Classification of autonomic drugs

Stimulate parasympathetic nervous system	Cholinergic parasympathomimetic or muscarinic agonists
Inhibit parasympathetic nervous system	Anticholinergics parasympatholytic or muscarinic blockers
Stimulate sympathetic nervous system	Adrenergic sympathomimetics or adrenergic agonists
Inhibit sympathetic nervous system	Adrenergic antagonists anti-adrenergics or adrenergic blockers

Classes of autonomic drugs

Cholinergics	Stimulate the parasympathetic nervous system rest-and-digest Receptor: Acetylcholine (muscarinic)	1. Direct acting 2.Indirect acting
Anticholinergics	Inhibit the parasympathetic nervous system, which induces fight-or-flight (sympathetic)	
Adrenergic	Stimulate the sympathetic nervous system Result depends on type and location of receptor (α or β)	α1 agonist α2 agonist β1 agonist β2 agonist Catecholamines
Adrenergic antagonist	Inhibit sympathetic nervous system Action depends greatly on type of receptor (α or β)	α1 antagonist 1 antagonist β2 antagonist

Primary neurotransmitters in the CNS

The CNS is responsible for our perception, mood, consciousness, behaviour, and cognition Therefore, drugs influence perception, mood, consciousness, behaviour, and cognition by altering neurotransmitter activity

Serotonin (5HT)	mood

Primary neurotransmitters in the CNS (cont)

GABA	inhibitory
Norepinephrine (NE)	stimulatory
Dopamine (D)	behaviour & movement
Glutamate	stimulatory

Adverse effects of CNS drugs

Benzodiazepine	drowsiness, sedation, memory loss, weakness, disorientation, ataxia, sleep disturbances, hypotension, blurred/double vision, nausea and vomiting
Barbiturates	Rarely prescribed anymore for anxiety or insomnia because of side effects
Hypnotic/sedatives	dizziness, headache, daytime drowsiness, dyspepsia, dry mouth, bitter metallic taste, nausea, anterograde amnesia
Melatonin	Adverse effects and monitoring mostly limited to drowsiness level (caution with endocrine dysfunction) because it's identical to endogenous
TCA's	sedation, dizziness, orthostasis, blurred vision, dry mouth, tachycardia, cognitive impairment, constipation, dry eyes, urinary retention

Adverse effects of CNS drugs (cont)

SSRI's	Transient: headaches, nervousness, insomnia, nausea, diarrhea	
	Long-term: Sexual dysfunction, withdrawal upon discontinuation	
MAOI's	: constipation, dry mouth, headaches, changes in heart rate and blood pressure, insomnia, nausea, loss of appetite	
	Food interactions	foods containing tyramine = Hypertensive Crisis!!!
Mood stabilizers	Dizziness, fatigue, short-term memory loss, increased urination, GI upset, dry mouth, muscular weakness, tremors, excessive loss of sodium can lead to toxicity	
	In the absence of sodium (Na), the cells take in lithium instead	

Adverse effects of CNS drugs (cont)

	lithium toxicity	Transient gastrointestinal symptoms are the earliest side effects to occur Mild degree of fine tremor of the hands may persist throughout therapy Thirst and polyuria may be followed by increased drowsiness, ataxia, tinnitus and blurred vision, indicating early toxicity As intoxication progresses the following manifestations may occur: confusion, increasing disorientation, muscle twitches, hyperreflexia, nystagmus, seizures, diarrhea, vomiting, and eventually coma and death
CNS stimulants	Insomnia, anxiety, restlessness, agitation, significant nausea/vomiting, anorexia (give with food), Cough, dry mouth, Tachycardia, hypertension, arrhythmias --> monitor and watch for signs of cardiovascular disease	
	dose in AM or early afternoon	

Adverse effects of CNS drugs (cont)	
Typical antipsychotics	dizziness, drowsiness, orthostatic hypotension, dry mouth, dry eyes, constipation, blood dyscrasias (abnormal lab tests)
	EPS and NMS occur with typical antipsychotics
Atypical antipsychotics (clozapine)	significant agranulocytosis, seizures, tachycardia, NMS • BUT HAS NO EPS
Atypical antipsychotics (all the rest)	drowsiness, dizziness, dry mouth, hyperglycemia, changes in cholesterol levels, weight gain, EPS
Barbiturates for seizures	Soft tissue irritant – avoid injecting if possible IM – inflammation; IV – tissue necrosis Can cause vitamin deficiencies (D, B12, folate) • Requires adequate supplementation

Adverse effects of CNS drugs (cont)	
Phenytoin	dysrhythmias, headache, nystagmus, confusion, slurred speech, changes urine colour (red/brown), blood dyscrasias, hyperglycemia, gingival hypertrophy, skin reactions, osteoporosis
Valproic Acid	: sedation, GI upset, prolonged bleeding time, visual disturbances, ataxia, vertigo, muscle weakness, hepatotoxicity, pancreatitis, bone marrow suppression
Succinimides	mental and physical impairment, psychosis, behavioural changes, CNS effects, bone marrow suppression
dopamine agonist	reduced impulse control
Opioid Analgesics	sedation, fatigue, euphoria, confusion, constipation, respiratory depression, nausea, vomiting

