

# Psychiatry 5<sup>th</sup> year



## *Chapter 7: Substance & Addictive Disorders*

Done by: Hala ALHmoud (018)

Re-Edited by: Lejan 021

# substance use disorder

- problematic pattern of substance use that leads to some form of functional impairment or distress
- Keep in mind that frequent use of a substance does not necessarily indicate a substance use disorder unless it is causing problems for the patient.

- Intoxication : condition caused by recent ingestion of a substance that alters a person consciousness , cognition , perception , judgment , affect and behavior
- Withdrawal : Physical &/ or mental effects that person experiences after stop using or reduce taking of a substance
- Withdrawal symptoms of a drug are usually the opposite of its intoxication effects. For example, alcohol is sedating, but alcohol withdrawal can cause brain excitation and seizures

## DIAGNOSIS AND DSM5 CRITERIA

manifested by at least two of the following within a 12-month period:

- Tolerance (needing higher amounts of the substance to achieve the desired effect and less effect of drugs over time)
- Withdrawal ( Symptoms that occur upon the abrupt discontinuation or decrease in the intake of drugs)
- Using substance more than originally intended.
- desire unsuccessful efforts to cut down
- Craving
- Significant time spent in obtaining, using, or recovering from substance.
- Failure to fulfill obligations at work, school, or home.
- Limiting social, occupational, or recreational activities because of substance use
- Limiting social, occupational, or recreational activities
- Use in dangerous situations (e.g., driving a car).
- Continued use despite subsequent physical or psychological problem



## EPIDEMIOLOGY

- One-year prevalence of any substance use disorder in the United States is approximately 8%.
- More common in men than women.
- Alcohol and nicotine are the most commonly used substances.

## PSYCHIATRIC SYMPTOMS

- Mood symptoms are common among persons with substance use disorders.
- Psychotic symptoms may occur with some substances.
- Personality disorders and psychiatric comorbidities (e.g., major depression, anxiety disorders) are common among persons with substance use disorders.
- It is often challenging to decide whether psychiatric symptoms are primary or substance-induced. Many patients may use substances to self-medicate for undertreated psychiatric symptoms.

## ACUTE INTOXICATION AND WITHDRAWAL

Both the intoxicated and withdrawing patient can present difficulties in diagnosis and treatment. Since it is common for persons to abuse several substances at once, the clinical presentation is often confusing, and signs/symptoms may be atypical. Always be on the lookout for use of multiple substances.

## TREATMENT OF SUBSTANCE USE DISORDERS

- Behavioral counseling should be part of every substance use disorder treatment.  
See Table 7-2.
- Psychosocial treatments are effective and include motivational intervention (MI), cognitive-behavioral therapy (CBT), contingency management, and individual and group therapy.
- For severe substance use disorders, residential (usually 28-day) “rehab” programs are common; some patients may choose to do partial hospitalization or intensive outpatient programming.

**TABLE 7-1. Direct Testing for Substance Use**

<b>Alcohol</b>	<ul style="list-style-type: none"> <li>■ Stays in system for only a few hours.</li> <li>■ Breathalyzer test, commonly used by law enforcement.</li> <li>■ Blood/urine testing more accurate.</li> <li>■ Urine screening for metabolite (ethyl glucuronide) — not useful for assessing acute intoxication, but can indicate alcohol use over the preceding 2–5 days.</li> </ul>
<b>Cocaine</b>	<ul style="list-style-type: none"> <li>■ Urine drug screen positive for 2–4 days (up to 8 days for heavy users).</li> </ul>
<b>Amphetamines</b>	<ul style="list-style-type: none"> <li>■ Urine drug screen positive for 1–3 days.</li> <li>■ Most assays have poor sensitivity and/or specificity.</li> </ul>
<b>Phencyclidine (PCP)</b>	<ul style="list-style-type: none"> <li>■ Urine drug screen positive for 4–7 days.</li> <li>■ OTC cold medications may yield false positive.</li> <li>■ Creatine kinase (CK) and aspartate aminotransferase (AST) are often elevated.</li> </ul>
<b>Sedative-hypnotics</b>	<p>In urine and blood for variable amounts of time.</p> <p><i>Barbiturates:</i></p> <ul style="list-style-type: none"> <li>■ Short-acting (pentobarbital): 24 hours</li> <li>■ Long-acting (phenobarbital): 3 weeks</li> </ul> <p><i>Benzodiazepines:</i></p> <ul style="list-style-type: none"> <li>■ Short-acting (e.g., lorazepam): up to 5 days</li> <li>■ Long-acting (diazepam): up to 30 days</li> </ul>
<b>Opioids</b>	<ul style="list-style-type: none"> <li>■ Urine drug test remains positive for 1–3 days, depending on opioid used.</li> <li>■ Routine screening tests detect morphine, which is the eventual metabolite of all natural opioids.</li> <li>■ Buprenorphine, synthetic opioids (methadone, fentanyl, tramadol) and semi-synthetic opioids (oxycodone, hydrocodone) will not be detected on routine screening (order separate assay).</li> </ul>
<b>Marijuana</b>	<p>Urine detection:</p> <ul style="list-style-type: none"> <li>■ After a single use, about 3 days. In heavy users, up to 4 weeks (THC is released from adipose stores).</li> </ul>



