at levels of 50 mg%.

- Chronic abusers are often alert and ambulatory at 400 - 600 mg%.

- Metabolism of ETOH may double in chronic abusers for short periods of time.

THE FOUR STAGES OF ALCOHOL WITHDRAWAL

Stage I: **Tremulousness**
6 - 12 hours post-cessation

Stage II: **Hallucinosis**
1 - 2 days post-cessation

Stage III: **Withdrawal Seizures**
1 - 2 days post-cessation

Stage IV: **Delirium Tremens**
3 - 5 days post-cessation

STAGE I: TREMULOUSNESS

- Seen only after cellular tolerance to chronic ingestion develops
- A withdrawal phenomena
- A hypersympathetic state
- Similar to hyperthyroidism
- Often self-treated with a drink
- Self-limiting
- 1 - 5% will progress to DTs if untreated

Treatment of Tremulousness/Early Withdrawal

The key to treatment is to detoxify the patient over 3 - 5 days. Shorter time intervals (i.e., 1 day) often result in rebound; longer programs require more than just the ED (i.e., inpatient or outpatient detoxification programs). If you want to maximize therapy with minimal risk:

Aggressively treat patient in ED
Don't provide more than 3 - 5 days of outpatient medication
Get patient into detox program

ED treatment of Alcoholic Tremulousness:

1. **Benzodiazepine**
2. **Nutritionally Replete**
   a. **Feed**
   or
   b. **Start IV alcohol cocktail**

For intravenous therapy, it is important to provide:

1. **Volume**
2. **Glucose**
3. **Multi-vitamins and Thiamine**
4. **Potassium**
5. **Magnesium**

Volume Repletion:

- Most alcoholics will be somewhat volume depleted due to nausea, vomiting, poor nutritional and hydration status, and alcohol's diuretic effects.
- If patient is not orthostatic or tachycardic D/C ASAP after medicating
- If patient is going to be in the ED for a few hours:
  - Start D_5NSS at 200 cc/hr
    - **Add: (1) MVI-(l-2 amps)**
      (2) Folate (1 - 5 mg)
      (3) KCL (40 meq)
      (4) $MgSO_4$ (5 grams)
      (5) Thiamine (100 mg IV when IV started)

Alcohol Cocktail

D_5*NSS (at 200 cc/hr)
MVI (1 amp)
Folate (1 - 5 mg)
Potassium (40 meq)
Magnesium (5 grams)

***Give 100 mg thiamine to** any alcoholic receiving glucose*

*Note: 200 cc/hr will usually take into account increased irreversible losses of a hypersympathetic state. These are my biases; there is no "right way"; however, you will decrease withdrawal symptoms and possibly **withdrawal seizures** with the $MgSO_4$; two amps of MVI will provide the right amount of niacin (100 mg) to avoid **pellagra**; and the KCI will help avoid **hypokalemic induced arrhythmias and rhabdomyolysis.***

If patient is dehydrated: Resuscitate!!!
Don't reverse dehydration gradually.

Glucose and Thiamine

Glucose is needed to avoid starvation ketosis (AKA); alcoholics have little or no glycogen stores; mild hyperglycemia is OK-their insulin levels are low and will take 2 - 6 hours to rise. Never treat hyperglycemia with insulin (unless DKA or insulin is above 500 - 600). Beware Wemicke's.

Thiamine -- *(Wernicke's discussed in detail on pages 12 - 13)*

- **Give 100 mg IV push**
- **IV is very safe**
- **Give to all alcoholics, malnourished, cachetic patients**
- **Give after glucose in emergencies; NOT before**

Benzodiazenines and/or Barbiturates (or other cross-tolerant medication)

I used to think librium was "the drug of choice. " Based on extensive experience of innumerable centers and experts. I can now say conclusively that the drug of choice is any one that you use safely, carefully, effectively and for 3 - 5 days. Pick a drug, learn its dose, $T_{½}$, side effects, cost and your choice is as good as anyone's.

Remember:

Give only 3 - 5 days of medication at time of D/C.
Do not undermedicate.
Multiply nonalcoholic's dose by 2 - 4 x for an alcoholic
Do not overdose already sedatedpatients.
Do not overdose liver failure patients.
Taper the dose over 3 - 5 days.

Phenobarbital

For ED patients, initial dosing may be with: 65 mg - 260 mg PO, IV, IM

Initial Drug and Doses in ETOH Withdrawal

	Dose	Route	t½
Librium	*75 - 100* mg	PO only	16 hrs
Valium	5 - 10 mg	PO, IV	Patients age
Ativan	1 - 2 mg	PO, IV or IM	12 hrs
Phenobarbital	*60 - 260* mg	PO, IV, IM	*3* - 5 days
Alcohol	1 - 2 drinks 100 cc/hr D_5E10	PO IV	A few hours A few hours

The key is calming the patient down enough to leave,
but not calming him down enough
to sleep in his "new home" for the next 8 24 hours!

