

Opioid Use Disorder Education and Treatment ECHO Series

Session 12 – Alcohol Intoxication and Withdrawal States

May 3, 2022

Heather Bell, MD and Kurt DeVine, MD

Slides adapted from Charlie Reznikoff, MD, FACP



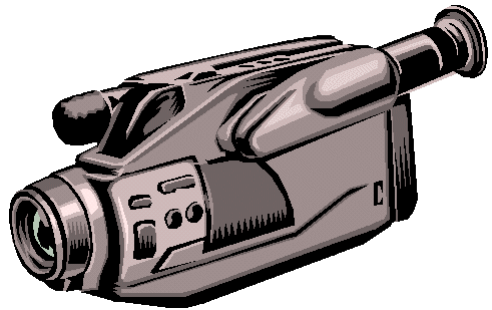
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STRONG MEDICINE FOR MINNESOTA

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Announcements

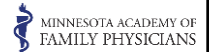


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SESSIONS ARE RECORDED

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YES, THERE'S *FREE* CME

This activity has been planned and implemented in accordance with the accreditation requirements and policies of the Minnesota Medical Association (MMA) through the joint providership of Stratis Health and the Minnesota Academy of Family Physicians. **Stratis Health is accredited by the MMA to provide continuing medical education for physicians.**

Stratis Health designates this educational activity for a maximum of **1 AMA PRA Category 1 Credits™**.

Physicians should claim credit commensurate with the extent of their participation in the activity.

Continuing Education Credits and Contact Hours for Other Health Professionals

The OUD Education and Treatment ECHO Series may meet continuing education requirements for your focus. It is the responsibility of the individual to determine if this activity fulfills that requirement.



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Attendance

- Please chat us the names of people on ECHO if there are multiple people in your room!
- “Re-name” your self so we know who’s here!
- Please turn your video on!
 - Human connection!
 - And we do NOT care if you are eating!



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Case Presentations!

The ECHO model is based on case-based learning!
The case presentation form is available on the MAFP website
(<https://bit.ly/OUDCase>) and in the announcements email!
BUT feel free to present in any de-identified format!

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Upcoming Tuesday ECHO Sessions

- **Tuesday, May 17, 2022:** OUD and Stimulants
- **Tuesday, June 7, 2022:** OUD and Pregnancy
- **Tuesday, June 21, 2022:** OUD and Neonatal Opioid Withdrawal Syndrome (NOWS)
- **Tuesday, July 19:** Perioperative Management
- **Tuesday, August 2:** Community Collaboration Engagement
- **Tuesday, August 16:** Motivational Interviewing (MI) Lapse/Relapse

Upcoming Wednesday ECHO Sessions

- **Wednesday, May 4, 2022**
Fentanyl Test Strips Part 3, with Charlie Reznikoff, MD
- **Wednesday, May 11, 2022**
The Pharmacotherapy of Alcohol Use Disorder, with Ricardo Restrepo, MD, MPH
- **Wednesday, May 18, 2022**
Understanding East African Clients, with Yussuf Shafie, MSW, LICSW, CEO, Alliance Wellness Center
- **Wednesday, May 25, 2022**
The Opioid Epidemic From The Perspective of an Emergency Physician and Toxicologist at a Safety-Net Hospital, with Jon Cole, MD, Hennepin Healthcare

“The Addiction Connection Podcast”

Weekly addiction topics- Tuesday release day!

www.buzzsprout.com/954034

(Or anywhere you get your podcasts!)

Email us questions:

theaddictionconnectionpodcast@gmail.com



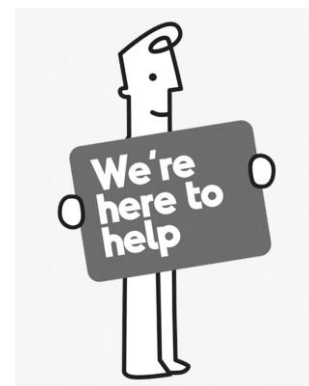
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TECHNICAL ASSISTANCE

- **We are ALWAYS here for you!**
 - Program implementation
 - Inductions
 - Difficult cases
 - Trouble-shooting
 - Anything!
- **Call us anytime:**
 - Erin Foss, RN, Program Manager/Nurse Specialist
efoss@stratishealth.org, Cell: 320-282-6553
 - Heather Bell: 320-630-5607
 - Kurt DeVine: 320-630-2507



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Objectives

- List the unique metabolism of alcohol
- Describe the relevance of alcohol binges
- Describe the metabolic disturbances from alcohol ingestion
- Explain alcohol withdrawal syndromes
- Identify different tests used to monitor alcohol use

Epidemiology

Epidemiology of Alcohol Use Disorder (AUD)