**TABLE 7-2. Stages of Change**

Stage	Definition	Example
<b>Precontemplation</b>	Patients do not view their addiction as a problem. They may see substance use as helpful and/or enjoyable.	A college student who drinks heavily feels that they need alcohol to overcome social anxiety and enjoy parties. They do not identify any negative consequences from their use.
<b>Contemplation</b>	The patient begins to think about cutting down or stopping altogether. They recognize potential benefits of making a change, but may be ambivalent or feel unable to do so.	The student misses several deadlines due to hangovers from drinking the night before. They think cutting down on alcohol might improve their grades, but aren't sure they want to stop.
<b>Preparation</b>	The patient plans for the process of change. They collect information, and may experiment with very small changes.	The student begins researching self-help strategies for reducing alcohol intake. They look up campus resources for individual and group therapy.
<b>Action</b>	The patient takes direct steps toward reducing or stopping substance use.	The student begins attending substance-use-focused groups on campus, and talks with their primary care doctor about starting naltrexone.
<b>Maintenance</b>	The patient has successfully made significant behavior change, and works to avoid relapse.	The student continues to drink, but limits themselves to 1–2 drinks per day, and only consumes alcohol on weekends.
<b>Relapse</b>	After a successful period of remission, patients resume substance use (or fall back into unhealthy patterns of use).	After graduating, the student is unemployed. They begin drinking again to cope with stress and unstructured time, and quickly escalates to near daily use.

Alcohol

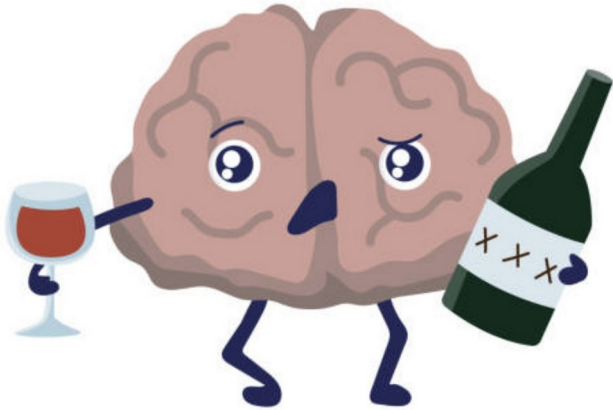
---



# Alcohol

- GABA receptors are inhibitory, and glutamate receptors are excitatory
- Alcohol activates (GABA), dopamine, and serotonin receptors in the central nervous system (CNS). It inhibits glutamate receptor activity and voltage-gated calcium channels.
- alcohol is a potent CNS depressant.

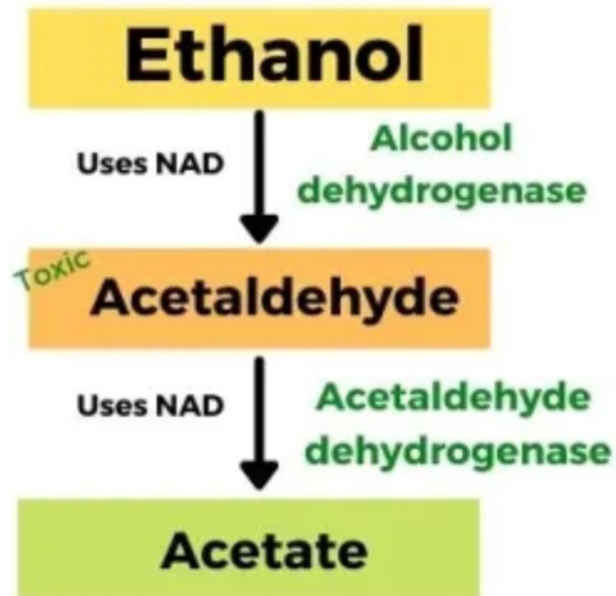
# Alcoholism



- Prevalence : 5% of women and 12% of men
- Alcohol is the most commonly used intoxicating substance in the United States.
- heavy drinking for men is more than 4 drinks per day or more than 14 drinks per week. For women, it is more than 3 drinks per day or more than 7 drinks per week.

# Alcohol metabolism

## Alcohol Metabolism



Genetic Lifehacks  
Learn. Experiment. Optimize.

- Enzymes associated with alcohol metabolism are upregulated in heavy drinkers

### KEY FACT

Most adults will show some signs of intoxication with BAL >100 and obvious signs with BAL >150 mg/dL.



# INTOXICATION

- Clinical Presentation
- The absorption and elimination rates of alcohol are variable and depend on many factors, including age, sex, body weight, chronic nature of use, duration of consumption, food in the stomach, and the state of nutrition and liver health

**TABLE 7 - 2 . Clinical Presentation of Alcohol Intoxication**

EFFECTS	BAL
Impaired fine motor control	20–50 mg/dL
Impaired judgment and coordination	50–100 mg/dL
Ataxic gait and poor balance	100–150 mg/dL
Lethargy, difficulty sitting upright, difficulty with memory, nausea/vomiting	150–250 mg/dL
Coma (in the novice drinker)	300 mg/dL
Respiratory depression, death possible	400 mg/dL

# Treatment

- **Monitor**: Airway, breathing, circulation, glucose, electrolytes, acid–base status.
  - **Thiamine and folate** Remember thiamine must be given before glucose, as it's a necessary cofactor for glucose metabolism
  - **Naloxone**
  - CT) scan of the head to rule out subdural hematoma or other brain injury.
  - Severely intoxicated patients may require mechanical ventilation
  - Gastrointestinal evacuation is not indicated in the treatment of EtOH overdose unless a significant amount of EtOH was ingested within the preceding 30–60 minutes
- ▷ with attention to acid-base balance, temperature, and electrolytes while they are recovering.

