Insomnia

- Stage of Sleep
  - Eyes Closed (awake) 1% Alpha/Beta Waves
  - Stage I (light sleep) 5% Theta Waves
  - Stage II (moderate sleep) 45% Sleep Spindles and K-Complexes
  - Stage III-IV (deep sleep) 25% Delta Waves, Parasomnias occur here
  - REM Sleep 25% Sawtooth Waves, REM, Dreams, Erection, Increase in HR/BP, lack of motor tone, Q1.5hrs lasting 10-40min, decreases w/ age
- **Dyssomnias** (disturbance in amount/quality/timing of sleep) = Insomnia, Hypersomnia, Narcolepsy
- **Parasomnias** (abnormal events during sleep) = Nightmare Disorder, Night Terror Disorder, Somnambulism
- Find out what kind of insomnia pt has had (trouble falling asleep, trouble staying asleep, walking up to early)
- Address sleep hygiene (no alcohol, no caffeine, no tobacco, no large meals, no daytime napping, no exercise, consistent sleep schedule, relaxation therapies, "Sleepy Time Tea" which has the herbal valerian, warm milk, herbal 5-HTPs, herbal GABA, melatonin etc, bed is only for "2 things")
- Assess secondary causes if pt has chronic insomnia: The Big Three (pain, anxiety, delirium), Psych (mood disorders, anxiety disorders, etc), Pulm (O/CSA, allergies, COPD, etc), GI (GERD, IBS, etc), Neuro (restless leg syndrome, peripheral neuropathies, etc), Chronic Medical Conditions (DM, arthritis, etc), Meds (decongestants, beta-agonists, corticosteroids, diuretics, antidepressants, histamine-2-blockers, etc)
- Mild Insomnia
  - Benzodiazepine-Like Agonist: (only act on Type 1 GABA receptor and thus have no Benzo SEs or withdrawal effects and can only Tx insomnia with no daytime sleepiness)
    - Short Half Life (~3hr): zalelpon (Sonata) or zolpidem (Ambien)
    - Intermediate Half-Life (~6hrs): zolpidem (Ambien-CR) or eszopiclone (Lunesta)
  - Antidepressants (great for insomnia 2/2 depression, good for pts having trouble staying asleep)
    - trazodone (Desyrel)
    - Doxepin (Sinequan)
    - Amitryptylin (Elavil)
    - Mirtazepine (Rameron)
  - First-Generation Antihistamine
    - Intermediate Half -Life (~8hrs): hydroxyzine (Vistaril)
  - Melatonin-Receptor Agonist
    - Short Half –Life (~3hrs): ramelteon (Rozerem)
  - Other
    - Intermediate Half-Life (~8hrs): chloral hydrate (Chloral Hydrate)
- Moderate Insomnia
  - Benzodiazepines
    - Short Half-Life (~3hrs): triazolam (Halcion)
    - Intermediate Half –Life (~10hrs): temazepam (Restoril)
    - Long Half-Life (~16hrs): estazolam (Prosom)
- Severe Insomnia
  - Cognitive Behavioral Therapy (CBT)
  - Polysomnography

Full Mental Status Exam

- **General** (general description of pt) appearance, motor activity, speech (pace, fluency, quantity, quality, etc), etc
- **Mood & Affect** (are they congruent?)
  - **Mood** (pt's subjective report of his/her emotional state)
    - Euthymia (nl), dysphoria (sad), euphoria (happy)
  - **Affect** (doctor's observation of pt's emotional state)
    - Full (appropriate), Blunted (only one or a few affects but appropriate), Flat (no affect), labile (inappropriate)
- **Thinking**
  - **Content** (what the pt is actually thinking about) delusions, paranoia, persecution, pre-occupations, obsessions, compulsions, phobias, suicidal/homicidal ideations, etc
  - **Process** (the way the pt puts ideas together) loose associations, forming associations, racing thoughts/pressured speech, tangentiality, circumstantiality, thought blocking/insertion/withdrawal, word salad, neologisms, echolalia, perseveration, etc
- **Perception** (normally pt should have none) hallucinations, depersonalization, derealization, illusion, etc
- **Judgment** (degree of rational decision making capability eg if you smell smoke what do you do?) & **Insight** (degree of rational understanding of a pt about his/her condition)
- **MMSE** (refer)

DSM-IV (Diagnostic and Statistical Manual of mental disorders 4th edition)

- Axis 1: Acute Psychiatric Problem
- Axis 2: Chronic Personality Disorder
- Axis 3: General Medical Problems
- Axis 4: Psychosocial Problems
- Axis 5: Global Assessment of Functioning (GAF) Scale from 1-100

General Treatment

- Psychotherapy
  - **Behavior Therapy** (used in Tx anxiety disorders, incremental exposure to what causes anxiety)
  - **Group Therapy** (used in Tx for any chronic psych disorder, groups of people provide peer feedback and help pt realize s/he is not alone)
  - **Family Therapy** (used in Tx of any child psych disorder, family is present)
  - **Marital Therapy** (used in Tx of marital problems, goal is to facilitate communication)
  - **Supportive Therapy** (used in Tx of acute stress in pts previously well, teaches coping mechanisms)
  - **Interpersonal Therapy** (used in Tx of mood disorders)
  - **Insight Oriented Psychodynamic Therapy** (used in Tx of PDs and mood disorders)
  - **Psychoanalysis** (used in Tx of ?, includes free association, dream interpretation, therapeutic alliance, transference, counter-transference)
    - Based on Freud's theory that behaviors result from unconscious mental processes specifically the conflicts between ego, id, superego and the external reality
    - Defense mechanism are used by the ego to protect the id from conflicts, the four principle normal ones are altruism, humor, sublimation, suppression, there are various bad ones including rationalizing, repression, acting out, splitting, dissociation, projection, rejection, denial
  - **Cognitive Behavior Therapy (CBT)** (used in Tx of depression/anxiety/substance abuse disorders, recognize pessimistic thoughts about self and replace w/ + thoughts)
- Medications
  - **NEpi**
    - Ritalin/Strettera then Adderral
  - **SNDIs (Serotonin and NorEpi and Dopamine Inhibitors) aka MonoAmine Oxidase Inhibitors (MAOIs)**
    - Types
      - Phenelzine (Nardil)
      - Tranylcypromine (Parnkle)
      - Isocarboxazid (Marplan)
    - SEs: (1) paradoxic orthostatic hypoTN, (2) if given w/ high tyramine containing foods (beer, wine, chesses, smoked meats, chianti, fava beans, etc) the tyramine is like tyrosine and is converted to NorEpi and in the presence of MAOIs you can have very high [ ] of NorEpi causing a sympathomimetic crisis w/ very high HTN
    - OD: Tx w/ ativan
  - **SNRIs (Serotonin and NEpi Reuptake Inhibitors) aka Tri/TetraCyclic Antidepressants (TCAs)**
    - Types
      - Des-ipramine (Norpramin) least HM SEs, only stimulating hormone
      - Pro-triptyline (Vivactil)
      - Nor-triptyline (Aventyl) least A SEs
      - Ami-tryptiline (Elavil) take at night, neuropathic pain
      - Clom-ipramine (Anafranil)
      - Doxepin (Sinequin) itching
      - Im-ipramine (Tofranil) used for enuresis
      - Mirtazapine (Remeron) used for sleep, increases appetite
      - Venlafaxine (Effexor ± XR) can cause HTN, used for sleepy depression
      - Duloxetine (Cymbalta) can cause liver failure, used for sleepy depression
    - SEs: (1) serotonin syndrome, (2) AV block and Prolonged QT, (3) Seizures, (4) Coma, (4) Block HAM
    - OD: Tx w/ NaHCO3
  - **SRIs (selective Serotonin Reuptake Inhibitors)** (used in Anxiety/Depression)
    - Types
      - Fluoxetine (Prozac) third best
      - Fluvoxamine (Luvox) mainly only used for Anxiety Disorders only
      - Paroxetine (Paxil) many SEs
      - Citalopram (Celexa) cheapest but fourth best
      - Escitalopram (Lexapro) first best
      - Sertraline (Zoloft) second best, best for pts w/ CV dz
      - NB St. John's Wort (Hypericum perforatum)
    - SEs: (1) Serotonin Syndrome, (2) Discontinuation Syndrome (when SRIs are suddenly stopped pt can develop dizziness, fatigue, HA, N, insomnia, flu-like Sx, not dangerous but unpleasant therefore taper), (3) Drug Interactions (SRIs affect P450 system), (4) 5HT1 (anti-depressive effects, weight gain), 5HT2 (short term HA, anorexia, insomnia and jitteriness and long term sexual SEs), 5HT3 (N/V)