- Consumption:
 - 88% of US population has some alcohol in their lifetime
 - 70% of 21-25yo consume alcohol, decreases as age
 - 50% of 60-64yo consume alcohol
- Scope of the problem:
 - ~100,000 people die annually from alcohol related causes in the U.S.
 - 3rd leading cause of preventable death in U.S.
 - ~50% of US liver disease attributed to alcohol misuse



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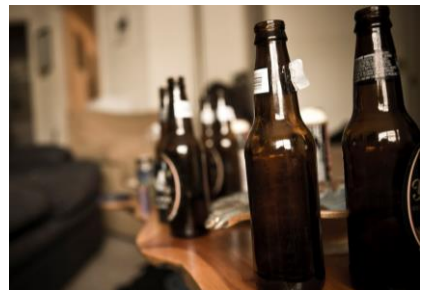
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Epidemiology of AUD cont.

- Prevalence of AUD:
 - Lifetime: 29.1%
 - 12 month: 13.9%
 - 15.1 million adults have AUD
 - 20% of the drinking population drinks 80% of EtOH (ethyl alcohol) sold
- Only 19.8% of respondents with lifetime AUD treated



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Epidemiology of AUD cont.

- Prevalence similar to other chronic diseases: asthma, diabetes, depression
- Significant associations were found between 12-month and lifetime AUD and:
 - Other substance use disorders
 - Major depressive
 - Bipolar I disorder
 - Antisocial personality
 - Borderline personality



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Epidemiology

- 50% likely genetic
- Severity of alcohol abuse- familial
- If both parents have AUD: 7Xs higher chance of having AUD
- AUD: men > women*
 - * Telescoping



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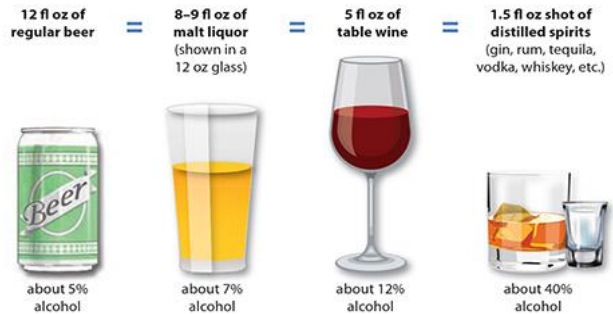
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How Much?

- Too much:
 - Men:
 - 5 or more in a sitting
 - 15 or more per week
 - Women:
 - 4 or more in a sitting
 - 8 or more per week



- Standard drink:
 - Average person metabolizes about 1 standard drink an hour (20mg/dL every 60-90min)
 - Men: Each drink adds 20mg/dL to one's blood alcohol level (BAL)
 - Women: Each drink adds 40mg/dL to one's BAL

Diagnosis

CAGE Questionnaire

- **C**ut down
- **A**nnoyed
- **G**uilty
- **E**ye-opener
 - 2 or more strongly associated with alcohol dependence



National Institute on Alcohol Abuse and Alcoholism (NIAAA)

AUDIT-C Questionnaire

| Questions | Scoring Table | | | | | Score |
|--|---------------|-------------------|-----------------------|--------------------|-----------------------|-------|
| | 0 | 1 | 2 | 3 | 4 | |
| How often do you have a drink containing alcohol? | Never | Monthly or less | 2 - 4 times per month | 2-3 times per week | 4+ times per week | |
| How many units of alcohol do you drink on a typical day when you are drinking? | 1 - 2 | 3 - 4 | 5 - 6 | 7 - 9 | 10+ | |
| How often have you had 6 or more units if female, or 8 or more if male, on a single occasion in the last year? | Never | Less than Monthly | Monthly | Weekly | Daily or almost Daily | |

- 0-3: Health Promotion
- 4-5: Brief Intervention
- 6-7: Brief Intervention +/- pharm +/- psychosocial
- 8-9: Pharmacotherapy +/- psychosocial +/- specialty care
- 10-12: Specialty care management

DSM-V: Criteria for AUD

1. Had times when you ended up drinking more, or longer, than you intended?
2. More than once wanted to cut down or stop drinking, or tried to, but couldn't?
3. Spent a lot of time drinking? Or being sick or getting over other aftereffects?
4. Wanted a drink so badly you couldn't think of anything else?
5. Found that drinking—or being sick from drinking—often interfered with taking care of your home or family? Or caused job troubles? Or school problems?
6. Continued to drink even though it was causing trouble with your family or friends?
7. Given up or cut back on activities that were important or interesting to you, or gave you pleasure, in order to drink?
8. More than once gotten into situations while or after drinking that increased your chances of getting hurt (such as driving, swimming, using machinery, walking in a dangerous area, or having unsafe sex)?
9. Continued to drink even though it was making you feel depressed or anxious or adding to another health problem? Or after having had a memory blackout?
10. Had to drink much more than you once did to get the effect you want? Or found that your usual number of drinks had much less effect than before?
11. Found that when the effects of alcohol were wearing off, you had withdrawal symptoms, such as trouble sleeping, shakiness, restlessness, nausea, sweating, a racing heart, or a seizure? Or sensed things that were not there?