# WITHDRAWAL

**TABLE 7-3. Alcohol Withdrawal Symptoms**

<i>Alcohol withdrawal symptoms</i> usually begin in 6–24 hours after the last drink and may last 2–7 days.
<i>Mild:</i> Irritability, tremor, insomnia.
<i>Moderate:</i> Diaphoresis, hypertension, tachycardia, fever, disorientation.
<i>Severe:</i> Tonic-clonic seizures, DTs, hallucinations.

- Alcohol withdrawal is potentially lethal !

## *Clinical Presentation*

- Signs and symptoms of **alcohol withdrawal syndrome** include insomnia, anxiety, hand tremor, irritability, anorexia, nausea, vomiting, autonomic hyperactivity (diaphoresis, tachycardia, hypertension), psychomotor agitation, fever, seizures, hallucinations, and delirium tremens (see Table 7-4).
- The earliest symptoms of EtOH withdrawal begin between 6 and 24 hours after the patient's last drink and depend on the duration and quantity of EtOH consumption, liver size, and body mass.
- Generalized tonic-clonic seizures usually occur between 12 and 48 hours after cessation of drinking, with a peak around 12–24 hours.
- About a third of persons with seizures develop delirium tremens (DTs).
- Hypomagnesemia may predispose to seizures; thus, it should be corrected promptly.
- Seizures are treated with benzodiazepines. Long-term treatment with anticonvulsants is not recommended for alcohol withdrawal seizures.

**TABLE 7-4. Timing of Alcohol Withdrawal Symptoms**

Syndrome	Clinical Findings	Onset After Last Drink
Minor withdrawal	Tremulousness, mild anxiety, headache, diaphoresis, palpitations, anorexia, gastrointestinal upset; normal mental status	6 to 36 hours
Seizures	Single or brief flurry of generalized tonic-clonic seizures, short postictal period, status epilepticus rare	6 to 48 hours
Alcoholic hallucinosis	Visual, auditory, and/or tactile hallucinations with intact orientation and normal vital signs	12 to 48 hours
Delirium tremens	Delirium, agitation, tachycardia, hypertension, fever, diaphoresis	48 to 96 hours

Source: Used, with permission, from Hoffman RS, Weinhouse GL. Management of moderate and severe alcohol withdrawal syndromes. <https://www.uptodate.com/contents/management-of-moderate-and-severe-alcohol-withdrawal-syndromes>. © 2021 UpToDate, Inc. and/or its affiliates. All Rights Reserved.

# Delirium Tremens

- the most serious form of EtOH withdrawal.
- involving mental status and neurological changes. Symptoms include disorientation, agitation, visual and tactile hallucinations, and autonomic instability (increase in respiratory rate, heart rate, and blood pressure)
- begins 48–96 hours after the last drink
- While only 5% of patients who experience EtOH withdrawal develop DTs, there is a roughly 5% mortality rate (up to 35% if left untreated)
- Age >30 and prior DTs increase the risk
- It is a medical emergency and should be treated with adequate doses of benzodiazepines

# Treatment

- Carbamazepine or valproic acid can be used in mild withdrawal.
- **Lorazepam, Diazepam, Chlordiazepoxide**
- Benzodiazepines
- thiamine, folic acid (Banana Bag)
- Electrolyte and fluid abnormalities must be corrected
- Check for signs of hepatic failure

Providers must pay careful attention to the level of consciousness, and consider the possibility of traumatic injuries.

# Alcoholic ketoacidosis

- seen in the setting of alcohol cessation after an alcohol binge secondary to protracted vomiting and lack of oral intake.
- Hallmark is ketosis without hyperglycemia and a negative alcohol level.
- high anion gap metabolic acidosis, ketonemia, and low levels of potassium, magnesium, and phosphorus.
- Treatment of hydration and replacing electrolytes.

↳ with D5NS





# ALCOHOL USE DISORDER

- the AUDIT-C is used to screen for alcohol use disorder
- Biochemical markers are useful in detecting recent prolonged drinking; ongoing monitoring of biomarkers can also help detect a relapse
- biomarkers are BAL, liver function tests (AST, ALT, GGT), and MCV.

# AUDIT-C

TABLE 7-4. AUDIT-C

**QUESTION #1: HOW OFTEN DID YOU HAVE A DRINK CONTAINING ALCOHOL IN THE PAST YEAR?**

■ Never	(0 points)
■ Monthly or less	(1 point)
■ Two to four times a month	(2 points)
■ Two to three times per week	(3 points)
■ Four or more times a week	(4 points)

**QUESTION #2: HOW MANY DRINKS DID YOU HAVE ON A TYPICAL DAY WHEN YOU WERE DRINKING IN THE PAST YEAR?**

■ 1 or 2	(0 points)
■ 3 or 4	(1 point)
■ 5 or 6	(2 points)
■ 7 to 9	(3 points)
■ 10 or more	(4 points)

**QUESTION #3: HOW OFTEN DID YOU HAVE SIX OR MORE DRINKS ON ONE OCCASION IN THE PAST YEAR?**

■ Never	(0 points)
■ Less than monthly	(1 point)
■ Monthly	(2 points)
■ Weekly	(3 points)
■ Daily or almost daily	(4 points)

The AUDIT-C is scored on a scale of 0–12 (scores of 0 reflect no alcohol use). In men, a score of 4 or more is considered positive; in women, a score of 3 or more is considered positive.

