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Alcohol, and the Evil it Brings

So, your patient has been drinking, or is drunk.

In EMERGENCY: drunks form a fair proportion of patients because

- They drink too much and get **ACUTE ALCOHOL POISONING,** which means
  Disinhibition, garrulous loquacity, agitation, nausea, vomiting, ataxia, slurred speech, sedation, coma, respiratory depression, depressed gag reflex and therefore airway compromise, aspiration and subsequent anaerobic lung abscess and horrible septic death.
- They fall over and get **SUBDURAL HAEMATOMAS** which tend to go unnoticed
- They are prone to fits of **ACUTE PSYCHOSIS**
- They become **DEPRESSED** and **LONELY** and seek comfort from emergency staff
- They run out of money and suffer **WITHDRAWAL,** featuring

  From 2 hours after cessation, until ~ 48 hours: (but usually after at least 12 hours)
  TONIC/ CLONIC SEIZURES
  at 6 – 12 hours: insomnia, tremulousness, mild anxiety, gastrointestinal upset, headache, diaphoresis, palpitations, anorexia
  at 12 – 24 hours: Alcoholic hallucinosis: visual, auditory, or tactile hallucinations
  at 24– 48 hours: Alcoholic hallucinosis: visual, auditory, or tactile hallucinations
  after 48 hours and peaking after 5 days: DELIRIUM TREMENS: hallucinations (predominately visual), disorientation, tachycardia, hypertension, low-grade fever, agitation, diaphoresis…

ALCOHOL HISTORY: as model for general drug history

How much do they drink?
MEN: 21 drinks per week is enough
… up to 50 is only "hazardous"
WOMEN: 14 drinks per week is enough
…up to 35 is only "hazardous"

How often do they drink?
Need at least 2 alcohol-free days per week.

When did they start? GET DETAILED CHRONOLOGY OF DRINKING PATTERN

Changed patterns of use? WHY?

Drink of choice: just for curiosity

How much is spent per week?

Do they drink alone?

Do they drink in the morning? A sign of dependance

CAGE questionnaire fits nicely in here:
Tried to **C**ut down? Been **A**NNOYED by CRITICISM?
Felt **G**UILTY about drinking? Need an **E**YE-OPENER in the morning?

How many drinks does it take to get any effect?
May point towards tolerance

What events or occasions require for them to be drunk?
Eg. do they combat social anxiety by pre-medicating with a few drinks?
Do they use it as an antidepressant?

Have there been LEGAL ISSUES?

RELATIONSHIP PROBLEMS?

Work or health problems?

Is the use PERCEIVED AS A PROBLEM?

EVER SOUGHT HELP FOR IT?

ALWAYS ASK QUESTIONS RELATING TO COMORBID MENTAL ILLNESS:
40% to 90% of substance abuse patients have one

PHYSICAL EXAMINATION:

General
- Jaundice, anaemia,
- Mental state
- Smell
- Ascites
- Body hair distrubution, spider naevi

Hand
- Clubbing,
- osler nodes,
- splinter hemorrhages
- Xanthomas (fatty deposits);
- Dupuytren's contracture:
- Hepatic Flap

Arm
- Pulse: AF, Tachycardia
- Track marks, bruises
- BP

Face
- Smell breath!
- Anaemia, jaundice
- Parotid enlargement

Neck
- JVP, Carotids, lymph nodes (esp. supraclavicular nodes)

Chest
- Gynaecomastia
- Cardiac palpation + auscultation (for AF, LV hypertrophy)
- Percuss and auscultate lung fields

FOR THE WITHDRAWAL SCALE:
- PERSPIRATION
- TREMOR
- ANXIETY
- AGITATION
- HALLUCINATIONS
- AXILLA TEMPRATURE
- ORIENTATION

THEY WILL HAVE LOW BLOOD PRESSURE IF PRESENTLY INTOXICATED.