For Discharge: (These are only some of the more common protocols)

Librium	Chlorazepate (Tranxene)
100 mg X QID Day I	15 mg X QID Day 1
75 mg X QID Day 2	15 mg X TID Day 2
50 mg X QID Day 3	15 mg X BID Day 3
25 mg X QID Day 4	15 mg X QD Days 4 - 5

Ativan	Phenobarbital
1 mg QID Day 1	30 mg TID Day 1
1 mg TID Day 2	30 mg BID Day 2
1 mg BID Day 3	30 mg QD Day 3 (or 3 - 5)
1 mg QD Day 4 (or 4 - 5)	

Note: Many programs do not use Valium. Although it is effective, patients may become quite dependent on its "pleasurable" CNS effects. The serum level of benzodiazepines doubles at I hour if ETOH consumed concomitantly.

The CNS depressant effects of ETOH, benzodiazepines and barbiturates are synergistic.

The most important reason to treat tremulousness with a cross-tolerant drug is to prevent withdrawal seizures and delirium tremens. Therapy for withdrawal may take up to 7 - 14 days for complete detoxitation; ED's cannot do this!

Incidence of Tremulousness Progressing to Withdrawal Seizures:

No treatment:

No prior seizures	Less than 1%
Prior seizures	About 10%

With treatment:

No prior seizures	Close to 0%
Prior seizures	0 - 5% (reports variable)

Progression of Tremulousness and Seizures to D.T.'s

No treatment: 5 - 30%	(New vs old reports highly variable)
With treatment: 1 - 6%	(D.T.'s are becoming quite rare)

Magnesium

The role of magnesium is not yet clearly defined. However, what I have listed below are the general comments. There is no data to suggest magnesium is harmful. The data is supportive magnessium's role in preventing withdrawal, withdrawal seizures, and D. T.'s.

Magnesium in Alcohol Withdrawal

- Decreases incidence of seizures
- Decreases tremulousness
- Decreases arrhythmias during withdrawal
- Decreases potassium losses
- Increases efficacy of Thiamine
- Corrects total body deficit

Treatment of Hypomagnesemia in ETOH withdrawal should be based on:

Total body deficit is IO - 20 grams
Takes 5 - 7 days to replete

Give:

2 grams over 20 - 60 minutes
5 grams Q 8H IV
Discharge on oral therapy

Also give:

Food, KCI, MVI, Folate, Thiamine

Serum Magnesium Levels Prior to Therapy Are:

•Silly
***Expensive**
***Unreliable**

Nutritionally Replete

The key is to provide enough vitamins, minerals, and carbohydrates to replenish stores but not do it so well as to open a soup kitchen. Alcoholics are deficient in everything, but we focus on (1) Thiamine, (2) Niacin, (3) Folate, (4) Magnesium, and (5) Glycogen.

Nutritional Repletion in ETOH Abuse

Thiamine	100 IV push on ED entry
Niacin	In IV (Need about 100 mg; use MVI)
Folate	In IV (Need about 1 mg; *use* MVI)
Magnesium	5 grams in IV (10 cc of 50% $MgSO_4$)
Glycogen	Feed patient (or least give D_5 in IV)

STAGE II: HALLUCINOSIS

Please note: Stages II and III (Hallucinosis and Withdrawal Seizures) overlap in time frame and some authors reverse II and III. I use Stage II for hallucinosis because many more patients present with tremulousness (Stage I) and hallucinations (Stage II). If treated properly, they should not usually "progress" to Stage III, Withdrawal Seizures.

- Seen in up to 25% of long time drinkers who withdraw
- Auditory more common than visual
- Visual misinterpretation most common
- Visual up to 5X more common than auditory hallucinations
- May be threatening in nature
- Auditory more benign and less likely to progress to DTs

Treatment

- See treatment for tremulousness above
- For patients who are agitated and getting more tremulous due to their hallucinations, use Haldol 5 mg IM Q 30 - 60 minutes.
- Haldol has never been shown to lower the seizure threshold in clinical studies using humans on low-medium doses.

STAGE III: WITHDRAWAL SEIZURES

This is a hot topic with somewhat confusing data (on first glance); it is not really that complicated and the bottom line is Dilantin used to be thought to be effective. The more recent literature says it is not. I used to strongly advocate using Dilantin for those patients with a prior history of ETOH seizures but who hadn't yet seized. Now I only use it for alcoholics who have had seizures not believed to be related to ETOH or withdrawal.

Classic Alcohol Withdrawal Seizures

Usually 1 - 6
Tonic-Clonic
Self-limited
Nonfocal
Nonstatus

Note: May be status: 3%
May be focal: 3 - 20%
May progress to DTs if not properly medicated
Are the number one cause of Status Seizures in some hospitals

There are a number of details that are very important to keep in mind.

Seizure Frequencies (From Victor, Epilepsia 1967)

1 **Seizure:**	**40%**
2 **Seizures:**	**24%**
3 **Seizures:**	**15%**
4 **Seizures:**	**10%**
5 - 12 **Seizures:**	**10%**
status:	**3%**

Status seizures or focal seizures in ETOH withdrawal may be indicative of major problems or nothing at all. ***It is impossible to tell by history and physical exam what status or focality mean. Therefore:***

Focal Seizure	**= CT scan (may be focal with prior cortical disease)**
Febrile Seizure	**= LP after CT. Start antibiotics STAT.**
Status Seizure	**= CT scan and metabolic screen**

There are a few relatively new studies that should be noted.