# Medications for Alcohol Use Disorder

- **First-line** treatments:

- 1- Naltrexone (Revia, IM-Vivitrol):

- Opioid receptor antagonist
- Reduces craving

- 2- Acamprosate (Campral):

- Use for relapse prevention in patients who have stopped drinking (post-detoxification)
- Major advantage is that it can be used in patients with liver disease.
- Contraindicated in severe renal disease.

- **Second-line** treatments

- 1- Disulfiram (Antabuse):

- Contraindicated in severe cardiac disease, pregnancy, and psychosis.

- 2- Topiramate (Topamax) :

- Anticonvulsant
- Reduces cravings

**TABLE 7-6. Pharmacological Treatment of Alcohol Use Disorder**

Medication	Mechanism	Pros	Cons
Naltrexone	Opioid receptor antagonist; reduces cravings and the “high” associated with alcohol intoxication.	First-line treatment.  Available as an oral tablet (can be taken daily, or as-needed on drinking days), or monthly injection. Can allow some patients to engage in moderate alcohol use without escalating to binge drinking.	Will precipitate withdrawal in patients with physical opioid dependence. Can interfere with anesthesia (e.g., for acute injury or planned surgeries). Risk of LFT elevation.
Acamprosate	Likely modulates glutamate transmission.	First-line treatment.  Can be used for patients with liver disease. Typically used for relapse prevention in patients who have already stopped drinking.	Contraindicated in severe renal disease.
Disulfiram	Blocks aldehyde dehydrogenase, causing buildup of acetaldehyde and aversive symptoms (flushing, headache, nausea/vomiting, palpitations, shortness of breath).	Second-line. Can be effective for highly motivated patients.	Medication adherence can be an issue. Contraindicated in severe cardiac disease, pregnancy, psychosis. Must monitor LFTs.
Topiramate	Anticonvulsant; potentiates GABA and inhibits glutamate receptors.	Second-line treatment. Reduces cravings for alcohol, and decreases alcohol use.	Common adverse effects: impaired cognition (“DOPE-a-max”), nausea / weight loss, metabolic acidosis.

# Long-Term Complications of Alcohol Intake

---

- Wernicke's encephalopathy : Caused by thiamine (vitamin B1) deficiency
- Ataxia , confusion , ocular abnormalities
- Acute and can be reversed with thiamine therapy
- If left untreated, Wernicke's encephalopathy may progress to Korsakoff syndrome :
- Chronic amnestic syndrome
- Impaired recent memory, anterograde amnesia, compensatory confabulation
- Reversible in only about 20% of patients

# Opioids

---



- stimulate mu, kappa, and delta opiate receptors
- analgesia, sedation, and dependence
- Examples : heroin, oxycodone, codeine, dextromethorphan, morphine, methadone, and meperidine (Demerol).
- Opioids are associated with more deaths (usually due to unintentional overdose) than any other drug.



- Prescription opioids (OxyContin [oxycodone], Vicodin [hydrocodone/acetaminophen], and Percocet [oxycodone/acetaminophen])—not heroin—are the most commonly used opioids.
- Behaviors such as losing medication, “doctor shopping,” and running out of medication early should alert clinicians of possible misuse.
- Opioids are associated with more deaths (usually due to unintentional overdose) than any other drug.

# Heroin

- processed from morphine
- Using heroin regularly results in tolerance
- Heroin is highly addictive
- How it is used (forms) ?
- Injections, Sniffing and Smoking.



# INTOXICATION

- Drowsiness, nausea, vomiting, sedation, decrease in pain perception, decrease in gastrointestinal motility, **pupil constriction**, and respiratory depression (which can be fatal)
- Meperidine and monoamine oxidase inhibitors taken in combination may cause serotonin syndrome: hyperthermia, confusion, hypertension or hypotension, and hyperreflexia



## KEY FACT

Meperidine is the exception to opioids producing miosis.  
"Demerol Dilates pupils."

# Treatment of intoxication

- Ensure adequate airway, breathing, and circulation
- In overdose : naloxone (an opioid antagonist ) improves respiratory depression but may cause severe withdrawal in an opioid-dependent patient
- Ventilatory support may be required.
- Patients at risk of opioid overdose should be prescribed a naloxone (Narcan) kit to keep at home for emergencies