        - OD not an issue
    - **SMs (Serotonin Modulators)** (used to augment sleep in pts w/ depression already on other meds)
        - Types
            - Nefazodone (Serzone) less sexual SEs but never used b/c of liver dz
            - Trazodone (Desyrel) priapism
    - **DNRIs (Dopamine and NEpi Reuptake Inhibitors)** (used in Sleepy/Depression)
        - Types
            - Buproprion (Wellbutruin ± SR/XR, Zyban) no 5HT SEs and no drug interactions like SSRIs, never used in anorexics b/c they have low seizure threshold and buproprion lowers it even more, used for seasonal D, smoking cessation, ADHD, migraine HAs
    - **GABA Cl- Channel Agonists** (opens Cl channels such that more Cl enters into neurons hyperpolarizing them making it more difficult to depolarize thus global CNS inhibition)
        - **Barb** (increases duration of channel staying open, Phenobarbital for seizures vs Pentobarbital for insomnia vs Thiopental for anesthesia, worse SEs compared to benzos in addition there is P450 induction)
        - **Benzo** (increases Hz of channel staying open, tolerance develops to all SEs but fortunately not to anxiolytic effects, distribution into fat is a problem, SEs: "drunk", resp/CV depression, addictive potential, withdrawal Sx, Flumazenil for reversal)
            - Short Acting (t1/2 1hr): midazolam (Versed) = IV anesthesia Q30min
            - Intermediate Acting (t1/2 12hr): lorazepam (Ativan) = IV agitation, alprazolam (Xanax) = PO anxiety Q6hr
            - Long Acting (t1/2 24hr): diazepam (Valium) = PO anxiety Q12hr
            - NB for each dose is about 0.5-10mg the difference is how long they last
        - NB **Buspirone (BuSpar),** anxiolytic that does not act on GABA but effects serotonin levels, unlike Benzos it takes weeks to have effect and can be used long term, no "drunk", no resp/CV depression, no addictive potential, no withdrawal Sx but r/o Serotonin Syndrome and dysphonia**)**

Mood Disorders

- **Depression aka Major Depressive Disorder (MDD)**
    - Def: ≥1 episodes of major depressive episodes characterized by ≥5/9 criteria (where ≥1/ 2 must be depressed or anhedonia) for >2wks that causes dysfxn in life
    - Mech: Locus Ceruleus (NE) and ? (D) and Dorsal/Medial Raphe Nucleus (5-HT) = Happiness
    - Criteria
        - (1) depressed mood
        - (2) anhedonia
        - (3) change in weight/appetite w/ decrease more common than increase
        - (4) sleep disturbance either insomnia more common than polysomnia
        - (5) psychomotor agitation/retardation
        - (6) fatigue
        - (7) excessive guilt or feeling of worthlessness or low self-esteem
        - (8) difficulty concentrating
        - (9) thoughts of death/suicide
        - NB Other S/S: vague somatic complaints similar to FM, poor libido, psuedodementia esp in elderly vs irritable mood in children, excessive rumination about the past, pre-occupation w/ physical health, +FHx, psych comorbidities including eating/anxiety disorders, substance abuse, etc
        - Dx Tests: PHQ-9, Beck Depression Inventory, PRIME-MD, etc
    - DDx/Variants
        - **Dysthymia**
            - Def: chronic (≥2yrs), more continual than episodic, milder form of MDD w/o criteria of suicide/guilt/psychomotor changes
            - NB "double depression" occurs when you have dysthymia punctuated by MDD episodes
            - 20% develop MDD or Bipolar Disorder
        - **Bereavement/AD** w/ similar Sx at MDD but occurs after a loss of a loved one and lasts <1/1-6mos
        - **Chronic** if >2yrs
        - **MDD w/ Psychotic Features** (never had psychotic features w/o depression) vs **Schizoaffective Disorder** (can have psychotic features that exist on own)
        - **MDD w/ Catatonic Features** if ≥2/5: immobility, purposeless motor activity, mutism, bizarre posturing or stereotyped movements or grimacing, echolalia/praxia
        - **MDD w/ Melancholic Features** if anhedonia and ≥3/6: very depressed, mood worse in morning, early morning awakening, psychomotor slowing, weight loss, guilt
        - **MDD w/ Atypical Features** if mood reactivity and ≥2/5: weight gain, hypersomnia, heavy feeling in extremities, rejection sensitivity
        - **MDD w/ Postpartum Onset** if w/in 4wks of parturition
        - **MDD w/ Seasonal Pattern** if onset at same time each year and full remission at other time each year

        - **2/2 Medical Problem or Medicine**
            - Meds: BB
            - CNS: CVA
            - Endo: HypoTH, HyperCa, DM, Addison's
            - Cancer: Lymphoma, Carcinoid
            - Infection: Mono, HIV, Syphilis
    - Tx Strategy (Anti-Depressants)
        - General
            - Neurovegetative Sx (lack of initiative and poor energy) will improve prior to improvement in Depressive Sx (2wks vs 6wks) after starting Tx which sometimes allows pts to follow thru on suicidal ideations
            - Meds (increase 5-HT, NorEpi, D) + Psycotherapy (Interpersonal, Insight Oriented, Cognitive Behavioral)
            - Adjuvants: stimulants, antipsychotics, thyroid hormone
            - NB anti-depressants have no effect (do not elevate mood) in a nl person
            - 70% of depressed pts respond to anti-depressants vs 50% of manic pts respond to mood stabilizers
        - 1st: start SSRI (based on FHx, SE profile, formulary, etc)
        - 2nd: increase dosage
        - 3rd: switch to different SSRI
        - 4th: increase dosage
        - 5th: switch to Effexor/Cymbalta or add Wellbutrin
        - 6th: consider Bipolar Disorder and add Abilify
        - 7th: psych referral for MAOIs (should only be Rx by psychiatrist given SEs but even then rarely use only for refractory depression b/c atypical, comorbid anxiety, etc)
        - 8th: Somatic Therapies for refractory to meds/psychotherapy, high suicide risk, depression w/ psychotic/bipolar/catatonic features, in elderly, in pts attempting to harm themselves, in pts who cannot tolerate meds, pregnant women, Parkinson's
            - **Electroconvulsive Therapy (ETC)** where basically a seizure is induced, SEs: memory loss, headache, delirium
            - **repetitive Transcranial Magnetic Stimulation (rTMS)** where an MRI like device stimulates the cerebral cortex
            - **Vagus Nerve Stimulation (VNS)** where a pacemaker like device is implanted in upper chest with lead attached to vagus nerve