CT Scans of "Alcohol Withdrawal Seizures." Earnest, et al. Neurology 1988:

259 patients all got CT scans
All seizures generalized and thought due to ETOH
No focal, trauma comatose or metabolic disorder patients
Results: 16 patients (6.2%) had intracranial lesions!
8 had subdurals!

You can't tell whether a "withdrawal seizure" is really a withdrawal seizure by history or physical.

Focality of ETOH withdrawal seizing. Earnest and Yarnell. *Epilepsia* 1976.
Hauser, et al. *Epilepsia* 1988.

Early studies quoted 3% of seizures to be focal; in these studies approximately 20% of seizures were focal.

You cannot tell without the CT. If a neurologist tells you he or she can, get another neurologist.

The Role of Dilantin (DPH) in Alcohol Withdrawal Seizures (AWS)

1971: Sampliner *JAMA*

157 patients Librium and placebo and DPH (100 mg TID x 5 d)
11 seizures with no DPH
0 seizures with DPH
This study proved DPH worked great; unfortunately on closer exam years later, the study was not well controlled and had a number of confounding variables.

1973: Rothstein *Am J Psychiatry*

200 patients; Librium vs Librium and DPH (200 mg BID x 5 d)
No seizures in either group
This study supported benzodiazepines with or without DPH.

1973-1988: Some giving DPH to all alcoholics
Some discharging patients on DPH forever

1989: Alldredge, Lowenstein, Simon *Am J Med*

90 patients after one or more AWS
45 no therapy (no $MgSO_4$, Benzo) vs 45 DPH (1000 mg IV)
6 seizures with no prophylactic therapy
6 seizures with DPH

1991: Chance. *Ann Emerg Med*

55 patients after one AWS
15 mg/kg of IV DPH vs placebo
No patients received $MgSO_4$ or Benzo
5 of 27 seized with placebo
6 of 28 seized with DPH

1994: Rathler, et al. *Ann Emerg Med*

100 patient after one AWS
15 mg/kg IV DPH vs placebo
Patients did receive 1 gram $MgSO_4$ and MVI
12 of 5 l seized with placebo
10 of 49 seized with DPH

Conclusions on Dilantin for AWS:

1. DO NOT use it routinely
2. It has no role *if* no prior seizures
3. It has no role if one or more AWS have already occurred
4. It probably has little or no role in AWS
5. All the data is NOT in

Who gets Dilantin:

1. Seizure etiology unclear
2. Post CNS trauma
3. Drinkers with Idiopathic Epilepsy

Lorazepam in Withdrawal Seizures

NEJM 1999;340:915-919

- 186 pts: Lorazepam vs Placebo
- 2 mg of Ativan vs 4 cc of NSS

- 3% of Lorazepam Pts Re-Seized (3/100)
- 24% of Placebo Pts Re-Seized (21/86)
- P < 0.001; odd ratio = 10.4

Treatment of Status Seizures in Alcoholic Withdrawal (most seizures):

1. **Secure ABC's**
2. **Evaluate for NGT**
3. **Begin henzodiazepine titration**
4. **R/O reversible causes**
5. **Begin barbiturate titration**

Drugs for Status Seizures in AWS:

- **Begin Benzodiazepine titration**
- **(Valium 5 mg Q 1 - 5 min or Ativan 1 - 2mg Q 5 min)**
- **Begin magnesium infusion: 2 grams over 20 minutes**
- **For uncontrollable patients: Begin barbiturate titration**
  - **Phenobarbital 100 mg Q 1 min or**
  - **Pentothal 50 - 100 mg Q 1 minute**

* *If you are not an expert in airway management (and/or Anesthesia is not present). also STAT page hospital attorney and your travel agent.*

Remember: *Barbiturates plus benzodiazepines plus ETOH abuse often equals Respiratory Arrest **and/or Hypotension**. However, if done properly, you can stop seizures. intubate, control patient, and do a rapid CT under controlled conditions.*

Alcohol Seizures vs AWS. **Hauser et al *Epilepsia* 1988**
Ng et al *NEJM* 1988

Alcoholics seize from drinking too. This group of authors studied a large number of ETOH abusers in NYC:

308 patients, 294 controls
16% seizures in ETOH abusers not in AWS time period
60% of seizures were random events in patients still drinking
Frequency of seizures increased as quantity of ETOH increased

3x normal if ½ pint/day
8x normal if 1 pint/day
20x normal if 1 quart/day

STAGE IV: DELIRIUM TREMENS

The DTs are a medical emergency that should be feared Therapy of any alcoholic should be directed toward avoiding this disease entity. Older studies talked of up to 1/4 of ETOH abusers developing DTs but the incidence is now quite low. Exact numbers are hard to come by, but somewhere between I - 5% ofpatients who go through withdrawal progress to DTs. The key to avoiding DTs is prevention by aggressive benzodiazepine (or barbiturate) therapy, rehydration, and nutritional repletion of magnesium, vitamin, mineral and carbohydrate stores.