# OPIATE USE DISORDER

TABLE 7-7. Pharmacological Treatment of Opioid Use Disorder

Medication	Mechanism	Pros	Cons
Methadone	Full agonist at mu-opioid receptor.	Administered once daily. Long half life.	Restricted to federally licensed substance abuse treatment programs. Can cause QTc interval prolongation: screening electrocardiogram is indicated, particularly in patients with high risk of cardiac disease.
		Presenting to the methadone clinic for regular pickups can be helpful for patients who benefit from daily structure and access to group therapy or case management.	Patients can still use other opioids on top of methadone.
Buprenorphine	Partial opioid receptor agonist—can precipitate withdrawal if used too soon after full opioid agonists.	Sublingual preparation that is safer than methadone, as its effects reach a plateau and make overdose unlikely.	In the outpatient setting, can only be prescribed by a physician with a special waiver on their controlled substances license.
		Combined formulation (buprenorphine-naloxone, or Suboxone) prevents intoxication from intravenous or intranasal use.	
Naltrexone	Competitive opioid antagonist, precipitates withdrawal if used within 7 days of heroin use	Available as daily oral medication or monthly depot injection. It is a good choice for highly motivated patients such as health care professionals.	Adherence is an issue for oral formulation. Risk of LFT elevation. Can interfere with anesthesia (e.g., for acute injuries or surgical procedures).
Naloxone	Competitive opioid antagonist, used in treatment of overdose.	Can be life-saving for patients or their peers, and should routinely be prescribed for all patients with opioid use disorder (especially for those who are receiving medication-assisted treatment).	Does not reduce opioid use or treat symptoms of opioid use disorder.  Very short half-life; patients must be educated about need to call EMS or present to ED after it's administered (even if the overdose appears to be reversed).

# WITHDRAWAL :

- not life threatening, abstinence in the opioid-dependent individual leads to an unpleasant withdrawal syndrome
- withdrawal symptoms of opiates: flu-like symptoms (body aches, anorexia, rhinorrhea, fever), diarrhea, anxiety, insomnia, and piloerection dysphoria, insomnia, lacrimation, rhinorrhea, yawning, weakness, sweating , **dilated pupils**, abdominal cramps,
- arthralgia/myalgia, hypertension, tachycardia

# Treatment

- **Moderate symptoms:** Symptomatic treatment with clonidine (for autonomic signs and symptoms of withdrawal), nonsteroidal anti inflammatory drugs (NSAIDs) for pain, loperamide for diarrhea, dicyclomine for abdominal cramps, etc.
- **Severe symptoms:** Detox with buprenorphine or methadone.
- Monitor degree of withdrawal with COWS (Clinical Opioid Withdrawal Scale), which uses objective measures (i.e., pulse, pupil size, tremor) to assess withdrawal severity



# SUBDTANCE USE DISORDERS







# PSYCHIATRIC SYMPTOMS

- Mood symptoms
- Psychotic symptoms
- Personality disorders
- psychiatric comorbidities (e.g., major depression, anxiety disorders)

# 1- COCAINE

- Cocaine blocks the reuptake of dopamine, epinephrine, and norepinephrine from the synaptic cleft, causing a stimulant effect
- Dopamine plays a role in the behavioral reinforcement (“reward”) system of the brain
- Route of administration: intranasally, inhallatinal, IV, & orally

Since cocaine is an indirect sympathomimetic, intoxication mimics the fight-or-flight response.



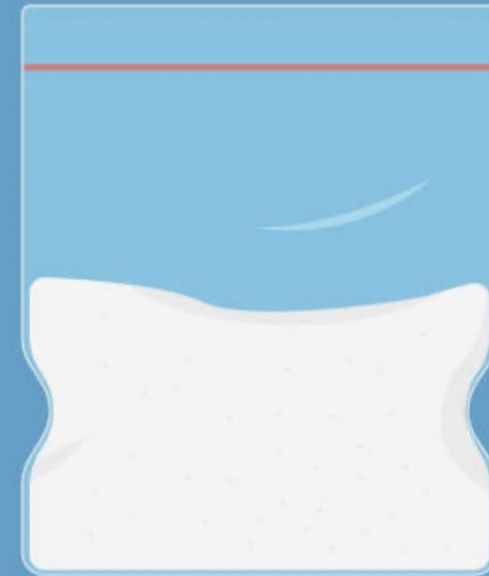
# INTOXICATION

- **General** : Euphoria, heightened self-esteem, increase or decrease in blood pressure, tachycardia or bradycardia, nausea, dilated pupils, weight loss, psychomotor agitation or depression, chills, and sweating.
- **Dangerous** : Seizures, cardiac arrhythmias, hyperthermia, paranoia, and Hallucinations
- **Deadly** : vasoconstrictive effect may result in MI, intracranial hemorrhage, or stroke.
- **Management :**
  - mild-to-moderate agitation and anxiety: **Reassurance of the patient and benzodiazepines.**
  - severe agitation or psychosis: **Antipsychotics** (haloperidol )
  - Symptomatic support (i.e., control hypertension, arrhythmias)
  - Temperature of >102°F should be treated aggressively with an ice bath, cooling blanket, and other supportive measures.