- **Mania aka Bipolar Disorder**
    - Def: ≥1 episodes of only mania or severe-mixed aka both mania and MDD (Type I) or mod-mixed aka both hypomania and MDD (Type II) episodes w/ mania episodes characterized by euphoric/irritable mood w/ ≥3/7 (≥4/7 if irritable) criteria for ≥1wk (may be ≤1wk if pt is hospitalized) that causes dysfxn in life
    - Mech: ?
    - Criteria
        - (1) inflated self-esteem or grandiosity
        - (2) decreased need for sleep
        - (3) more talkative than usual, pressured speech, loud and intrusive
        - (4) flight of ideas or racing of thoughts
        - (5) distractibility
        - (6) increased goal directed activity or psychomotor agitation w/ excessive planning but overall non-productive
        - (7) excessive involvement in pleasurable but reckless activities w/ potential for negative consequences (eg. shopping sprees, sexual promiscuity, etc)
        - NB Other S/S: inability to sleep for days (but unlike in depression the insomnia is not bothersome), grandiose delusions, lack insight, can be assaultive, dysphoria at height of euphoria resulting in suicide, first episode is usually depression in females and mania in males, often a late dx w/ usually 10yrs from onset of Sx to Dx, episodes increase in Hz w/ age, 15% suicide rate, psychiatric comorbidities (eating disorders, substance abuse, etc), strong FHx, presents ~20yo
    - DDx/Variants (similar to MDD)
        - **Cyclothymia**
            - Def: chronic (≥2yrs), more continual than episodic, milder form of Bipolar w/ mild-mixed aka both hypomania and dysthymia
            - No dysfxn in life, does not require hospitalization, no psychotic features
            - Often coexists w/ borderline personality disorder
        - **"Rapid Cycling"** if ≥4 episode per year w/ no Sx for ≥2mo b/t episodes
    - Tx Strategy (Mood Stabilizers)
        - Never give anti-depressants alone b/c can precipitate manic episodes but pts often need anti-depressants therefore start after mania is being effectively Tx
        - Worse prognosis than MDD b/c only 50% respond to meds

        - Combination is the rule, good for also impulse disorder and aggressive behavior
        - **Acute Tx w/ Lithium, Neuroleptics, Benzo if Agitation & Hospitalization**
            - SEs: nephrogenic DI (give amiloride), tremor (give BB), hypoTH (give HRT), acne, leukocytosis, flat T-wave (d/c LI!!!), alopecia, metallic taste
            - Therapeutic (0.7-1.2) vs Toxicity (>1.5) vs Death (>2.0) levels rise when pt is ill, fasting, taking certain meds (ACE-I, NSAIDs, Diuretics, Flagyl, etc)
            - OD Tx: dialysis
        - **Chronic Tx w/ AEDs**: 1° valproic acid (Depakote), 2° carbamazepine (Tegretol), lemotrigine (Lamictal)

Anxiety Disorders

- **General**
    - Definition: fear out of proportion to external stimulus resulting in interference w/ fxn and significant distress
    - DDx: primary psych condition (**↑Nepi, ↓Serotonin, ↓GABA @ Locus Ceruleus**), endogenous (**hyperTH, COPD, RCC, arrhythmias, pheo, carcinoid**), exogenous (**stimulants or withdrawal from downers**)
    - NB b/c high comorbidity assess other psychiatric conditions especially **depression and substance abuse**
    - Tx
        - pharmacotherapy provides relief but cure is unattainable unless you initiate Cognitive Behavioral Therapy (**CBT**) (esp relaxation techniques and cognitive restructuring which helps the pt control thoughts, perception, and reaction to any event)
        - consider **alternative treatments**: reflexology (yoga, acupuncture, massage), herbals (Kava aka Piper methystica), etc
        - institute **lifestyle changes**: decrease caffeine/alcohol/smoking, get adequate sleep, engage in moderate exercise
- **General Anxiety Disorder (GAD)**
    - **chronic** (>6mo) **moderate** anxiety that is present most days and **never** related to a **specific stimulus**
    - DSM-IV 3/6: restlessness, easy fatigability, irritability, difficulty [ ], muscle tension, insomnia (NB other features: motor changes (trembling, aches, soreness, twitching), autonomic hyperactivity (SOB, palpitations, dyspnea, dry mouth, cold/clammy hands, dizziness, light-headed, ab pain, diarrhea , urinary frequency, dysphagia), increased vigilance, increased startling, "I just can't stop worrying")
    - **25%** develop **panic disorder**, develops during early adulthood, 5% life-time prevalence, many times a lifelong problem with rare spontaneous remission
    - **Tx: Any SSRI + Short-Term Benzo → Switch to Any TCA**
- **Panic Disorder (PD)**
    - **episodic** (not chronic) **severe** (not moderate) anxiety that quickly begins to a **specific stimulus**
    - peaking in ~10min and lasting ~30min **followed by >1mo of concern for future attack, concern about implications of the attack, or change in behavior related to the attack**
    - DSM-IV 4/13: palpitations/tachycardia, sweating, trembling/shaking, SOB, choking, CP, nausea/ab distress, dizzy/lightheaded/faint, derealization/depersonalization, fear of losing control/going crazy, fear of dying, paresthesia, chills/hot flashes
    - **40%** have concomitant **agoraphobia** due to apprehension about having attacks in public places and being embarrassed, not having the ability to escape or getting help
    - Tx: Any SSRI + Short-Term Benzo + Gabapentin **→ Switch to Any TCA**
- **Obsessive Compulsive Disorder (OCD)**
    - Obsession (recurrent/persistent thoughts, impulses, or images that are intrusive; pt attempts to ignore, suppress, or neutralize the obsession with other thoughts but fails; pt recognizes that the obsession is a product of his/her mind, situations that provoke symptoms are often avoided) → Anxiety **→** Compulsion (to relieve the anxiety the pt succumbs to the obsession and performs repetitive behaviors which are "aimed" at preventing distress or preventing a specific dreaded event, but they are not connected in a realistic way to what they are attempting to prevent or they are clearly excessive) → Relief
    - **OCD (ego-dystonic: pt sees it is a problem)** vs. **OCPD (ego-systonic: pt feels s/he is fine, preoccupied with perfectionism, order, control)**
    - **5%** of pts w/ OCD have concomitant **Tourette's** Disorder while 50% of pts with Tourette's have concomitant OCD
    - **Tx:** One TCA specifically Clomipramine **→ Switch to Any SSRI →** Switch to Any MAOI **→** ECT/Cingulotomy
- **Phobias**
    - **Agora Phobia**: fear **of just being** in places b/c escape might be difficult or embarrassing or in which help may not be available should unexpected panic symptoms occur (NB often occurs from unTx GAD/PD)
    - **Social Phobia aka Social Anxiety Disorder**: fear **of performing, presenting, speaking, etc** in places b/c fear of doing something embarrassing
    - **Specific Phobia**: fear **of a specific object/situation** (NB 75% of pts with a fear of blood undergo vasovagal fainting, often do not come to clinical attention b/c they do not interfere with functioning, etc)
    - **Tx: Any SSRI + Short Term Benzo + BB**
- **Stress Disorder**
    - **Post Traumatic Stress Disorder (PTSD)**

        - pt **reexperiences** the traumatic event thru nightmares, flashbacks, etc
        - pt **avoids** anything that has to do with the traumatic event
        - pt **lives in an increased arousal state** with 2/5 of the following: hypervigilance, decreased concentration, insomnia, exaggerated startle response, and irritability
        - pt **becomes numb** to other aspects of his/her life with 3/4 of the following: blunted affect, anhedonia, amnesia,and feeling of detachment other features: survivor guilt, personality change, aggression, poor impulse control, dissociative symptoms, perceptual disturbances
    - **Tx: Any SSRI → Switch to Buspirone/Li/BB/Clonidine/AED,** never use benzos b/c of the dramatic incidence of substance abuse