REMEMBER in DTs:

Mortality should be less than 6% with aggressive therapy
Mortality will be 15 - 20% in patients who have an underlying disease

Beware: ***Pneumonia, GI Bleed, Sepsis, CNS Trauma, Meningitis***

Stage IV - Delirium Tremens:

1. **Delirium** Coma = head trauma, hypoglycemia, toxic/metabolic cause
   Tremor Seizures = CT to R/O other causes

2. **Vital sign abnormalities**

DT's:	*DT's if present*:
Hypertension	Hypotension = dehydration
Tachycardia	Bradycardia = overmedicated; elevated ICP
Hyperventilation	Hypoventilation = overmedicated; elevated ICP
Hyperthermia	Hypothermia = sepsis, CNS trauma, Wemicke's

Treatment of DTs

- Secure ABCs
- Sedation*
- Volume 250 - 500 cc/hr. (Follow v.s. and urine output - use Foley)
- Glucose D_5NS alternating with D_5 ½NS
- IV Potassium at least 10 - 20 meq/liter (follow serum K -- see page 20)
- Magnesium 10 grams in first day (see pages 6 and 21)
- Thiamine 100 mg IV daily
- Multivitamins and Folate (see p. 6)
- Follow: Vital Signs
  Lytes
  Urine output

Sedation in DTs: Titrated benzodiazepine and beta blocker (i.e., 5 mg Valium Q 5 - 10 minutes and 0.5 IV Inderal Q 5 - 10 minutes). Use beta blocker only after multiple doses of Valium and/or significant tachyarrhythmias. IV midazolam has been used to treat DTs. It has a much shorter half-life than Valium. Be *careful,* it may cause respiratory depression when used at too high a dose or when used by the inexperienced.

MORTALITY IN DT's

- Oversedation
  - aspiration
  - respiratory arrests
  - arrhythmias
- Electrolyte Abnormalities
  - arrhythmias
  - seizures

WERNICKE'S ENCEPHALOPATHY

The classic triad of Wernicke's is:

1) Ataxia
2) Ocular findings (nystagmus or lateral rectus palsy)
3) Encephalopathy

Less common but reported symptoms which may mask the classic triad arc:

1) Coma
2) Miosis
3) Hypothermia
4) Hypotension
5) Bradycardia

Thus an ETOH abuser in coma with pinpoint pupils, a core temperature of 92°F with a pulse of 50 and a systolic or 70 may be just an exposure victim or may have 5/5 unusual signs for Wemicke's!

Wemicke's develops in ETOH abusers because of: Poor thiamine intake; poor thiamine absorption in the GI tract; and poor storage of thiamine in the liver. Thiamine is converted to thiamine pyrophosphate, a co-enzyme for the pentose-phosphate shunt and the tricarboxylic acid cycle. Lack of it destroys different areas of the brain, including the cerebellum **(ataxia);** the hypothalamus and brain stem **(coma, hypotension, bradycardia),** the mamillary bodies, memory and the vestibular system **(nystagmus).**

Wemicke's is preventable, but once it develops 10 - 20% of patients die. Eighty percent of survivors develop **Korsakoffs Psychosis,** a retro and antegrade amnesia. Suspicion or risk of Wemicke's requires 100 mg IV thiamine and 100 mg x 3 days if possible. True Wemicke's requires up to 1,000 mgs of thiamine in the first 24 hours, magnesium and nutritional support.

Remember: ***Patients with AIDS, those on hyperalimentation, any patient with chronic malnutrition, poorly controlled diabetes and those with anorexia nervosa are all at risk for thiamine depletion.***

There was some controversy over the use of IV thiamine. We have shown (Ann Emerg Med 1989) that it is very safe given IV push. Use it aggressively. Prevention of Wernicke's is the key.

> ***Patients rarely have the classic triad** of **Wernicke's.***
> *Usually only one or two signs arepresent.*
> ***Be Suspicious!***

IV THIAMINE-INDUCED ANAPHYLAXIS

There has been one report (Am *J Emerg Med* 1992) of anaphylaxis in the American literature in the past 30 years. Grady Hospital in Atlanta, Denver General Hospital in Colorado, and Bcllevue Hospital in New York City have given more than 500,000 IV injections of thiamine over the past 15 years without a problem.

l/500,000 for IV Thiamine equals at most a 0.0002% chance of an allergic reaction. Compare that to a 1 - 10% chance of allergic reaction for penicillin (.04 - .27% of true anaphylaxis), 2 - 3% chance of IVP dye, or 1 - 18% with Streptokinase.

ACIDOSIS IN THE ALCOHOLIC:

Alcoholics often present to the ED mildly acidotic.