# Treatment of cocaine use disorder

- there is no (FDA)-approved pharmacotherapy for cocaine use disorder.
- Off-label medications are sometimes used (naltrexone, modafinil, topiramate).
- Psychological interventions (contingency management, relapse prevention, NA, etc.) are the mainstay of treatment

## COCAINE



# Withdrawal

- Usually **not life-threatening**
- post-intoxication depression (“**crash**”): Malaise, fatigue, hypersomnolence, depression, anhedonia, hunger, constricted pupils, patients can become suicidal.
- With mild-to-moderate cocaine use, withdrawal resolve within 72 hours ,With heavy chronic use last for 1–2 weeks
- Treatment is supportive



## 2- Amphetamines

- Block reuptake and facilitate release of dopamine and norepinephrine from nerve endings, causing a stimulant effect
- Examples : Dextroamphetamine (Dexedrine), methylphenidate (Ritalin), methamphetamine (Desoxyn, “ice,” “speed,” “crystal meth,” “crank”).
- Methamphetamines are easily manufactured in home laboratories , using over-the-counter medications (e.g., pseudoephedrine).
- Methamphetamines are used medically in the treatment of : (ADHD), binge eating, and occasionally depressive disorders.



■ Substituted (“designer,” “club drugs”) amphetamines:

- Release dopamine, norepinephrine, and serotonin from nerve endings.
- *Examples:* MDMA (“ecstasy”), MDEA (“eve”).
- Often used in dance clubs and raves.
- Have both stimulant and hallucinogenic properties.
- Serotonin syndrome is possible if designer amphetamines are combined with selective serotonin reuptake inhibitors (SSRIs).

# INTOXICATION

- similar to those of cocaine
- Amphetamine withdrawal can cause prolonged depression
- **Complications** of their long half-life can cause ongoing psychosis, even during abstinence.
- **Overdose** : hyperthermia, dehydration , rhabdomyolysis, and renal failure.
- **Treatment** : Rehydrate, correct electrolyte balance, and treat hyperthermia

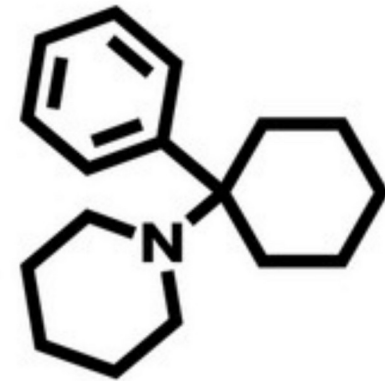




# 3- Phencyclidine (PCP)

- “angel dust,” is a dissociative, hallucinogenic drug that antagonizes (NMDA) glutamate receptors and activates dopaminergic neurons
- . It can have stimulant or CNS depressant effects, depending on the dose
- PCP can be smoked as “wet” (sprinkled on cigarette) or as a “joint” (sprinkled on marijuana).

■ Ketamine is similar to PCP, but is less potent. Ketamine is sometimes used as a “date rape” drug, as it is odorless and tasteless.



phencyclidine

# INTOXICATION

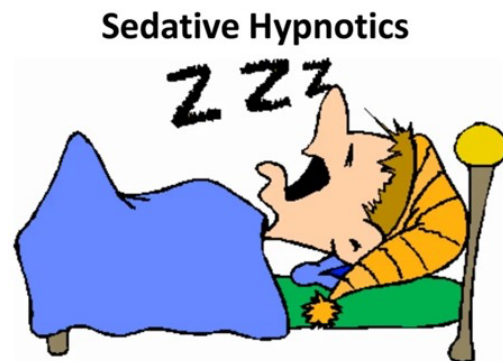
- agitation, depersonalization, hallucinations, synesthesia (one sensory stimulation evokes another —e.g., hearing a sound causes one to see a color )
- impaired judgment, memory impairment, combativeness, nystagmus, ataxia, dysarthria, hypertension, tachycardia, muscle rigidity, and high tolerance to pain.
- Overdose can cause seizures, delirium, coma, and even death.
- **Treatment :**
  - Monitor vitals
  - benzodiazepines ( to treat agitation, anxiety, muscle spasms, and seizures)
  - antipsychotics (to control severe agitation or psychotic symptoms.)

# WITHDRAWAL

- No withdrawal syndrome, but “flashbacks” (recurrence of intoxication symptoms due to release of the drug from body lipid stores) may occur

# 4- Sedative-Hypnotics

- Benzodiazepines (BZDs)
- used in treatment of anxiety disorders
- Easily obtained via prescription from physicians' offices
- Potentiate the effects of GABA by modulating the receptor, thereby increasing frequency of chloride channel opening.



- Barbiturates
- Used in the treatment of epilepsy and as anesthetics
- Potentiate the effects of GABA by binding to the receptor and increasing duration of chloride channel opening.
- At high doses, barbiturates act as direct GABA agonists, and therefore have a lower margin of safety relative to BZD
- respiratory depression can occur