National Institute on Alcohol Abuse and Alcoholism (NIAAA)

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Signs/Symptoms and Comorbidities of AUD



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Signs/Symptoms of AUD

- Signs
 - Ascites
 - Skin changes
 - Telangiectasia
 - Icterus
 - Hypertension
 - Cardiomyopathy
 - Hepatomegaly
 - Rhinophyma
 - Muscle wasting
- Symptoms
 - GI bleeds
 - Sleep disturbance/apnea
 - DWI
 - Violence
 - Falls
 - Memory issues
 - Esophageal cancer
 - Stomach cancer



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Opioids

- Opioids
 - Together slower disposal rates = increase toxicity
 - Ethanol may modify some opiate receptors and the effects of opioids
 - Patients with OUD have unusually high rates of alcoholism



(J. Advances in Alcohol and Substance Use)

MMWR Oct 10, 2014. 63(40) 881-885.

Effects of Ethanol and Oxycodone. Anesthesiology (126)3: 534-542. March 2017.

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OUD and Alcohol

- Hser et al. 2017
 - 5307 adults with OUD – most also had chronic pain:
 - 23.4% also had AUD
 - Patients with OUD prior to chronic pain had AUD 33.4% of the time



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OUD and Alcohol cont.

- Of adults in treatment for AUD or other drugs:
 - 68% had used opioids recreationally in previous 30 days (Price et al. 2011)
- General absence of research examining co-use
- What is known:
 - Nonmedical use of opioids and OUD increases risk of AUD (NESARC)

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Comorbidities cont.



- Benzos

- EtOH: involved in 18.5% of opioid abuse ED visits and 27.2% of benzo related visits
- EtOH related to 20% of OD deaths with opioids or benzos
- Ethanol and oxycodone cause greater ventilation depression than either alone, with the magnitude being clinically relevant. Older patients more affected.

(J. Advances in Alcohol and Substance Use)

MMWR Oct 10, 2014. 63(40) 881-885.

Effects of Ethanol and Oxycodone. Anesthesiology (126)3: 534-542. March 2017.

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Comorbidities cont.



- Cocaine

- Form: cocaethylene
 - Actions similar to cocaine but less potent
 - Longer T1/2 than cocaine
- Enhanced/prolonged cocaine effects

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Comorbidities cont.

- Smoking
 - Synergistic adverse effects especially with certain cancers
 - Smoking may counteract deleterious effects of alcohol- especially cognitive
 - Together cause addictive/synergistic analgesic effects (activation of the opioid system)
 - Potentiate the rewarding effects of one another- dopamine reward pathway