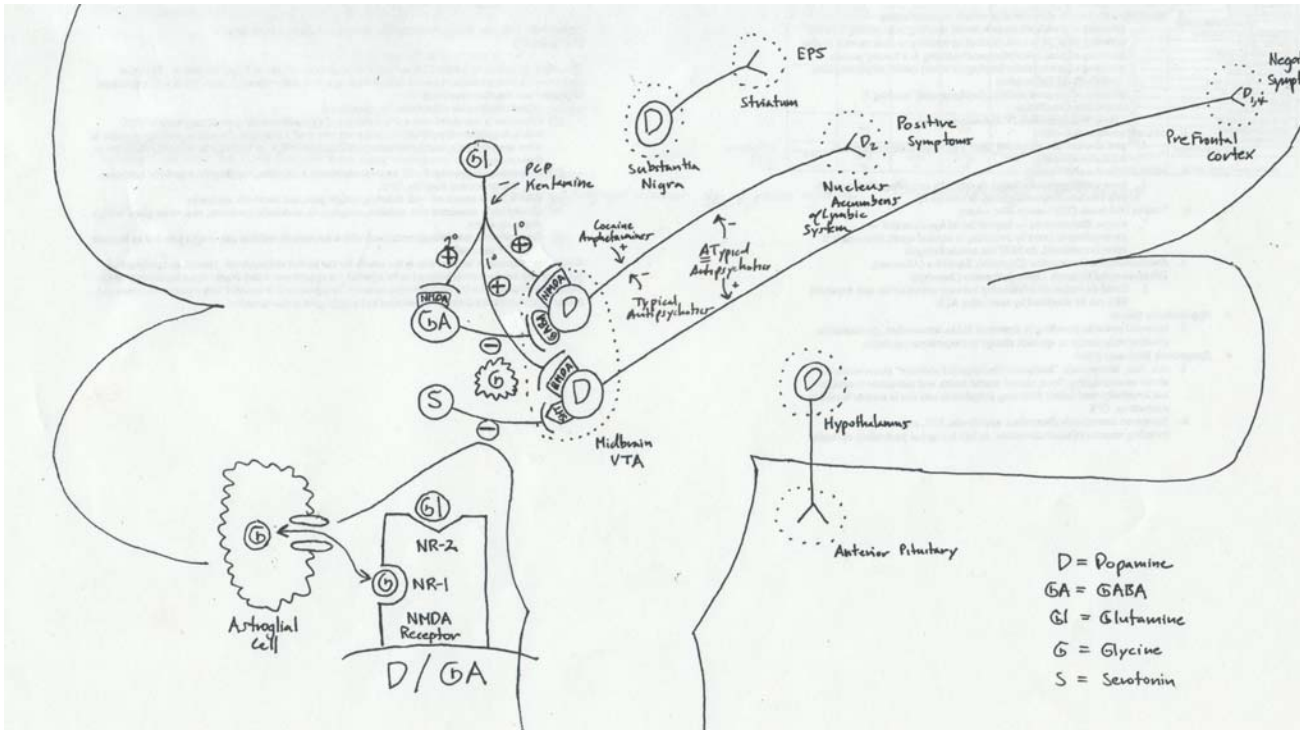
		<1mo	1-3mo	>3mo
Duration	<1mo	ASD (Acute Stress Disorder) severe traumatic event resulting in severe anxiety, good prognosis, remind pts that this is a nl rxn, no Tx	A-PTSD	A-DO-PTSD (Acute Delayed Onset)
	1-6mo		AD (Adjustment Disorder) mild traumatic event resulting in mild anxiety and depression	
	>6mo		C-PTSD	C-DO-PTSD (Chronic Delayed Onset)

Schizophrenia

- Types
    - Schizophrenia
        - Subtypes
            - Paranoid (mainly H/I and D/IOR)
            - Disorganized (mainly disorganized speech/behavior and negative Sx)
            - Catatonic (Immobile, Hypermobile, Peculiar movements, Negativism or Mutism, Echolalia or Echopraxia)
            - Residual (mild Sx)
    - Weird Personality Disorder (lifelong but mild/brief psychotic Sx)
    - Schizoaffective Disorder (criteria for both Schizophrenia AND Depression are met as opposed to Depression/Schizophrenia w/ Pyschotic/Depressive Features)
    - Brief Psychotic Episode (1d-1mo, no –Sx, Sx occur in relation to severe stressor)
    - Schizophreniform Disorder (1mo-6mo)
    - Delusional Disorder (NON bizarre aka plausible delusions for >1mo)
    - Really Bad Depression has Sx similar to -Sx
    - Really Bad Mania has Sx similar to +Sx
- Anatomy
    - Lymbic System – primitive emotions (fear and pleasure) and memory (primordial human qualities), Cocaine and Amphetamines – at low levels stimulate the limbic system resulting in a pleasurable state but at higher doses psychotic features manifest, paranoid subtype
    - Frontal Cortex – initiative, appropriate behavior, concentration, orientation, abstraction, judgment, problem solving (advanced human qualities), Patients with Stroke 2/2 to Anterior Cerebral Artery Embolism – negative like symptoms and cognitive deficits, disorganize subtype
- Epidemiology
    - 1% of population
    - M = F but male earlier onset
    - 10% suicide rate
- S/S (>6mo w/ >1mo of active Sx w/ 2/5 +/- Sx, NB sensorium intact aka A/Ox3
    - Positive Symptoms (H/I, D/IOR, disorganized behavior, thought process problems)
    - Negative Symptoms (blunted affect, alogia, anhedonia, attention deficit)
    - Cognitive Deficits (executive function and memory problems) (atypicals help)
    - Other Psych Issues: Depression, anxiety, OCD
    - Poor Insight/Judgment
- Physiology
    - *Meso-Limbic Pathway* projects dopaminergic neurons from the midbrain ventral tegmental area (VTA) to the nucleus accumbens of the limbic system. The limbic system directs primordial qualities such as fear and pleasure. In fact substances like amphetamine and cocaine stimulate dopamine release from this system inducing pleasure. However, dopamine release reaches a critical point after which increases result in the positive symptoms of psychosis as seen in schizophrenia and cocaine intoxication.
    - *Meso-Cortical Pathway* projects dopaminergic neurons from the midbrain VTA to the frontal cortex. The frontal cortex directs human qualities that begin to distinguish humans from other mammals such as mood and emotion. A drop in dopamine release in the frontal cortex results in three of “Bleuler’s Five A’s” **a**utism,

affect blunting, and **a**mbivalence (negative symptoms of schizophrenia) and disorganization of thought, speech, behavior (cognitive deficits of schizophrenia).

- *Nigro-striatal Pathway* projects dopaminergic neurons from the substantia nigra to the striatum of the basal ganglia. The basal ganglia is the gatekeeper that mediates movement from the conscious cortex to the muscles that carry out the movement. An increase in dopamine release manifest as positive movement abnormalities as seen in Huntington's Disease. Of more relevance to schizophrenia decreases in dopamine release manifests as negative movement abnormalities as seen in Parkinsons's Disease and antipsychotic side effects – so-called "Extra Pyramidal Symptoms" (EPS). The details of basal ganglial circuitry are complex and beyond the level needed to understand antipsychotic therapy. One fact however must be noted – there exists a reciprocal relationship between acetylcholine and dopamine levels in the basal ganglia. This relationship becomes useful when trying to eliminate EPS.
- *Tubulo-Infundibular Pathway* projects dopaminergic neurons from the hypothalamus to the pituitary gland. The pituitary gland is the second portion of the hypothalamic-pituitary-end organ endocrine axis which regulates the release of six hormones. Dopamine released from neurons inhibits lactotropes in the anterior pituitary gland from releasing prolactin. When this tonic inhibition is disturbed, as seen in antipsychotic therapy, prolactin production is released resulting in milk production, decreased libido, etc.