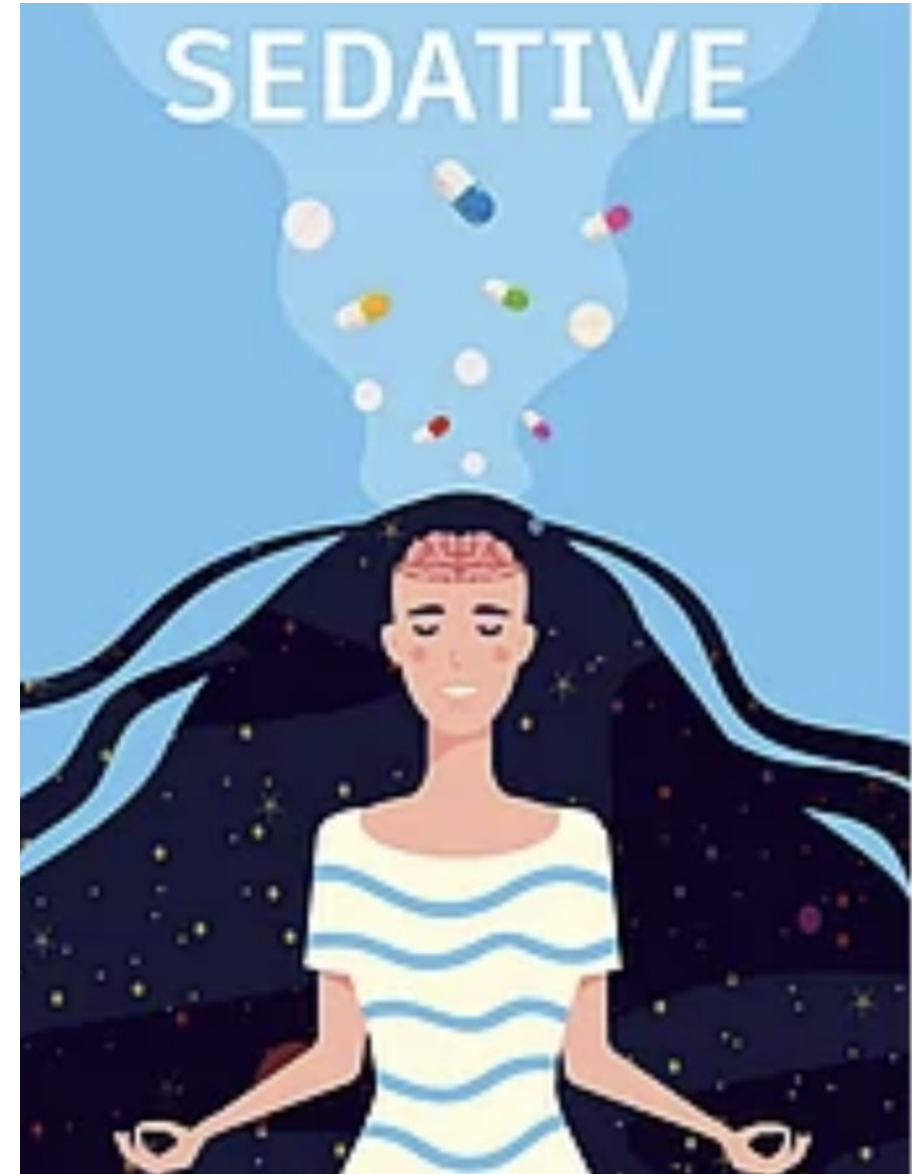
# INTOXICATION

- drowsiness, confusion, hypotension, slurred speech, incoordination, ataxia, mood lability, impaired judgment, nystagmus, respiratory depression, and coma or death in overdose.
- Long-term sedative use may lead to dependence and may cause depressive symptoms

■ Symptoms are synergistic when combined with EtOH or opioids/narcotics.

- **Treatment** : Maintain airway, breathing, and circulation. Monitor vital signs.
- barbiturates only :Alkalinize urine with sodium bicarbonate to promote renal excretion.
- benzodiazepines only: Flumazenil in overdose.

■ Activated charcoal and gastric lavage to prevent further gastrointestinal absorption (if drug was ingested in the prior 4-6 hours).



# WITHDRAWAL

- life threatening.
- Signs and symptoms of withdrawal are the same as these of EtOH withdrawal. Tonic-clonic seizures may occur
- **Treatment :**
  - Benzodiazepines (stabilize patient, then taper gradually).
  - carbamazepine or valproic acid taper not as beneficial.

# 5- Hallucinogens

Pharmacological effects vary, but LSD is believed to act on the serotonergic system. Hallucinogens do not cause physical dependence or withdrawal, though users can rarely develop psychological dependence.

- psilocybin (mushrooms), mescaline (peyote cactus), and lysergic acid diethylamide (LSD)
- **INTOXICATION**: perceptual changes (illusions, hallucinations, body image distortions, synesthesia), labile affect, dilated pupils, tachycardia, hypertension, hyperthermia, tremors, incoordination, sweating, and palpitations.
- lasts 6–12 hours
- **Treatment**: Monitor for dangerous behavior and reassure patient, Use benzodiazepines as first-line for agitation
- No withdrawal syndrome







## 6- Marijuana

- Cannabis (“marijuana,” “pot,” “weed,” “grass”)
- Marijuana has shown some efficacy in treating nausea and vomiting in chemotherapy patients, increasing appetite in AIDS patients, in chronic pain and lowering intraocular pressure in glaucoma

■ The main psychoactive component which produces the “high” in cannabis is THC (tetrahydrocannabinol).

■ Cannabinoid receptors in the brain inhibit adenylate cyclase.