The differential diagnosis of a wide gap metabolic acidosis requires knowledge of four concepts:

1. **Anion Gap (AG)**

   - $AG = Na - (HCO_3 + Cl) = 5 - 15$

2. **Osmolar Gap (OG)**

   - Calculated osmolarity = 2 x Na + Glu/18 + BUN/2.8
   - OG = true osmolarity - calculated osmolarity = 10 - 20

3. **Osmolar forces contributed by alcohols:**

Ethanol	Divide mg % by 4.4 = mosm (or multiply osmolar gap by **4.4 = mg%** Ethanol)
Methanol	Multiply osmolar gap by 3.2 = mg% of Methanol
Isopropyl Alcohol	Multiply osmolar gap by 6.0 = mg% of Isopropyl Alcohol
Ethylene Glycol	Multiply osmolar gap by 6.2 = mg% of Ethylene Glycol

Note: A simpler set of numbers to memorize is **3, 4, 6** for these toxins. It is not as accurate, but it puts you in the ballpark.

ALCOHOLIC KETOACIDOSIS

- Not yet completely described or understood
- Not uncommon
- Develops in chronic ETOH abusers

  Five requirements of AKA:

  - Dehydration
  - Low glycogen stores
  - A precipitating event
  - No available carbohydrate
  - No available alcohol

AKA:

- Protein and fat break down
- ↑ Lactate and betahydroxybutyrate levels
- A low bicarbonate (5 - 15)
- A low serum pH (7.10 7.35)
- Borderline glucose values

Treatment of Alcoholic Ketoacidosis

1. Hydrate and give Glucose (D_5NS 200 cc/hr)
2. Treat withdrawal (Benzodiazepine, $MgSO_4$, etc.)
3. Thiamine, multivitamins, folate, Niacin
4. Food
5. Treat underlying cause: Pancreatitis, CNS event, sepsis, pneumonia, GI bleed, etc.

KEYS TO GOOD CARE

- There is no role for bicarbonate in AKA (unless the pH is below 7.0, the R/O other causes of acidosis).
- There is no role for insulin in AKA (even with hyperglycemia unless glucose above 500).
- Follow K, PO, and venous pH values.
- If pH falls once treatment has begun, this is NOT just AKA!
- If serum phosphorus falls to below 1.5 - 1:

1. Give 1/4 of potassium requirement as K_2PO_4

2. K_2PO_4 = 4.4 meq of K per cc of K_2PO_4; 3 mmol of PO, per cc

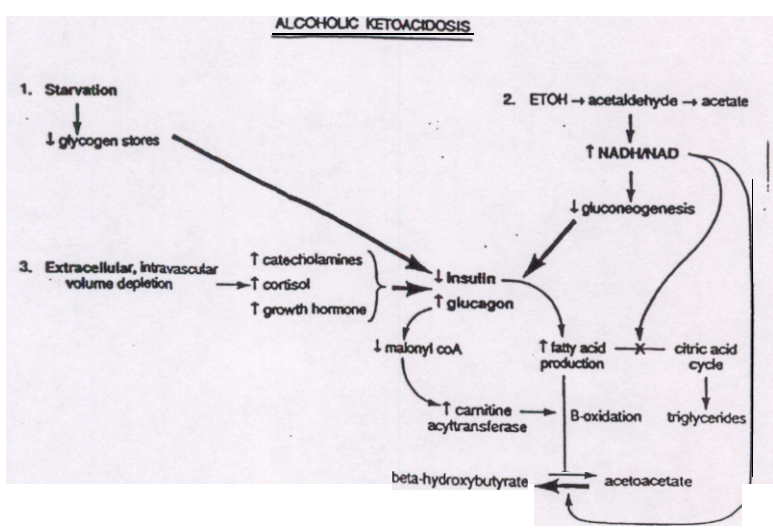

Diagram courtesy of Keith Wrenn, M.D.

METHANOL

- CH_3-OH; MW 32; each 3.2 mg% of methanol = 1 mosm
- Creates metabolic roadblocks in: CNS, Eye, Abdomen
- Diagnosis: **Profound acidosis, blindness, retinal edema, pancreatitis**
- Alcohol blocks breakdown of methanol to formic acid and formaldehyde
- Methanol is non-toxic; its breakdown products are not
- **If congested with ETOH, acidosis and symptoms delayed**

GENERAL TREATMENT OF OVERDOSES

1. ABCs
2. Assess the need for NGT
3. Call Poison Control / give specific antidote
4. Block absorption
5. Enhance elimination

Specific Treatment of Methanol

A. Poorly absorbed by charcoal
B. Do not ipecac
C. Excellent removal by hemodialysis
D. Keys:
   a. Reverse acidosis with bicarbonate
   b. Infuse ETOH to a level of 100 - 150 mg%
   c. Keep ETOH on board until methanol is below 20 - 30 mg%
   d. Or, if available, 4-methylpyrazole will block breakdown of *methanol* (and ethylene glycol):
      - Blocks alcohol dehydrogenase
      - It may be given PO or IV
      - Starting dose is 15 mg/kg IV

Loading and Maintenance of Ethanol

- Give 10% ethanol in D_5W
- Load with 10 cc/kg
- Maintain about 150 cc/hr
- Follow ETOH levels
- Continue until levels below 20 - 30 mg%