- Tx (goals of antipsychotics are to d ecrease dopamine in the striatum and increase dopamine in the frontal cortex
  - Dopamine Blockers have Anti-Psychotic Effects operating on the Dopamine Hypothesis ("Typicals")
  - Problems: Do not treat negative symptoms AND "dirty" blocking a variety of other receptors.
  - One approach at categorizing typical antipsychotics is based on relative potency from low (L) to medium (M) to high (H). Low potency agents have low D_2 antagonism but high M_2, H_1, and α_1 antagonism. The combination of low D2 and high M2 antagonism results in less EPS side effects but less effectiveness at reducing positive symptoms. In contract high potency agents have high D_2 antagonism but low M_2, H_1, and α_1 antagonsim.
  - As you move down more antipsychotic effects but more EPS
  - Typicals in general have greater EPS and anticholinergic side-effects with only alleviation of positive symptoms while atypicals have greater antihistaminergic and antiserotonergic side-effects with alleviation of positive and negative symptoms.
    - Dopamine Blockade
      - "Extra Pyramidal Symptoms" (EPS) based on reciprocal relationship between acetylcholine and dopamine EPS can be alleviated by decreasing ACh (Anticholinergics: Benztropine (Cogentin), Biperiden (Akineton), Trihexyphenidyl (Artane), Diphenhydramine (Benadryl))
        - Acute Dystonia (occurs after ~4hrs) (common in young males)
          - spasming of neck/back (opisthotonos) resulting in pt arching forward

        - spasming of SCM in neck (torticollis) resulting in neck moving to side
        - spasming of torso (pleurothotonos) resulting in a leaning posture
        - spasming of extraoculular (oculogyric crisis) eyelid (blepharospams)
        - spasming of larygneal muscles (laryngospams) resulting in resp compromise
        - spasming of mouth/tongue/jaw
        - spasming of vocal cords
    - Akinesia (occurs after ~4d)
        - Parkinsonian-like symptoms (treat with anticholinergic agents or dopamine agonists)
    - Akathesia (occurs after ~4wks)
        - inner restlessness/anxiousness manifesting as difficulty remaining still (treat with beta blockers, benzodiazepines, or vitamin E, decrease dose and switch to other agent)
    - Tardive Dyskinesia (TD) (occurs after ~4mo usually >1yr)
        - tongue fasciculations (tongue darting) → lingual/fascial hyperkinesias (lip smacking and chewing) (treat by switching to atypical agent, irreversible if agent is continued, do NOT use anticholinergic, interestingly reducing dopamine blocker actually temporarily worsens TD, best approach is prevention w/ judicious use of neuroleptic and drug holidays)
- Hypothalamic Effects
    - increased prolactin (resulting in decreased libido, amenorrhea, gynecomastia, galactorrhea), change in appetite, change in temperature regulation
- Malignant Neuroleptic Syndrome (~ Malignant Hyperthermia Syndrome)
    - Etiology: rare, fatal, idiosyncratic reaction to neuroleptics, possibly genetic predisposition therefore ask if +FHx reaction to neuroleptics
    - Sx: (1) hyperthermia, (2) muscle rigidity → rhabdomyolysis → ARF, (3) AMS, (4) autonomic instability (hypertension/tachycardia/diaphoresis/dyspnea)
    - Labs: leukocytosis, elevated CPK
    - Tx: neuroleptic cessation and switch to atypical, cooling blankets, hydrate, dantrolene (prevents the release of Ca from SR)
    - Prognosis: 20% mortality

- *Acetylcholine Receptor Blockade* (dry mouth, constipation, blurry vision and dilated pupils, urinary retention, tachycardia, dry skin (if severe can cause "Central Anticholinergic Syndrome" characterized by the addition of delirium))
- *Alpha-1 Adrenergic Receptor Blockade* (orthostatic hypotension)
- *Histamine-1 Receptor Blockade* (sedation, weight gain, metabolic syndrome)
- *Specific Typicals*
    - cardiac toxicity specifically increased QT interval (especially for thioridazine, chlorpromazine, pimozide, and ziprasidone)
    - retinitis pigmentosa resulting in irreversible blindness (seen in patients taking >800mg of thioridazine per day)
    - retrograde ejaculation
    - photosensitivity (seen in low potency typicals)
    - cholestatic jaundice resulting in jaundice, F, N, malaise, pruritus esp in chlorpromazine (seen in low potency typicals)
- Specific Atypicals
    - Clozapine is associated with a 1% incidence of **agranulocytosis** necessitating weekly WBC monitoring for the first six months and every two weeks thereafter. Clozapine is clearly superior to other neuroleptics in treating refractory patients and, in light of side effects, is usually restricted to those patients.
    - Resperidone is associated with **hyperprolactinemia**, sedation, weight gain, metabolic syndrome, and, if doses exceed 6mg/day, EPS.
    - Olanzapine is associated with sedation, weight gain, and metabolic syndrome.
    - Quetiapine is associated with sedation, weight gain, metabolic syndrome, and, when given in high doses, **cataracts**.
    - Ziprasadone is interestingly associated with a decrease in sedation and weight gain but an increase in the **QT interval**.
    - Aripiprazole represents the next step in the search for the perfect antipsychotic. Though its mechanism is still a mystery aripiprazole is believed to be a partial D2 agonist one which blocks hyperfunctioning while stimulating hypofunctioning dopaminergic neurons.

Aripiprazole is associated with nausea and tremor but similar to ziprasadone a decrease in sedation and weight gain is also observed.