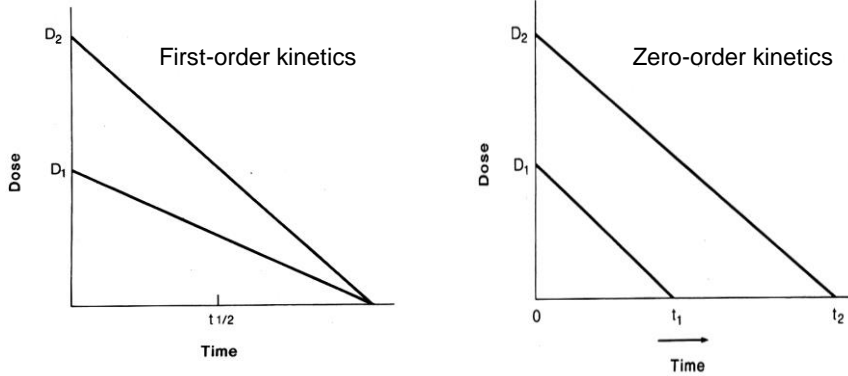


Alcohol Metabolism

Alcohol Has Zero-Order Kinetics

- All other drugs have first-order kinetics

First-order vs. Zero-order kinetics

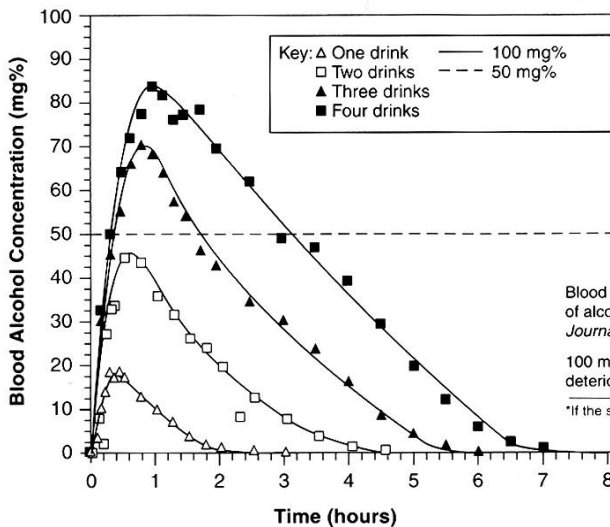


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Blood alcohol concentration (BAC) after the rapid consumption of different amounts of alcohol by eight adult fasting male subjects.* (Adapted from Wilkinson et al., *Journal of Pharmacokinetics and Biopharmaceutics* 5(3):207-224, 1977.)
100 mg% is the legal level of intoxication in most States. 50 mg% is the level at which deterioration of driving skills begins. (*JAMA* 255:522-527, 1986.)

*If the same number of drinks are consumed over a longer period of time, BAC's will be lower.

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Metabolism Kinetics

- First-Order:
 - Fraction of drug removed at any one time is independent of dose
 - Exponential decline
 - Rate of elimination is proportional to the drug concentration
 - Most drugs
- Zero-Order:
 - Amount of drug removed at any one time is **CONSTANT**
 - Dependent on dose (rather than the fraction of drug)
 - Linear decline
 - Ethanol, phenytoin, omeprazole, salicylates

Tolerance

Types of Tolerance

- **Learned/Functional:**
 - The brain adapts to the presence of alcohol
 - The brain develops mechanisms to slow itself down and act deliberately
- **Metabolic:**
 - Breakdown and elimination of drugs from the body
 - Genetics most significant



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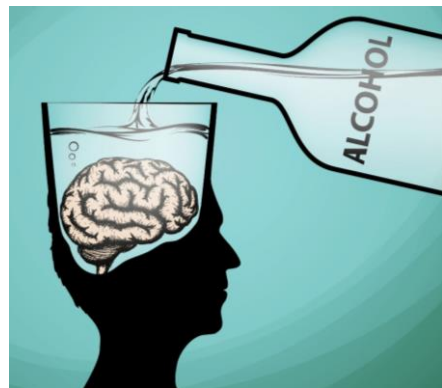
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Functional Tolerance

- Practiced tasks while under the affects of alcohol
- Tasks become “easier” and performed “better” with practice
- Bar A-> Home vs Bar B-> home
- *Several other types of functional tolerance... that'll be another talk!



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Functional Tolerance

- Physiological mechanisms:
 - Desensitization of alcohol-sensitivity receptors in the CNS
 - GABA-ergic
 - Changes in neuron firing rates
 - Compensate for deterioration of GABA-ergic inhibition



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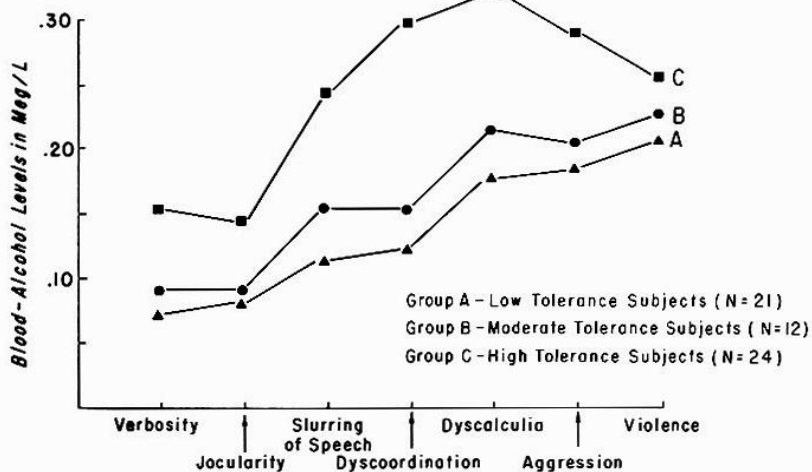
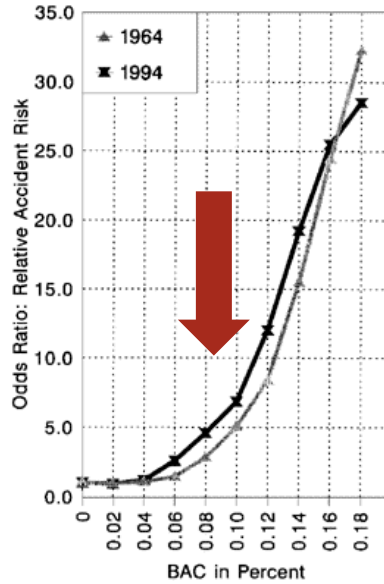


FIGURE 5

Behavioral Manifestations of Alcohol as a Function of Blood-Alcohol Levels in Low, Moderate and High Tolerance Groups

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Blood Alcohol and Probability of Accident



Tolerance shifts the curve to the right

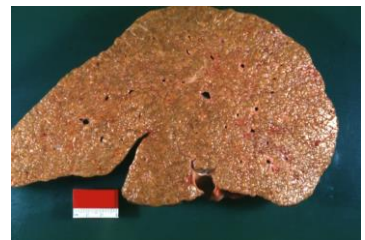
Inexperience shifts it to the left

To get over BAL 0.1 you need to “binge”

Alcohol increases suicidal actions.