ETHYLENE GLYCOL

$HO\text{-}CH_2\text{-}CH_2\text{-}OH$ MW 62 each 6.2 mg% = 1 mosm

- An antifreeze agent
- a sweetener for wine
- 40 - 60 deaths a year in USA
- Has no odor
- Profound acidosis from oxalic acid
- **Renal failure from oxalate crystals in kidney**

Three target Organs for Ethylene Glycol:

CNS: First Symptoms

- Euphoria and nystagmus - early
- Seizures and coma - later

Cardiac: Hours Later

- Hypertension
- ST - T Wave changes
- Pulmonary edema

Renal: Within First Day

- Crystalluria occurs first
- RF may take 1 - 2 days to occur
- RF due to multiple factors: acidosis, calcium oxalate

Treatment of Ethylene Glycol

- Reverse acidosis with bicarbonate
- ETOH drip or 4-methylpyrazole (if available)
- Thiamine to help conversion of glycolic acid to non-toxic metabolite
- Pyridoxine to assist in metabolizing of glycoxalate to glycine

ISOPROPYL ALCOHOL

$CH_3\text{-}CHOH\text{-}CH_3$ MW 60 each 6.0 mg = 1 mosm

- Usually benign
- twice as drunk, twice as sick, twice as long
- **Ketosis without acidosis**
- No anion gap
- Hemorrhagic gastritis, hypotension

ALTERED MENTAL STATUS IN THE ALCOHOLIC

This is my initial approach. Most patients are just drunk, but you only have a short time to find potentially reversible lethal causes. I divide AMS into only five causes. There are innumerable causes, hut this gives me a workable list and allows an orderly work-up. Before starting..

- Secure ABCs
- Give 2 mg of Narcan, fingerstick glucose, 100 mg of thiamine
- Do a rapid primary survey and 2" assessment looking for occult trauma or focal signs
- Start Alcohol Cocktail at 200 cc/hour
- Begin AMS workup

There are only five (5) causes of AMS:

1. **Vital Sign Abnormalities**
2. **Toxic/Metabolic Causes**
3. **Structural Abnormalities**
4. **Infectious Etiologies**
5. **Psychiatric Illness**

Vital Sien Abnormalities:

BP Hypotension due to ETOH, dehydration; sepsis
Pulse Holiday Heart Syndrome; abnormal pulse = EKG
Resp Hypoventilation from CNS depression; "KUSMAL" respirations
Temp Hypothermia due to Wemicke's, sepsis, exposure, hypoglycemia
Hyperthermia from dehydration, exposure, or sepsis

Toxic/Metabolic:

SMA-7
Na (high or low)
Glu (high or low)
BUN/Cr (dehydration)
HCO, (acidosis; co-ingestants)
ABGs (hypoxia, pneumonia acidosis)
ETOH level: if below 200 mg%, think other cause if patient deeply comatose; coma in chronic ETOH abusers rare below levels of 400.
Tox Screen - rarely indicated

Structural:

- Search for signs of trauma for focal findings or deep coma:
  - CAT
  - LP
  - EEG

Infectious:

CBC Sepsis (also assess for anemia, B_{12}, lead)
U/A Ketones, oxalate crystals
CXR TB, Pneumonia, Lung Abscess
LP Meningitis

Psychiatric:

- A diagnosis of exclusion

OTHER ALCOHOLIC EMERGENCIES

Hvaotbermia:

- In ETOH, relatively benign
  - Use heated mist and blankets
  - Avoid hypoxia, acidosis, cardiac drugs, instrumentation
  - Use PD only in non-responders to mist
  - Beware Wemicke's, Occult Head Trauma, Sepsis

Holidav Heart:

- Afib and Aflutter common
  - PAT and V-Tach also seen
  - May be early finding of cardiomyopathy
  - Assess K, Mg, Ca, PO, levels
  - Treat conservatively, treat withdrawal
  - High dose Digoxin with hypokalemia and withdrawal don't mix well
  - Treat hypotension with alpha agonists (if patient not volume depleted)

- *My present drugs of choice are magnesium, Valium, and beta blockers.*

CT vs LP:

A. Alcoholics are prone to occult trauma
B. Beware subdurals due to trauma and cortical atrophy
C. In general, do CT than LP

D. In febrile patients who you can't decide whether to CT or LP:

- do blood cultures
- 2 grams of ceftriaxone (or other broad spectrum antibiotic)
- send for non-contrast CT
- do LP if CT negative

HYPOKALEMIA, FUIABDOMYOLYSIS, MYOPATHY, AND HYPOMAGNESEMIA

Hypokalemia

Alcoholics are often hypokalemic due to:

1. Diet
2. Diuretic effects of ETOH
3. Hypomagnesemia
4. NN
5. Diarrhea

- K levels of 2.0-2.5 meq not that uncommon in chronic malnourished alcoholics.
- Patients may require 300 + meq of KCL in first day.
- 50% + of chronic ETOH abusers in withdrawal may be hypokalemic.
- With significant hypomagnesemia, renal wasting of K will occur even when massive amounts of PO or IV KCL are given.