- Mechanism Behind Tx
  - Although atypicals diminish negative symptoms better than typicals it is unclear whether this is secondary to a reduction of acute psychosis or a true primary effect. Those who champion a primary mechanism suggest that the predominant 5-$HT_{2\alpha}$ blockage in comparison to the moderate D_2 blockage in the frontal cortex accounts for negative symptom relief. It is speculated that 5-$HT_{2\alpha}$ antagonism promotes dopamine release. In the limbic system 5-$HT_{2\alpha}$ regulation does not exist accounting for positive symptom relief. Interestingly, the only atypical that was studied up to recently has been clozapine. A multisite, double-blind comparison study between typicals and atypicals, the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE), has concluded that atypicals other than clozapine and olanzapine have not demonstrated comparable negative symptom relief. This disappointment in addition with higher costs and other problematic side effects, particularly the metabolic syndrome, has pushed a concerted effort at developing newer generation antipsychotics with different antogonism/agonism profiles. The first of such agents is aripiprazole which is a partial agonist at low dopamine levels and a partial antagonist at high dopamine levels.
  - The simple dopamine hypothesis of the 1980s has now evolved into a complex multiple neurotransmitter system that offers additional sites for pharmacotherapy. The most recent models include a dysfunction of dopaminergic, glutaminergic, seroternergic, and GABAnergic systems (figure 1). In the midbrain the dopaminergic neuron carries both GABA inhibitory and glutamine (NMDA) stimulatory receptors. The GABAnergic neuron behaves as an interneuron between the glutaminergic and dopaminergic neurons such that glutamine (NMDA) stimulatory receptors now exist on both GABAnergic and dopaminergic neurons. Studies have demonstrated that the concentration of NMDA receptors on GABAnergic is greater than those found on dopaminergic neurons resulting in a net inhibitory effect for glutamine on dopamine release in the striatum. The psychotic effects seen in patients on phencyclidine (PCP) or kentamine is attributed to NMDA antagonism. For neurons projecting to the frontal cortex a slightly different circuitry exists. The GABAnergic interneuron is not present. Rather, the glutaminergic neuron acts only on the dopaminergic neuron stimulating dopamine release. A serotonergic neuron innervates the dopaminergic neuron inhibiting dopamine release. This dual innervation explains how clozapine provides negative symptom relief.
  - Since stimulation of the NDMA receptor would provide both positive and negative symptoms relief, currently, research is focusing on stimulating the NMDA receptor. Characterization of the NMDA receptor shows two binding sites: NR-2 which binds the primary agonist glutamine and NR-1, an allosteric site, which binds glycine potentiating glutamanergic action. Given their low molecular weight it is difficult to create glycine and glutamine agonists. Recently, it has been found that glycine concentration around NMDA receptors is closely controlled by glycine transporters on nearby astroglial cells which take up glycine. Given the structure of transporters it is much easier to create a Glycine Transporter Inhibitors (GTIs) than direct receptor agonists. With these inhibitors immediate concentration of glycine around NMDA receptors is increased leading to increased NMDA stimulation of dopamine in frontal cortex and increased GABA inhibition of dopamine in the limbic system and thus improvement of both negative and positive symptoms.
- General Tx Principles
  - Treatment strategy includes pharmacotherapy, psychotherapy, and electro shock therapy (ECT). Pharmacotherapy of schizophrenia and other disorders with psychotic features includes typical and atypical neuroleptics. All neuroleptics are generally equal in efficacy with the exception of clozapine which is best for refractory cases. Thus, when choosing a neuroleptic for a patient the decision can be based on past history of response, family history of response, and side effect profile of a certain agent. When an agent has been initiated one must wait at least two weeks before effect can be appreciated. Hospitalization is indicated if (1) it is an initial presentation so as to determine the exact cause of psychosis, (2) suicidal or homicidal ideations or command hallucinations are present, (3) there is a change in features in a patient with chronic schizophrenia, or (4) the patient is unable to take care of his own needs. During an acute decompensation psychosis can be managed with divided doses (3-4 times per day) of a neuroleptic allowing for treatment while minimizing side effects. Once managed out-patient maintenance therapy can be achieved with once a day dosing at bedtime. Agents are usually administered orally; however, if a patient is noncompliant or has a history of dangerous behavior long acting depot IM forms exist. For acutely agitated patients a fast acting IM form can be used.
  - In patients compliant with pharmacotherapy an average recidivism rate of 40% after first year has been documented. Such a relapse rate combined with the current limitations of antipsychotics at alleviating negative symptoms has led to a renewed interest in psychosocial therapy. Such a global approach, especially in a disease with significant behavior dysfunction, would clearly be of benefit. Pharmacotherapy is “necessary but not sufficient”. Three general models of psychosocial intervention have been used: (1) group therapy, (2) individual therapy, and (3) family therapy.
  - Group therapy aims at improving social skills. Individual therapy aims at strengthening a patient’s coping mechanisms and management of disease. The role of family in schizophrenia has been controversial due to early, now disregarded, claims that family dynamics might be a cause of schizophrenia. Currently, it is recognized that families endure a great deal of stress when caring for chronically mentally ill relatives. Family therapy aimed at addressing their concerns, educating them of the disease itself, and providing them with tools to better take care of their relative will inevitably translate to a better outcome for the patient.
  - Pharmacotherapy has permitted schizophrenics to be treated as outpatients obviating lifelong institutionalization in state mental hospitals. However, the limitations of medicine alone have not allowed

these patients to live successfully in their communities. It is the hope of psychosocial therapy, and programs similar to CHT, that patients will be able to be reinstituted into their social fabric by addressing behavioral issues.

		Potency (L,M,H)	D_2B	D_4B	H_1B	α_1B	M_2B	$5\text{-}HT_{2\alpha}B$
Typicals	Chlorpromazine (Thorazine)	L	+	-	+++	+++	+++	-
	Thioridazine (Mellaril)	L						
	Mesoridazine (Serentil)	L						
	Loxapine (Loxitine)	M	++	-	++	++	++	-
	Thiothixene (Navane)	M						
	Perphenzine (Triafon)	M						
	Trifluoperazine (Stelazine)	M						
	Molindone (Moban)	M						
	Haloperidol (Haldol)	H	+++	-	+	+	+	-
	Fluphenazine (Prolixin)	H						
	Pimozide (Orap)	H						
	Prochlorperazine (Compazine)	H						
Atypicals	Clozapine (Clozaril)	NA	+	+++	+++	+++	+++	++
	Aripiprazole (Abilify)	NA	+	+	+++	+	+	+++
	Resperidone (Risperdal)	NA						
	Olanzapine (Zyprexa)	NA						
	Quetiapine (Seroquel)	NA						
	Ziprasidone (Geodon)	NA						

Pediatric Psych

- **ADHD** (Attention Deficit Hyperactive Disorder)
  - Tx: methylphenidate (Ritalin, Concerta), dextroamphetamine (Adderrall, Dexedrin), atomoxetine (Strattera)
- **Autism** (NB Asperger's is a mild form of Autism where communication is fine)
  - Social Interaction Deficits in non-verbal communicative behaviors, peer relationships, taking the initiative in social interactions, interpersonal reciprocity w/in relationships
  - Communications Deficits as in impairment in language development, impairment in conversational skills, use of repetitive/idiosyncratic language, impairment in the development of imaginative play, echololia, mutism
  - Behavior Deficits such as consuming preoccupation w/ one or a few idiosyncratic interests and restricted breadth of interest in the surrounding environment, excessive need for routine ritualized behaviors, inordinate resistance to change, stereotyped motor movements like hand flapping or rocking, preoccupation w/ parts of objects
- **Other: Tourette's, Enuresis, Oppositional Defiance Disorder vs Conduct Disorder**

Somatoform Disorders

- General: comorbidities (OCD, depression, anxiety), Tx: SSRIs/clomipramine and behavioral therapy
- Unconscious
  - 1° Gain
    - **Somatization Disorder** (CHRONIC disorder where pt constantly worries about MANY SYMPTOMS w/o focusing on disease, symptoms are usually vague often pain, GI, GU, neuro, cannot be explained by organic dz, pts often undergo multiple procedures/treatments, Tx: behavioral therapy so pt can express their emotional needs, focus on life stressors not Sx, but if a Sx emerges initially r/o organic causes b/c they still do occur)
    - **Hypochondriasis** (CHRONIC disorder where pt constantly worries about ONE Dz w/o focusing on Sx, pts often misinterpret a minor Sx like a cough or normal physiologic states like sweating, pts are very scared and often doctor shop, Tx: Group Therapy, reassurance thru regular visits)
    - **Conversion Disorder** (ACUTE disorder where pt develops ONE SYMPTOM following a stressful event lasting <2wks, symptoms are unusual like psuedoseizures, psuedocyesis aka psuedopregnancy, globus sensation, neurologic deficits esp blindness or mutism etc, pt also has "le belle indifference" where the concern for the Sx is minimal, Sx usually do not have normal anatomical distribution, etc,Tx: once conversion disorder is explained to the pt usually the Sx spontaneously resolves if not then consider intensive I&O therapy and hypnosis)
- Conscious
  - 1° Gain (expression of unacceptable feelings as a physical Sx in order to avoid facing the feeling)
    - **Factious Disorder** (pt knows s/he is making up complaint but they don't know why they just want to be sick, pt produces a Sx eg. use insulin, self-inoculation, etc pt often knows a lot about the disease b/c of research, usually healthcare/wealthy/males except if by proxy then usually mother w/ child, no Tx w/ poor prognosis)
  - 2° Gain (use Sx to benefit pt as in attention, money, avoid law, avoid military duty etc)