# INTOXICATION

- euphoria, anxiety, impaired motor coordination, perceptual disturbances mild tachycardia, anxiety, conjunctival injection (red eyes), dry mouth, and increased appetite ↗ (The Munchies)
- Cannabis-induced psychotic disorders with paranoia, hallucinations, or delusions may occur
- Chronic use may cause respiratory problems such as asthma and chronic bronchitis, immunosuppression, cancer
- **Treatment:** Supportive, psychosocial interventions (e.g., contingency management, groups)



# Withdrawal

- irritability, anxiety, restlessness, aggression, strange dreams, depression, headaches, sweating, chills, insomnia, and low appetite.
- **Treatment:** Supportive and symptomatic

## 7- Inhalants

- drugs that are inhaled and absorbed through the lungs
- CNS depressants
- m/c used by preadolescents or adolescents rate of use is similar between boys and girls (but rare in adult females).
- Examples: Solvents, glue, paint thinners, fuels, isobutyl nitrates

# INTOXICATION

- **Effects:** paranoia, lethargy, dizziness, nausea/ vomiting, headache, nystagmus, tremor, muscle weakness Perceptual disturbances,
  - Acute intoxication: 15–30 minutes.
  - **Overdose:** May be fatal secondary to respiratory depression or cardiac arrhythmias.
  - **Treatment:** Monitor airway, breathing, and circulation; may need oxygen with hypoxic states.
  - **withdrawal :** does not usually occur, but symptoms may include irritability, sleep disturbance, anxiety, depression, nausea, vomiting, and craving.
- Long-term use may cause permanent damage to CNS (e.g., neurocognitive impairment, cerebellar dysfunction, Parkinsonism, seizures), peripheral neuropathy, myopathy, aplastic anemia, malignancy, metabolic acidosis, urinary calculi, glomerulonephritis, myocarditis, MI, and hepatotoxicity.



## 8- Caffeine

- is the most commonly used psychoactive substance
- form of coffee, tea, or energy drinks
- adenosine antagonist causing increase (cAMP) stimulating the release of excitatory neurotransmitters
- Stimulant

# OVERDOSE

- More than 250 mg (2 cups of coffee): Anxiety, insomnia, muscle twitching, rambling speech, flushed face, diuresis, gastrointestinal disturbance, restlessness, excitement, and tachycardia
- More than 1 g: tinnitus, severe agitation, visual light flashes, and cardiac arrhythmias
- More than 10 g: Death may occur secondary to seizures and respiratory failure
- **Treatment:** Supportive and symptomatic.

# WITHDRAWAL

- occur in 50–75% of caffeine users
- headache, fatigue, irritability, nausea, vomiting, drowsiness, muscle pain, and depression
- resolves within 1½ weeks.



# 9- Nicotine

- derived from the tobacco plant, It is highly addictive through its effects on the dopaminergic system
- causes both tolerance and physical dependence
- Cigarette smoking is the leading cause of preventable morbidity and mortality
- (COPD), cardiovascular diseases, and various cancers
- 15% of U.S. adults
- **Effects:** Restlessness, insomnia, anxiety, and increase in GI motility
- **Withdrawal symptoms:** Intense craving, dysphoria, anxiety, poor concentration, increase in appetite, weight gain, irritability, restlessness, and insomnia
- Cigarette smoking during pregnancy is associated with low birth weight, (SIDS) Sudden infant death syndrome , and a variety of postnatal morbidities.



# TREATMENT OF NICOTINE DEPENDENCE

- Nicotine replacement therapy (NRT): Available as transdermal patch, gum, lozenge, nasal spray, and inhaler
  - Varenicline (Chantix)
  - Bupropion (Zyban)
  - Behavioral support
- تفاضل الأدوية
- Varenicline (Chantix):  $\alpha 4\beta 2$  nicotinic cholinergic receptor (nAChR) partial agonist that mimics the action of nicotine, reducing the rewarding aspects and preventing withdrawal symptoms.
  - Bupropion (Zyban): Antidepressant inhibits reuptake of dopamine and norepinephrine; helps reduce craving and withdrawal symptoms.
  - Nicotine replacement therapy (NRT): Available as transdermal patch, gum, lozenge, nasal spray, and inhaler.
  - Behavioral support/counseling should be part of every treatment.
  - Relapse after abstinence is common.

# Gambling Disorder

## DIAGNOSIS AND *DSM-5* CRITERIA

Persistent and recurrent problematic gambling behavior, as evidenced by four or more of the following in a 12-month period:

1. Preoccupation with gambling.
2. Need to gamble with increasing amount of money to achieve pleasure.
3. Repeated and unsuccessful attempts to cut down on or stop gambling.
4. Restlessness or irritability when attempting to stop gambling.
5. Gambling when feeling distressed (depressed, anxious, etc.).
6. Returning to reclaim losses after gambling (“get even”).
7. Lying to hide level of gambling.
8. Jeopardizing relationships or job because of gambling.
9. Relying on others to financially support gambling.

## EPIDEMIOLOGY/ETIOLOGY

- Prevalence: 0.4–1.0% of adults in the United States.
- Men represent most of the cases.
- More common in young adults and middle-aged, and lower rates in older adults.
- Similar to substance use disorders, the course is marked by periods of abstinence and relapse.
- Increased incidence of mood disorders, anxiety disorders, substance use disorders, and personality disorders.
- Etiology may involve genetic, temperamental, environmental, and neurochemical factors.
- One-third may achieve recovery without treatment.

## TREATMENT

- Participation in Gamblers Anonymous (a 12-step program) is the most common treatment.
- Cognitive-behavioral therapy has been shown to be effective, particularly when combined with Gamblers Anonymous.
- Important to treat comorbid mood disorders, anxiety disorders, and substance use disorders where appropriate.

THANK YOU