Rhabdomyolysis

Rbabdo may present with:

Pain
Weakness
Partial paralysis
Muscle swelling

- Urine may be myoglobin negative - use CPK and clinical picture
- Urine is not usually the classic Coca-Cola brown due to chronicity and the low level of muscle destruction
- Must replete K, Mg and follow Ca + PO,

- Aggressive hydration and urine flow rates of at least 100 ccs per hour
- Do not try too hard to alkalinize the urine (K washout, pulmonary edema, hyperosmolarity, electrolyte problems)
- If patient can eat - feed him and (if no lactase deficiency) use skim milk

Myopathy

ETOH abusers are at significant risk for myopathy due to:

- alcohol ingestion
- seizures
- trauma
- rhabdo
- DTs
- hypoK
- hypoMg
- hypoPO_4

Myopathy usually proximal with normal DTRs.

Hypomagnesemia

- May increase incidence of ETOH withdrawal seizures
- Promotes hypokalemia
- Promotes hypocalcemia
- Prolongs Q-T interval
- May not see positive Chvostek or Trousseau's signs

*Treatment of **Hypomagnesia***

- Serum levels DO NOT predict total body deficits accurately
- Deficits require about 10 grams on day 1 and about 6 grams X 2-5 days
- Use $MgSO_4$ IV slowly and follow BP
- 50% $MgSO_4$ = 5 grams/10 cc = 0.5 grams/cc
- Give at least 1 gram per hour
- DO NOT ORDER Magnesium by the Amp.
  (1 Amp = 2 cc, 10 cc or 50 cc)

Conclusions in Treating Alcoholic Emergencies

- **Be careful!!**
- **Assume nothing**
- **Trust No One**
- **Beware occult trauma**
- **Beware non-AKA acidosis**

The most common errors in alcoholics are made by doctors and nurses who assume "He's just drunk."

Alcohol Cocktail

D_5*NSS (at 200 cc/hr)
MVI (1 amp)
Folate (1 mg)
Potassium (40 meq)
Magnesium (5 grams)

***Give 100 mg thiamine to any alcoholic receiving glucose**

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ALCOHOLIC EMERGENCIES

Questions and Cases

A. An ED physician is at home minding his own business when he is called in to work a shift. His blood alcohol level (BAL) is 100 mg%, although he has probably "only had two beers."

1. When will his BAL be O?
2. Would fructose help increase alcohol metabolism?
3. Would Narcan help?
4. Would Flumazenil be of assistance?

B. A long-time ED patron returns in police detention after a disagreement over the rights of an individual to assume ownership of a bus stop waiting area. The patient is withdrawing from alcohol. When was his last drink if he is now:

1. Tremulous
2. Seizing
3. Hallucinating
4. Delirious and Febrile

C. The safest way to give Thiamine is:

a. IV
b. PO
c. IM

D. Name the "perfect" IV fluid to use in almost any alcoholic requiring IV fluids. What 5 fluid-electrolyte-nutritional deficiencies are you trying to treat (or prevent)?

E. Which of the following should be used in the treatment of alcohol withdrawal?

1. Librium
2. Valium
3. Halcion
4. Ativan
5. Tranxene
6. Phenobarbital

F. Match the side effect with the treatment

V Valium	A Ativan	Ph Phenothiazines	Ox Oxazepam
Ha Halcion	Ah Antihistamine	Pb Phenobarbitol	M Midazolam

1. Hypotension
2. Not helpful
3. Alpha blockade
4. Best choice PO
5. Coma
6. Lowers seizure threshold
7. $T_{½}$ = 3.5 days
8. $T_{½}$ = patient's age
9. $T_{½}$ = 6 8 hours
10. $T_{½}$ = 1.5 hours

G. What is the "magic drug" to add for uncontrolled hallucinosis if benzodiazepines don't adequately calm the patient?

H. Which words best describe a classic alcohol withdrawal seizure?

Focal	Self-limited
status	Generalized
Occur over 3-5 days	Over 6 - 12 hours

I. A 46 year old alcoholic has what is believed to be an "alcohol withdrawal seizure" (AWS). A complete workup including CT, LP, EEG, MRI, VDRL, ECHO, MUGA, V/Q, IVP, SMA-7, SMA-12, CBC, EKG and CXR are all pretty normal. He returns 6 months later with a focal withdrawal seizure after suddenly stopping drinking. What is the likelihood that this is an alcohol withdrawal seizure?

a. 0
b. 3%
c. 20 - 30%
d. more than 50%

J. Is Dilantin (Phenytoin)

a) Prophylactically, or
b) Therapeutically indicated for AWS, or
c) Not indicated

K. What is the best test in AWS patients?

L. An alcoholic presents in withdrawal. The patient is quite ill appearing and develops status seizures. The patient is poorly controlled with 20 mg of Valium, 4 mg lorazepam and 1000 mg of dilantin.

ABGs: pH = 6.80 pCO_2 = 50 pO_2 = 70

What OD in alcoholics gives refractory status seizures?

M. An alcoholic presents with AWS and a very unusual "mothball" odor kind of breath. Eosinophilia is noted on his CBC. What has he ingested?