Abdo
- Caput medusa
- Pancreatitis pain
- Hepatosplenomegaly
- Shifting dullness

Legs
- Peripheral oedema

Test cerebellar
- Test for nystagmus
- Walk heel-toe, on toes, on heels
- Finger nose testing

INVESTIGATIONS:

BLOOD ACOHOL LEVELS FOR ALL

FBC + blood microscopy:
looking for macrocytic anaemia of alcoholism

alpha-fetoprotein: very specific marker of hepatocellular carcinoma;

LIVER FUNCTION TESTING:

GGT =induceable acoholism enzyme

SERUM ALBUMIN → Looking for synthetic liver failure; the albumin level tells you about the last 2 months of liver function (as it ha a longer half life than clotting enzymes)

PROTHROMBIN Time → **low due to reduced rate of cloting factor synthesis in acute liver failure**

Thiamine =low mainly due to malnutrition

RBC Folate (alcohol inhibits the gut tranbsporter of folate)

Liver Biopsy: only way to objectively diagnose alcoholic liver disease. RARELY NEEDED.

ALSO…. While youre there… do an EUC- may be **HYPOKALEMIC** from spewing and **HYPONATREMIC** from excessive diuresis

Are they nauseous/vomiting, have abdo pain, haven't been eating recently, ….have binged in the recent past? Tachycardic, tachypnoeic? Diffuse mild abdo tenderness? HYPOTENSION?? This could be → **Treatment is symptomatic**

ALCOHOLIC KETOACIDOSIS: a wide anion gap acidosis

So: do an ABG to see how severe; …pH may be alkalaemic as well….

AND TEST THEIR URIN FOR KETONES to feel better about calling this a keto-acidosis

Exact criteria are glucose below 3, recent massive alcohol intake with rapid cessation, and a lack of a decent explanation for their wide anion-gap metabolic acidosis

MANAGEMENT

DSM-IV defines alcohol dependence as:

"repeated ***alcohol-related difficulties*** in at least three of seven areas of functioning that cluster together **over any 12-month period**."

These problems include ANY COMBINATION OF:

- **tolerance,**
- **withdrawal ,**
  - special emphasis on tolerance and withdrawal:
    as **"dependence with a physiological component" = severe clinical course**
- taking larger amounts of alcohol over longer periods than intended,
- an inability to control use,
- spending a great deal of time associated with alcohol use,
- giving up important activities to drink,
- **continued use of alcohol despite physical or psychological consequences.**

ACUTE: in the EMERGENCY setting:

Intoxication is managed by observation until arrival of sobriety.

Youre mainly interested in keeping them stable until the drug and alcohol team takes over. This means:

ADDRESS THE PRESENTING PROBLEM: rehab cannot begin until the subdural hematoma is taken care of

ADDRESS WITHDRAWAL: Diazepam 5-20mg q2h manages the shakes and the fears, and keeps them nicely sedated until the D&A people get to seeing them. Midazolam if they misbehave.

ADDRESS VITAMIN DEFICIT: Not strictly speaking an ED issue, but if the patient does not move on to rehab at last they have had some intramuscular thiamine.
So: **THIAMINE 300mg STAT**

DETOXIFICATION: requires some motivation, even if it is in the form of a court order.

THIAMINE 100 mg po tds
VIGABATRIN 2000mg po daily for 3 weeks (anticonvulsant and mood stabilizer)
TEMAZEPAM 10-20mg nocte PRN
DIAZEPAM 5-20mg q2h – IF THE WITHDRAWAL IS SEVERE, i.e SCALE OVER 5
ACAMPROSATE (Campral) 666mg (yes, two 333mg pills) – uncertain activity but it seems to reduce alcohol craving, as does NALTREXONE but via a different mechanism

MAINTENANCE: requires A LOT of motivation; usually punctuated by episodes of relapse