Metabolic Tolerance

- Increased alcohol break-down by the liver
- Slows the increase of blood alcohol concentration (BAC) -> attenuation or disguise of intoxication
- Organs affected by alcohol not necessarily more tolerant however
- Cirrhosis may cause sensitization to alcohol



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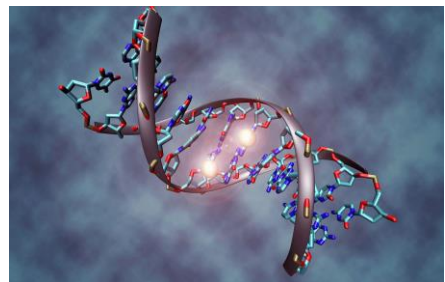
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Metabolic Tolerance

- Alcohol dehydrogenase (ADH) enzymes increase their activity (and likely overall amount)
 - Mechanism not well understood
 - (Barbiturates also do this to ADH...)
 - Genetic influence



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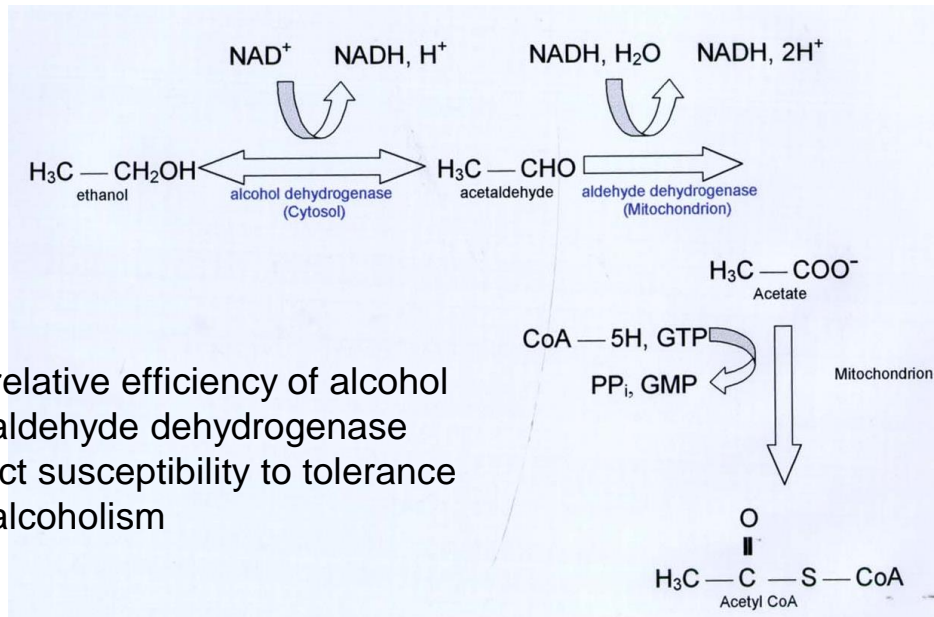
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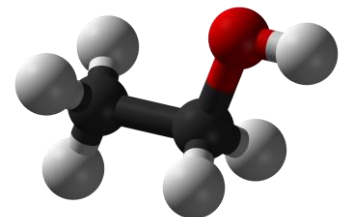
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- The relative efficiency of alcohol and aldehyde dehydrogenase predict susceptibility to tolerance and alcoholism

Variables Determining of BAL

- Body water volume (muscle mass)
- Genetics
- Speed of consumption and absorption
 - Temperature of beverage (cold>warm)
 - Presence of food in stomach (empty>full)
 - Body position (standing>sitting)
 - Carbonation (carbonated>flat)