Adverse effects of CNS drugs (cont)

Opioid Antagonist	minimal toxicity, however the effect of reversing analgesia will cause increased blood pressure, tremors, hyperventilation, nausea/vomiting and drowsiness (i.e. sudden withdrawal symptoms)
NSAIDs	gastric and epigastric discomfort, increased bleeding time, nausea, possible nephrotoxicity, cardiovascular events with long term use
acetam-inophen	possible liver damage (hepatotoxic metabolite), causes less gastric irritation than aspirin, does not affect blood coagulation BUT can interact with warfarin
Gabapentin	Fatigue, weight gain, heartburn, ataxia, dizziness very common
Pregabalin (Lyrica®)	Dizziness, fatigue, peripheral edema, dry mouth
	better tolerated than Gabapentin

Adverse effects of CNS drugs (cont)

Corticost-eroids	infections, hyperglycemia, hypertension, thinning skin, easy bruising, moon face, osteoporosis, HPA-axis suppression
Muscle relaxants	sedation, dry mouth, urinary retention (anticholinergic effects)
Anesth-etics	tingling, mucosal irritation, CNS toxicity, cardiovascular collapse
Duloxetine (Cymba-lta®)	Nausea, dizziness, fatigue all common
Triptans	dizziness, drowsiness, warming & prickling sensation, may experience rebound headache Vasoconstriction =↑ BP
Ergot Alkaloids	leg weakness, muscle pain in extremities, nausea and vomiting

Serotonin Syndrome
↑ risk when >1 drug that increases serotonin in the body
Not always obvious due to promiscuity – triptans, tramadol, etc.
symptoms: Hypertension, tremors, sweating, shivering, confusion, anxiety, restlessness, tachycardia, muscle twitching
Anywhere from 30 mins after dose --> weeks after dose of the 2nd drug

Emotional & Mood Disorders

Depression
Mood Disorders (Bipolar)
Post-traumatic Stress Disorder (PTSD)
Attention Deficit Hyperactivity Disorder (ADHD)
Many more (hundreds)

Medication for Emotional & Mood Disorders

Antidepressants	**1.Tricyclic antidepressants (TCAs)**	Work by inhibiting reuptake of norepinephrine, serotonin, and dopamine, leaves more neurotransmitter within cleft
	-triptyline; -pramine -oxepine	
	2.Selective serotonin inhibiters (SSRIs)	Work by inhibiting reuptake of serotonin only
	Citalopram, escitalopram, fluoxetine, fluvoxamine, paroxetine, sertraline	
	3. Monoamine oxidase inhibitors (MAOIs)	Reserved for people who haven't responded to SSRI or TCA Inhibits monoamine oxidase (MAO) which breaks down norepinephrine leaves more norepinephrine in the synaptic cleft breaks down dopamine, epinephrine, and serotonin leaves more of these neurotransmitters as well causing many side effects and interactions
	Phenelzine, tranylcypromine, moclobemide	
	4. Atypical antidepressants	Inhibiting reuptake of serotonin, norepinephrine and dopamine activity with different affinities Also work on other receptors like histamine

Medication for Emotional & Mood Disorders (cont)

	Bupropion (Wellbutrin®, Zyban®)(NDRI), mirtazapine (Remeron®)(SNRI), venlafaxine (Effexor®)(SNRI), duloxetine (Cymblata®)(SNRI), trazodone (Desryl®)-(SARI)	
Mood stabilizers	Work by altering sodium transport across cell membranes By altering sodium transport, it influences the release, synthesis, and reuptake of multiple neurotransmitters	
	Primarily used for bipolar disorder (manic-depression)	
	Lithium carbonate	
	Anticonvulsants: carbamazepine, divalproex, lamotrigine, valproic acid, gabapentin, topiramate	Anticonvulsants are also used as mood stabilizers because they also alter transport of ions across cell membranes
CNS stimulants	Work by heightening awareness and increasing focus (non-specifically) Primarily used for ADHD in children and adults All cause an increase in attentiveness and heightened awareness by influencing NE and D release somehow	

Medication for Emotional & Mood Disorders (cont)

Methylphenidate (Ritalin®, Concerta®, Biphentin®) Dextroamphetamine (Dexedrine®) Dextroamphetamine and amphetamine (Adderall®) Lisdexamfetamine (Vyvanse®)

Degenerative diseases

Parkinson's disease	Gradual destruction of neurons from substantia nigra → striatum of brain that use dopamine to communicate Movements and impulses essential to performance of movements ↓ number of dopaminergic neurons → ↓ dopamine Symptoms are a characterization of ↓ dopamine
Parkinson's symptoms	Classic features: Tremor, Bradykinesia, Rigidity, Loss of balance Other features; Depression, anxiety, mood change, Memory loss --> dementia, Difficulty concentrating, Change in sense of smell, Change in sleeping patterns, Constipation, light-headedness, sweaty, Difficulty swallowing, chewing, speaking, blinking
Dementia	A term that describes a decline in a variety of functions (e.g. memory, language, motor activities, ability to recognize or identify objects, complex decision-making) which eventually causes a person to have difficulty performing everyday activities