!! MANAGE UNDERLYING MENTAL HEALTH CONDITION !!
Teach coping skills through CBT, psychotherapy and group therapy.
Consider a regular support group, Jesus-related or otherwise.
DISULFIRAM (Antabuse) is useless, no better than placebo in reducing relapse rates.
NALTREXONE appears to be better than placebo at reducing relapse
MIRTAZAPINE (Avansa) is a sedating antidepressant, evidence equivocal….
Support family and relationships with councelling

NEUROPHARMACOLOGY OF ETHANOL

Absorbed rapidly from **DUODENUM** by diffusion

Rate of delivery into duodenum Influenced by stomach emptying rate;
Rate of absorption influenced by CO2 in carbonated beverages
Rate of absorption lso influenced by food type (high-fat foods can impair the diffusion across the gut wall by retaining dissolved alcohol)

FIRST PASS METABOLISM IN THE LIVER

ethanol → acetaldehyde → acetate

Alcohol dehydrogenase
Easily saturated, maximal capacity about 7 to 10 grams per hour

Aldehyde dehydrogenase
not so easily saturated; requires a great amount of alcohol to saturate- and then acetaldehyde accumulates, contributing to a hangover

NADH NADPH

CYP-2E1; induceable enzyme
The greater the regular consumption, the more enzyme activity and thus more adverse effects

DISULFIRAM targets this enzyme:
Horrible aldehyde poisoning = unpleasantess and therefore hopefully abstinence…

CO2 + H2O

It is estimated that the plasma concentration necessary to cause threshold effects in humans is 5mM (40mg/100mL) while in a review of 808 fatal alcohol poisonings, the mean plasma concentration was 72mM To put blood concentrations further in context, the blood-alcohol limit in most states in the US is 0.08% wt/vol or 17.4 mM

MECHANISM OF INTOXICATION: effects in descending order of appearance

Receptor Affected	Effects Experienced
Delta-A GABA receptors = potentiated found primarily in the granule cells of the denate gyrus of the hippocampus and the granule cells of the cerebellum.	Mild euphoria, sociability, talkitiveness Increased self-confidence; decreased Inhibitions Diminution of attention, loss of judgment Beginning of sensory-motor impairment. Loss of efficiency in finer performance tests
Alpha-7 Nicotinic Ach receptors = potentiated influence rate of dopamine release in reward centres of the brain	
Glycine Receptor =potentiated spinal cord and brain stem inhibitory receptor, may be responsible for late respiratory depression	
NMDA glutamate receptor = inhibited Major excitatory receptor; involved in formation of memories among many other functions	Emotional instability; loss of critical Judgment, Impairment of perception, memory and comprehension Decreased sensitory response; increased reaction time, Reduced visual acuity; peripheral vision, and glare recovery. Sensory-motor incoordination; impaired balance. Drowsiness.
Voltage Gated Calcium Channels = inhibited The opening of voltage-gated calcium channels is responsible for the release of neurotransmitters throughout the body; thus inhibition results in decreased neurotransmitter release.	
GIRK Potassium Channels = inhibited G-protein coupled inward rectifying (GIRK) channel is a channel modulated by CB1-cannabinoid and Mu-opioid receptors (as well as many others); responsible for depressant effects and analgesia at high concentrations.	Disorientation, mental confusion; dizziness Exaggerated emotional states. Disturbances of vision and of perception, of color, form, motion and dimensions. Increased pain threshold. Increased muscular incoordination; staggering gait; slurred speechApathy, lethargy. General inertia; approaching loss of motor functions. Markedly decreased response to stimuli. Marked muscular incoordination; inabilityto stand or walk. Vomiting; incontinence Impaired consciousness; sleep or stupor.
AMPA and Kainate inhibition, Serotonin 5HT-3 potentiation: At doses in the realm of the ridiculous, these receptor subtypes are affected. The significance of this in fatalities related to alcohol is uncertain.	

EFFECTS AT LOW DOSE: Blood Alcohol 0.03 - 0.12

EFFECTS AT SOCIAL DOSE 0.09 - 0.25

EFFECTS AT HIGH DOSE: 0.25 - 0.40