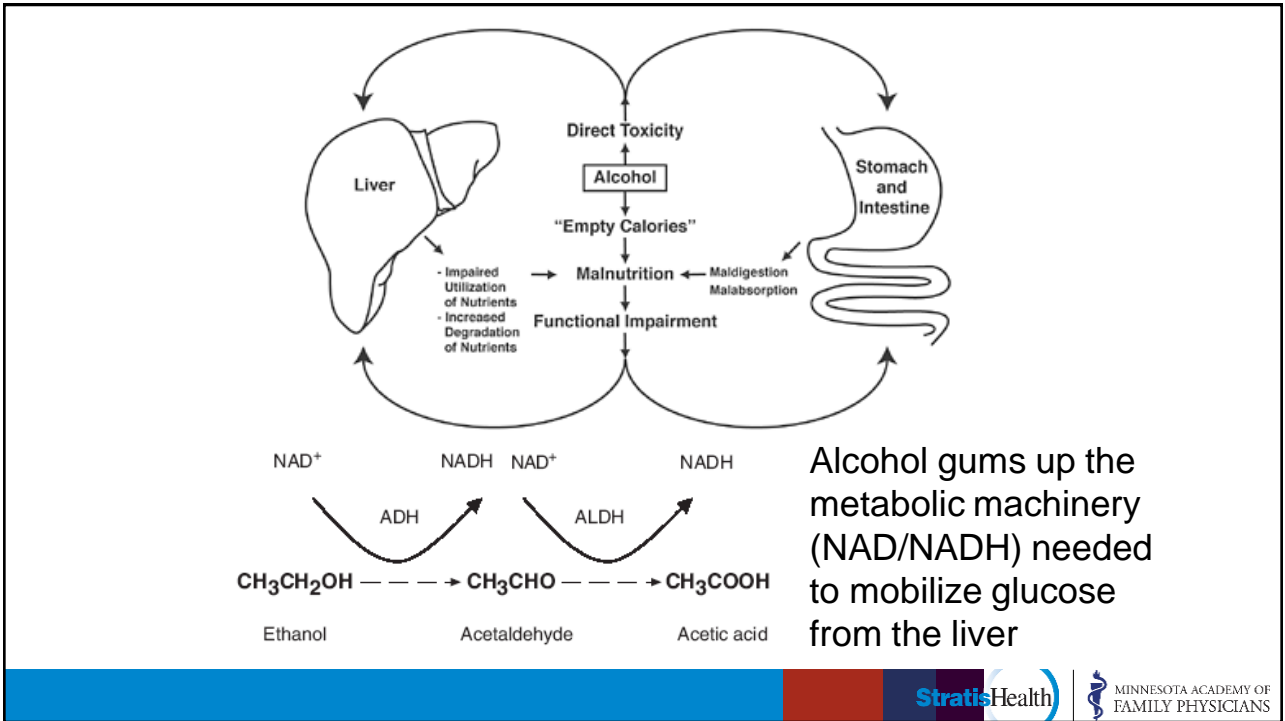


Metabolic and Nutritional Effects of Alcohol Consumption

Alcoholic Ketosis

- Alcoholic ketosis (ketoacidosis) is starvation ketosis complicated by build up of acidic alcohol metabolites

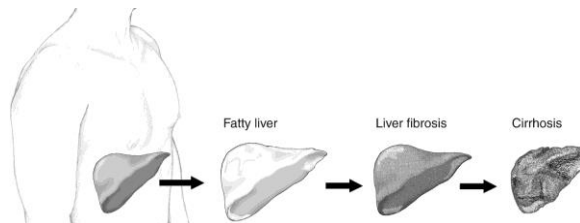




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Alcoholic Ketosis cont.

- Low carbohydrate intake puts the body in a catabolic state
- Decreased gluconeogenesis due to alcoholic hepatitis
- Increased acid load to the body due to byproducts of alcohol and anaerobic metabolism blockades sugar mobilization



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Alcoholic Ketosis

- Elevated anion gap
 - Hypovolemia and anaerobic metabolism lead to lactic acidosis
 - Ketones from alcohol breakdown
 - Beta-HO-Butyrate predominates
 - This is not the ketone reported on U/As
- Acidosis commonly severe ($\text{HCO}_3^- < 10$)
- Metabolic alkalosis from emesis is also possible



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Alcoholic Ketosis

- Give fluids with dextrose
- Elevated anion gap very common in acute and recent alcohol intoxication
- Consider other causes of anion gap when kidney malfunction present:
 - Ethylene glycol
 - Methanol ingestion
 - Salicylate ingestion



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Hypoglycemia in Alcoholic Diabetics

- Multifactorial: med compliance, nutrition, liver disease, acid/base balance
- Hypoglycemia unresponsive to glucagon
- Often requires IV dextrose
- May have severe hypokalemia



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Remember: He is malnourished!

- People who drink a case of beer daily (more than 3000 calories) gain weight without eating any food. They are profoundly malnourished and at risk of vitamin and electrolyte abnormalities.



“How many meals do you eat per day?”
“What do you eat?”