- **Malingering** (pt intentionally makes up a complaint and they know why)

Eating Disorders NB Eating Disorder NOS when criteria are not met	**Anorexia Nervosa**	**Bulimia Nervosa**	**Binge Eating**
Criteria/Definition	• **Ego Syntonic** (pt does not feel that what s/he is doing is unacceptable and therefore doesn't seek Tx) • **Under (>15%)** Expected/Ideal Body Weight • **Disturbed Body Image** (see themselves as fat and they have an intense fear of gaining weight despite being underweight) • **1°/2° Amenorrhea** • **Restrict** (eat very little) OR **Binge** and then **Compensatory Purging**	• **Ego Dystonic** (pt realizes that what s/he is doing unacceptable and therefore sometimes seeks help) • **Normal** Expected/Ideal Body Weight • **Disturbed Body Image** (convinced that self-worth is excessively influenced by body weight/shape) • NO Amenorrhea • **Binge** (excessive food intake in <2hr period w/ a sense of lack of control >2x/wk for >3mo) unlike in anorexics bulimics never restrict , 3/5: eat rapidly, eat until uncomfortably full, eat when not hungry, eat alone b/c embarrassed about how s/he eats, feels disgusted/depressed/guilty after binging) and then **Compensatory Purging** (induced vomiting, laxative/diuretic use, excessive exercising, fast)	• **Ego Dystonic** • **Over** Expected/Ideal Body Weight • NO Disturbed Body Image • NO Amenorrhea • **Binge** but NO Compensatory Purging
Epidemiology • 15x more common in women	• 0.5% of women • Seen in athletes, modeling, ballet, etc	• 2% of young women	• Very common
Concurrent Psych Dz • Mood Disorders • Substance Abuse	• OC PD • Schizoid PD	• Histrionic PD • Schizoid PD	• None
Complications	• 2/2 Low Nutrition: Pancytopenia, Lanugo, Brittle Hair, Dry Skin, Hypercarotenemia, Decreased Metabolic Rate w/ Decreased HR/BP/Temp, Arrhythmias 2/2 Electrolyte Abnormalities, OP, Peripheral Edema, Refeeding Syndrome, Superior Mesenteric Artery Syndrome, Pancreatitis, LFTs, High Amylase • 2/2 Purging: refer	• 2/2 Purging: Melanosis Coli, Calloused Knuckles, Salivary Gland Swelling, Dental Caries, Pharngitis, Diuretic induced metabolic changes • NB you can check a diuretic screen in the urine	• 2/2 Obesity
Prognosis	• Variable course based on "Rule of Thirds" 1/3 improve, 1/3 stay the same, 1/3 worsen w/ 10% mortality from starvation, suicide or electrolyte disturbance	• 3% mortality (therefore better prognosis)	
DDx • Psych: MDD (pts w/ depression have a poor appetite vs aneroxics have a good appetite), Somatization, Schizophrenia • Metabolic: Addison's • CV: Superior Mesenteric Artery Syndrome • Genetic: Prader-Willi Syndrome, Kluver-Bucy Syndrome			
Tx • Multidisciplinary: PCP, Psych, Nutritionist • NB If pt is medically stable and <20% ideal body weight then do not admit just Tx as out-pt • Therapy: CBT, group therapy, family therapy, supervised weight-gain programs • Rx: weight promoting antidepressants like SSRIs, appetite stimulants, etc			

Personality Disorders

- General: PDs occur when personality traits become inflexible/pervasive/maladaptive to the point where they cause significant social/occupational dysfunction and distress, no insight, traits must be stable and date back to childhood, traits

cannot be caused by stress, another mental disorder, drugs/meds, medical condition, etc, Dx Tests (Rorschack Test, MMPI-2, etc), Tx (psychotherapy)

- Cluster A "The Weird" (higher r/o developing schizophrenia and likely consider to be on the mild end of the schizophrenia spectrum)
  - **Paranoid** (distrust others, suspect other are exploiting them, interpret benign remarks as threatening, bear grudges, hypervigilant, argumentative, hostile, need for control, cannot relax, no sense of humor, etc)
  - **Schizoid** (restricted affect, socially detached, no sexual experiences, no close friends except first degree relatives, often intellectual in math/sciences but overall anhedonia, unlike schizotypal they have no odd thinking behavior, etc)
  - **Schizotypal** (odd beliefs/speech, paranoid, constricted affect, social anxiety, ideas of reference, pts often have charms or believe in clairvoyance, pts are not isolated like schizoids above)
- Cluster B "The Wild"
  - **Antisocial** (unable to control their impulses, unlawful, disregard for rights of others, deceitful, never plan ahead, aggressive, reckless, lack of remorse, diagnosed w/ conduct disorder when young, abusive, exploitive, lying, steal, fight, arrogant but have charm)
  - **Borderline** (splitting perceptions of people esp doctors one day good the other day bad, everything is either black or white, border b/t neurosis and psychosis, unstable interpersonal relationships, unstable self-image, swinging unstable mood, impulsive, suicidal, self-mutilating, h/o childhood abuse/neglect, female)
  - **Histrionic** (excessive emotionality, attention seeking, center of attention, sexually seductive, shifting emotions, uses physical appearance to attract attention, impressionistic speech, dramatic/theatrical expression, bored w/ routine, may resort to suicidal gestures and threats to get attention, animated, verbose, r/o conversion disorder and depression)
  - **Narcistic** (grandiosity, need for admiration, lack of empathy, exaggerated sense of self-importance, fantasies of unlimited success/power/brilliance/beauty, believes s/he is special, takes advantage of others, lacks empathy, arrogant, devalue others, pursue relationships that will benefit them in some way, very sensitive to criticism)
- Cluster C "The Worried"
  - **Avoidant** (anxious, feeling in adequate, hypersensitive, avoid occupational activities w/ significant interpersonal contact due to fear of criticism, unwilling to get involved w/ people unless certain of being liked, preoccupied w/ being criticized, shy, quiet, devastated by minor comments,
  - **Dependent** (need to be cared for, submissive, clinging, fears of separation, difficulty making everyday situations w/o excessive advice and reassurance, needs others to assume responsibility, difficulty initiating projects, feels helpless, will endure great discomfort in order to perpetuate the caretaking relationship)
  - **OCPD** (pre-occupation w/ orderliness/perfectinonism/control at the expense of flexibility/openness/efficiency, interferes w/ task completion, devoted to work/productivity to the exclusion of leisure activity/friendship, reluctant to delegate tasks)