N. Why do patients in DTs die?

0. A cold alcoholic is brought to the ED. The patient slept out all night and is unresponsive to verbal stimuli. Physical exam reveals:

1. coma
2. Hypothermia
3. Hypotension
4. Bradycardia
5. Miosis

The patient does not rewarm. If this isn't just exposure, what is it?

a. Subdural/epidural
b. Sepsis
c. TCA OD
d. Wernicke's encephalopathy
e. Profound hyponatremia

P. A 33 year old ETOH abuser presents with nausea and vomiting. Physical exam reveals a thin patient in mild distress. The abdomen is diffusely tender and bowel sounds are diminished.

VS: BP = 110160 P = 100 Resp = 28 Afebrile

140	95
3.8	15

= 7.30

3.8 140 15 BUN Glu 65 11

What is the differential?

What is the most likely diagnosis?

Q. Which of the following are useful in the routine treatment of alcoholic ketoacidosis (AKA)?

1. Bicarbonate
2. Glucose
3. Phosphorus
4. Potassium
5. Volume

R. A patient with presumed AKA presents with a three-day history of N/V and abdominal pain. His venous pH is 7.46; could this be AKA?

S. A 27 year old chronic binge drinker presents wildly intoxicated claiming he's having trouble seeing. His venous pH = 7.28;

140	90
3.2	15

BUN = 12
Glu = 80

The patient's serum ethanol is 280 mg% and the measured (or true) osmolarity is 360 mOsm:

a. What is his osmolar gap?

b. Could this be a methanol OD?

Calculations

AG = 140 (90 + 15) = 35 (significantly elevated)
Calc Osmo = 2 x Na + Glu/18 + BUN 2.8 + $\frac{\text{ETOH}}{4.2}$ = about (I used Glu/20 + BUN/3 + ETOH/4)
OG = 360 - 348 = 12

Answer: With an osmolar gap of 12 (once alcohol's osmolar contribution is known), there is NO chance of a significant methanol OD.

c. Assuming the patient's serum ethanol was only 150 mg% and his osmolar gap was 88, what is his methanol level?

d. Does the patient need to be treated for a methanol OD?

Calculations:

150 mg% divided by 4.4 = 34 mosm's by ETOH
88-34 = 54 mosm unaccounted for = due to methanol
54 mosm X 3.2 = 173 mg% blood level of methanol

Answer: YES!

T. MATCHING QUESTIONS ON THE ALCOHOLS

Alcohol (A) Ethylene Glycol (EG) Isopropyl (IP) Methanol(M)

Pancreatitis-like pain	Profound acidosis
Blind	Formaldehyde
Papilledema	Oxalic Acid
Urinary Crystals	"Windex"
No acidosis	Sterno
"Snowstorm"	Treat with bicarbonate
Tetany	Treat with D_5NS
Sweet	Renal Failure

U. An alcoholic presents in deep coma. Physical exam reveals dilated pupils, papilledema, a normal cardiopulmonary exam. ECG shows diffuse ischemic changes. ABG's: pH = 7.20; pCO_2 = 30; pO_2 = 80; 02 Sat. = 95 %. What diagnostic test is now indicated?

V. A long time drinker presents in deep stupor. He makes no purposeful movements to deep pain His BAL is 225 mg%. Assuming he receives conservative care, when will he wake up?

W. An alcoholic presents with a withdrawal seizure and AMS. He is hypocalcemic, hypomagnesemic, and hypokalemic. He receives 20 cc of CaCl, 5 grams of $MgSO_4$ and is getting 20 meq KCl/hour. Physical exam reveals dilated pupils, gastric atony and his ECG has ST., multifocal PVC's and a Q-T of .49.

Diagnosis?
Treatment?

X. Which of the following are indicated in treating new onset atria1 flutter (HR 150) in a binge drinker:

a. Adenosine	g. Propanolol
b. Carotid massage	h. Potassium
c. Digoxin	I. Quinidine
d. Esmolol	j. 200 WS
e. Ethanol	k. Valium
f. Magnesium	1. Verapamil

Y. An alcoholic has been sick for a number of days. He presents with a temperature of 38.6 and a questionably stiff neck. There is no papilledema. Which of the following should be done fist:

A. CT
B. LP
C. Both
D. Neither

Z. A long-time alcoholic presents with leg pain, Physical exam is WNL. The patient returns with inability to walk and the following ABG's:

$pH = 7.30$
$pCO_2 = 50$
$pO_2 = 70$

Which electrolyte abnormalities are probably present?

a. Hypocalcemia
b. Hypokalemia
c. Hypomagnesemia
d. Hyponatremia
e. Hypercalcemia
f. Hyperkalemia
g. Hypermagnesemia
h. Hypematremia

Aa. A patient develops alcohol induced rhabdomyolysis. Should bicarbonate be used to alkalinize the patient's urine?

Bb. An alcoholic presents in a drunken rage. He is dirty and incontinent of urine and feces. He is laughing wildly, vomiting and in pulmonary edema. Why is his skin crawling?

Cc. An alcoholic presents with mottled "lobster skin" colored blotches all over his body. What has he been seeing on his body? What has he treated himself with?

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