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Watch electrolytes, give multivitamin

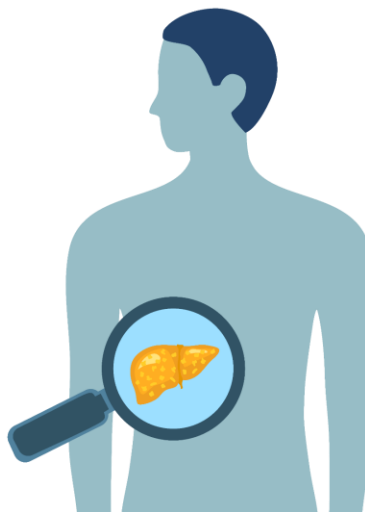
- **Thiamine:**
Wernicke's probably more common than you think
- **Potassium:**
will fall w/correction of acidosis
- **Phosphorus:**
will fall with refeeding
- **Magnesium:**
needed for K+ replacement
- These may be persistently low, as in refeeding syndrome



Liver “Function” Tests

Alcohol Transaminitis

- AST/ALT >2
- AST absolute value <500
- Take note of TRUE “liver function” tests
 - Bilirubin
 - Albumin
 - INR
 - Plts



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Role of GGT

- Gamma-glutamyl Transferase (GGT) rarely used anymore
- The HBA1C of alcohol (now CDT)
- Get a baseline “sober” GGT level
- Rule out other causes of liver/biliary irritation
- GGT increase indicate alcohol consumption over weeks



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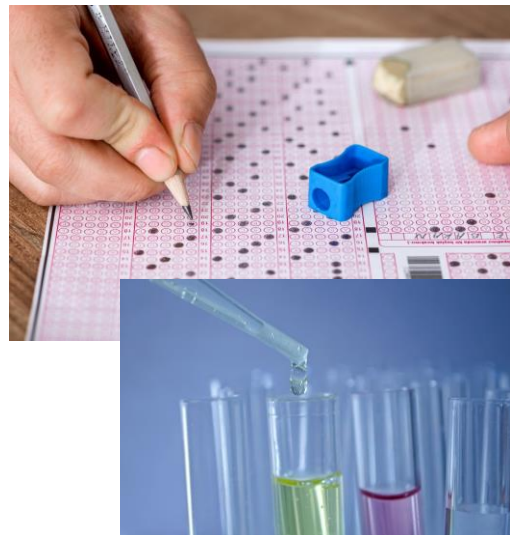
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So... is there an AUD “test”?

EtOH “Tests”

- EtG/EtS
- CDT
- PEth



EtG/EtS

- EtG:
 - Described in 1950s
 - Clinical use 2001 (Dr. Friedrich Wurst and Dr. Gregory Skipper)
 - More sensitive and reliable indicator of drinking and abstinence than urine alcohol
 - Used by Federation of State Physician Health Programs (HPSP) for physician abstinence



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EtG/EtS cont.

- EtG:
 - “False positives”
 - incidental exposure (similar to poppy seeds)
 - “1000s” of incidentals that make the “list”
 - Should have closer monitoring
 - 50% admit to drinking right away and another 40% shortly thereafter (in studies)
 - Especially if levels <1,000ng/mL should not be “presumed” positive
 - No “bright line” to distinguish: SAMHSA guidelines 2006:
EtG >1,000ng/mL “likely due to drinking”



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EtG/EtS cont.



- Intersubject variation in amount EtG produced from a given exposure
 - Same amount of consumption with as much as 200-fold difference
 - “Hyperproducers”
- Elevated concentration of urine artificially increases EtG and EtS
 - Few labs “correct” levels for concentration (urine creatinine)
 - Normalizing should be considered especially for EtG levels under 20,000ng/mL

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EtG/EtS cont.



- Alcohol-based hand sanitizer: ++ false positives: from vapor rather than through skin
- EtG vs EtS stability:
 - EtG can be degraded in urine at room temp but not frozen or heated
 - EtG can be degraded by certain bacteria in urine (heating or cooling inhibits bacterial activity so should be done)
 - EtG can be synthesized by bacteria in-vitro
- EtS: superior marker-more sensitive and specific
- Auto-Brewery Syndrome: consuming sugar and baker’s yeast orally can produce elevated EtG/EtS

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CDT

- Carbohydrate Deficient Transferrin
 - Combination of minor isoforms (not 'important')
 - Account for <1.6% of total transferrin found in plasma
 - Formation directly proportional to alcohol intake
 - Ethanol inhibits enzymes responsible for addition of carbohydrate side-chains and induce sialidase that removes terminal sialic acid residues from side-chains
 - %CDT independent of extent of alcohol-related liver damage EXCEPT with severe cholestatic liver disease (falsely high)

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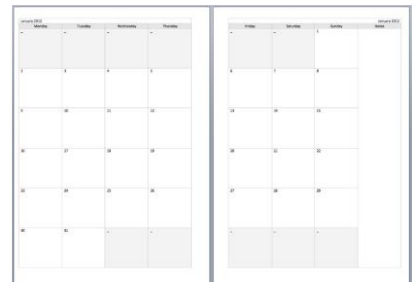
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CDT cont.