Toxin	Mechanism/Labs/Uses	Complications (Intoxication/Withdrawal)	Tx
al Tylenol (refer) Alcohols (refer)	• check full UTox, Salicylate, Acetaminophen, AG, OG	• watch for withdrawal phase which has the opposite symptoms with the addition of GI Sx (N/V/ab pain) • Withdrawal from downers is life threatening while withdrawal from uppers is not • Toxidromes	• 1-800-222-1222 (Poison Control Center) • Always supportive care w/ attention to AE DON'T, electrolyte/temp/metabolic stabil etc • Gastric Lavage: Tx of choice (best if massiv ingestion, early presentation, very ill, w/o antecedent vomiting, bezoar forming age like iron, salicylates, any sustained release for most poisonings, always have a cuffed in place to prevent aspiration if pt has AM depressed gag reflex, seizures, etc • Small Ionic Compounds eg Li, Arsenic, Alc etc (HD) vs Large Non-Ionic Compounds e everything else (Charcoal) • Whole Bowel Irrigation w/ PEG-ELS
co	• "5 A's": (1) Ask if the pt smokes (2) Advise to quite (3) Assess if pt is willing to set a quite date (4) Assist the pt in creating a quite plan (5) Arrange for f/u (NB counseling for just 3min can result in a 5-10% cessation rate)	• Cancer: resp (oral x9, throat x9, lung x9), GI (esophageal x7, gastric x2, pancreatic x2), GU (renal x5, bladder x7)	• varenicline (Chantix) levels out dopamine that nicotine does not cause spikes in plea centers of brain (NB some studies showin increased nightmares, suicidal thoughts, c psych sx, etc) • bupropion (Zyban/Wellbutrin) doesn't wo that well • nicotine replacement (Nicorette Gum, Co Lozenge, Nicoderm Patch, Nicotrol Nasal Inhaler) • you can buy shredded mint plant which ca used as a substitute for chewing tobacco

			smokeless tobacco
l			
olinergics drugs: atropine, tamines, TCAs, eptics, etc)		• "dry as a bone" (dry skin/MM/eye w/ decreased sweating/salivation/lacrimation) • "red as a beet" (flushing) • "blind as a bat" (mydriasis) • "mad as a hatter" (anxiety/confusion/seizures) • "hot as hades" (hyperthermia) • "fast as lightening" (HTN/tachycardia) • "slow as a snail" (ileus, urinary retention)	• Physostigmine
ergics (organo-nate poisoning)		• Opposite of Anticholinergics	• Atropine
renergics (BB)		• Opposite of Adrenergics	• Glucagon • Gastric Lavage • Ca Gluconate • Atropine/Pacing
ergics (cocaine, tamine, yclidine)	• Mechanism: sympathomimetic by blocking endogenous catecholamine uptake, inhibits serotonin uptake causing euphoric symptoms, increases excitatory glutamate and aspartate NT causing energetic symptoms, blocks neuronal Na channels causing anesthetic symptoms • Hx: from Coca plant, used by natives in Andes, was the main ingredient in original Coca-Cola • Uses: hard rock cocaine aka crack for smoking (most unpure form, cheap ~$15/rock, called crack b/c makes that sound when u heat it up in a crack pipe) that is then purified to cocaine powder for snorting (most pure form, expensive) NB injection rarely done any more • Methamphetamine (Speed, Meth, Crystal Meth, Ice), MDMA (Ecstasy), MDEA (Eve) • Phencyclidine (PCP, Angel Dust)	• same as anticholinergics except no real effect on skin/MM/eyes and GI/Bladder and there is the addition of… • Rhabdo w/ AKI and ACS, NB can produce toxicity in any organ therefore don't dismiss any complaints • Pulm (airway burns, "crack lung", hemorrhagic alveolitis from smoked crack, nasal septum perforation 2/2 necrosis) • PV (decreased pulses, any extremity symptoms are especially worrisome, big concern for dissection) • GI (perforated ulcers, bowel ischemia, obstruction from body packing therefore check KUB, splenic infarct) • Psych (formication aka bugs crawling under skin)	• don't administer BB not even those w/ alp blocking properties like carvedilol or labet rather give phentolamine (straight alpha blocker) • for agitation give benzos • admit to tele • GI decontamination not helpful unless bo packers then charcoal and P • NTG for cardiac ischemia
	• Mech: stimulate opioid receptors	• pinpoint miosis • euphoria and depressed mental status • depressed respiratory effort w/ pulmonary edema • flushed warm skin • Withdrawal: opposite w/ dysphoria, insomnia but w/ violent yawning, Irritable, dilation w/ lacrimation, rhinorrhea, piloerection ("Goind Cold Turkey") w/ sweating	• Intoxication: nalaxone (Narcan) and naltre (ReVia) • Withdrawal: Methadone (for psych Sx) an Clonidine/Buprenorphine/LAAM (for sympathetic Sx)
uate		• CNS/CV/Pulm depression	• Flumazenil
nogens	• Mech: serotonin agonist • Psilocybin (Mushrooms), Lysergic Acid Diethylamide (LSD)	• Hallucinations, synesthesia (change in sensation aka taste color, hear touch, etc) • mydriasis	• Benzo/Haldol
bis	• Tetrahydrocannabinoid (THC) active ingredient in marijuana	• Euphoria but anxious • Increased appetite ("munchies") • Injected conjunctiva w/ nystagmus • Xerostomia • Impaired short term memory formation	•
nts	• Solvents, glue, paint thinners, etc	• euphoria • Nystagmus w/ blurred vision	•
e	• Mech: decouples ETS • Found in: cyanide salts, smoke from burning nitrogen containing polymers like vinyl, silk, etc, esp seen	• S/S: very rapid seizure/death • Dx: metabolic acidosis 2/2 LA, ABG, Cyanide Level	• Charcoal • Oxygen • Amyl Nitrite + Na Nitrite + Na Thiosulfate

	in lab and jeweler workers		
n Monoxide	• Mech: binds more avidly to Hgb/Mgb than oxygen and decouples ETS • Produced during combustion of organic material • Bad b/c clear, odorless, non-irritating	• S/S: flu-like Sx w/ HA, N, dizziness, red skin • Labs: metabolic acidosis, +carboxyhemoglobin (nl: <5% non-smoker vs <15% smoker) NB remember that pulse ox cannot be used to differentiate carboxy from oxy, you have to use ABG	• 100% oxygen by tight fitting mask or ETT consider hyperbaric oxygen
	• Mechanism: inhibit presynaptic uptake, inhibit cardiac Na channels, inhibit ACh, inhibit alpha-1, inhibit H-1, inhibit GABA • Labs: measuring TCA levels has limited utility b/c they don't necessarily correlate w/ organ damage, follow ABG, check LFTs b/c metabolized by liver • the most serious OD w/ 25% mortality	• Similar to Anticholinergics but instead of CV activation there is hypoTN and weird arrhythmias which is the most common cause of death	• NGT then lavage if <2hrs • activated charcoal if no GI complications • maintain BP w/ fluids/pressors • protect airway w/ intubation • bicarb if QRS widening >100msec, vent arrhythmias, hypotension, bicarb increase which promotes protein binding thus low free [drug] and the Na binds the negative charged TCA making it neutral and thus le available for interaction w/ sodium chann try not to use any anti-arrhythmics • benzos for seizures • admit to tele • dialysis has NO use b/c lipophilic thus larg and highly bound to proteins thus difficul remove using dialysis
Hydroxy-Butyrate	• NB often there is coingestion w/ alcohol	• Unconsciousness, vomiting, myoclonic movements during recovery, bradycardia, hypotension	• Don't do much just watch pt
ates	• Lots of medicines contain salicylates including alka-selzer, pepto, etc • Lab: salicylate level (no nomogram exists)	• 0-1g: Analgesia/Antipyresis/Antiplatelet • 1-5g: Anti-Inflammatory w/ Tinnitus • 5-10g: N/V/Ab Pain, Resp Alkalosis (2/2 resp center stimulation), AMS • 10-15g: Metabolic Acidosis (2/2 oxidative phosphorylation uncoupling therefore anaerobic metabolism therefore lactic acidosis), Hyperthermia & Diaphoresis • >15g: dead	• Charcoal • HD • Alkalinize Urine