- Carbohydrate Deficient Transferrin
 - CDT Alone (units/L) or %CDT
 - Consumption of 50-60 g EtOH per day chronically (at least 2 or 3 weeks) increases CDT
 - Half-life usually 14-17 days
 - Values return to normal 3-4 weeks after abstinence



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CDT cont.



- Women may have higher than men
 - May be less elevated in women than men after chronic consumption
- CDT absolute value
 - Gender dependent
 - Values may be elevated by:
 - Anemia
 - OCP
 - Pregnancy
- %CDT is not gender dependent
- Immunocompromised patients may have lower CDT

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CDT cont.



- Limits:
 - Upper limit CDT:
 - Men: 20U/L
 - Females: 26U/L
 - Cutoff %CDT between 2-3% (most 2.5-2.6%)
- Sensitivity and specificity vary widely in reports (~85% for both) but in general %CDT “better” than GGT or MCV

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CDT cont.

- Applications:
 - Typically used for routine detection of heavy consumption and follow-up treatments or following treatment and regranting of drivers' licenses following DWI
 - Screening tool for AUD in surgical and trauma patients
 - Monitoring of relapse drinking in patients following liver transplant for alcoholic cirrhosis



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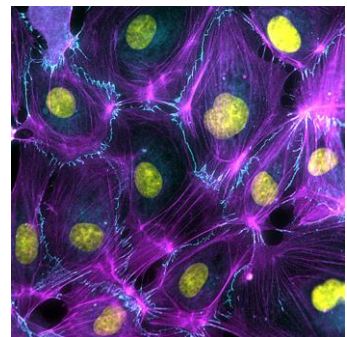
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PEth Test

- Phosphatidyl Ethanol
 - Minor metabolite
 - Formed when ethanol binds to phosphatidylcholine lipids in cell membranes (including RBC)
 - PLD (phospholipase D):
 - Enzyme that causes binding
 - Activity can vary between individuals



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PEth Test cont.

- PEth:
 - Takes >7 standard drinks to “cause” positive
- Other alcohol tests (EtG or EtS) may be altered by external exposures
- OTC meds, mouthwash etc. unlikely to result in a positive test



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PEth Test cont.

- Studies, to date:
 - Include small numbers of subjects
 - Non have shown effects of medications:
 - Medications
 - Age
 - Disease states
 - Etc.
 - False (+): some have shown in participants that denied EtOH use



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PEth Test cont.

- Conclusion:
 - “Probably” useful
 - Be cautious with “low” positive tests
 - It is unlikely that the specificity is 100%
- *Gold Standard to diagnose lapse/relapse:
- Admission of use



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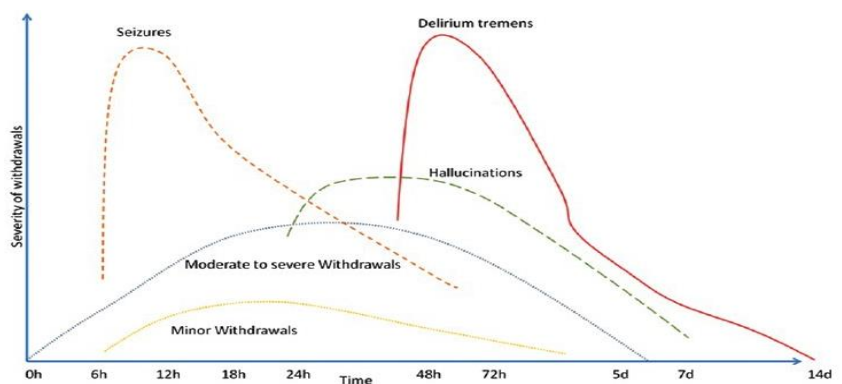
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Alcohol withdrawal is not one thing!

Differentiate prior/expected mild withdrawal from patients with:

- -Past serious withdrawal including delirium or seizure
- -Age >50 or concomitant medical issues (pneumonia)
- -Severe malnutrition, alcohol only source of caloric intake



Ind Psychiatry J. 2013 Jul-Dec; 22(2): 100-108.

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Outpatient Alcohol Withdrawal Treatment for Low-risk People

- No benzos!
- Gabapentin 300 TID, increase to 600 if needed
 - Taking at night sometimes preferred
 - Continue ongoing after detox complete
- Clonidine 0.1 TID for 7-10 days
- Multivitamin
- Nutrition



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Summary

- DRINK SLOWLY WITH FOOD
- Alcohol binges high risk for accidents and metabolic disturbances
- People with DM type 1 need to be very careful with alcohol
- Alcohol causes acid/base, electrolyte, vitamin and liver issues
- Outpatient tx of mild alcohol wd is a thing!



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Podcast:
The Addiction
Connection



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