Types of Dementia

Degenerative diseases (cont)

Alzheimer's Disease	amyloid plaques and tangles
Vascular Dementia	reduced blood supply
Frontotemporal Dementia	younger patients, highly genetic, odd behaviours
Lewy Body Dementia	presence of Lewy Bodies, well-formed hallucinations
Parkinson's Disease Dementia	Parkinson's usually diagnosed first – both neurodegenerative

Parkinson's disease management: All pharmacotherapy focuses on ↑ dopamine levels (directly or indirectly)

Classes of medication for Parkinson's

Levodopa	Effective cornerstone of therapy Dopamine cannot cross blood-brain barrier (BBB) The enzyme that creates dopamine (decarboxylase) is everywhere in the body Levodopa → crosses BBB → converted to dopamine via decarboxylase It is a prodrug Levodopa is always paired with either carbidopa or benserazide (decarboxylase inhibitors that DO NOT cross BBB), which does two things: 1) Enhances distribution to brain 2) Minimizes acute side effects Because conversion to dopamine occurring past BBB (mostly)
Dopamine Agonists	stimulate dopamine receptors
MAO-B Inhibitors (MAOIs)	inhibit the enzyme that breaks down dopamine
Amantadine	either releases more dopamine or inhibits re-uptake of dopamine (exact mechanism unknown) *also anti-viral*
COMT Inhibitors	inhibit peripheral conversion of levodopa to dopamine (making levodopa more efficient)

Classes of medication for Parkinson's (cont)

Anticholinergics	block acetylcholine, which restores balance of acetylcholine and dopamine for tremor only

All other medications for Parkinson's (excluding Levodopa) work to either directly or indirectly to↑ dopamine in brain

Classes of medication for Dementia

Treatment of Dementia

1.Choline-sterase Inhibitors *Donepezil, galantamine, rivastigmine*	Prevent breakdown of acetylcholine (Theory: lack of acetylcholine causes plaques & tangles) May show small improvements in measures of cognition and activities of daily living (ADL) (1-3 points on MMSE) May slow progression (by months, not years) If benefit, seen in 3-6 months Only approved for Alzheimer's but prescribed for all types
2.N-methyl-D-aspartate (NMDA) antagonist *Memantine*	Block glutamate (excitatory amino acid) at NMDA receptor (Theory: persistent activation of NMDA contributes to symptoms) No effect on acetylcholine Alone or in combo with cholinesterase inhibitor – directly conflicting evidence re: benefit Indication: Moderate → Severe Alzheimer's Renally excreted (dosage adjustment needed for impairment)

Management of Behavioural & Psychological Symptoms of Dementia (BPSD)

Antipsychotics, benzodiazepines, antidepressants, stimulants and more

Classification of Pain

Duration

A.Acute pain	Intense, Less than 6 months E.g. sprained ankle
B. Chronic pain	Persists for longer than 6 months, Interferes with daily activities, Associated with feelings of hopelessness E.g. permanent nerve injury

Source

A. Nociceptor	Pain Due to injury to tissues Sharp, localized; or Dull, throbbing, aching E.g. paper cut, broken bones
B. Neurop-athic Pain	Due to injury to nerves Burning, shooting, numbing E.g. nerve injury, shingles

Pharmacological management
Requires thorough:
Health history (including allergies)
BPMH – best possible medication history
Includes an assessment of stress, coping mechanisms, potential for dependency
Baseline assessment including character, location, duration and intensity of pain

Migraines

Goal of treatment	To reduce acute pain via 1.Triptans or 2.Ergot alkaloids
	To prevent further migraines from occurring If patient experiences a significant amount of migraines β-blockers, anticonvulsants (topiramate, valproic acid), calcium channel blockers, TCAs, venlafaxine

Classes of drugs for migraines

Migraines (cont)

1.Triptans	Selective serotonin receptor agonist on intracranial blood vessels and sensory nerves on the trigeminal system Causes vasoconstriction and reduces neurogenic inflammation, relieving migraine headache Used for acute cluster headaches or migraines (with or without aura) as early as possible Available as regular oral tabs, oral disintegrating tablets, injections, nasal spray (due to frequent nausea/vomiting) – we want quick onset Expensive (require EDS in Sask) Interaction with any other drug that also ↑ serotonin serotonin syndrome Tolerance can develop – remind patients to use only when necessary and as few doses as needed
2.Ergot alkaloids	Serotonin receptor agonist and interacts with dopamine and adrenergic receptors (α-blocker) Therefore, more adverse effects Dihydroergotamine – given IV, may see repeated administration for 3-7 days to break cycle of repeat migraines DO NOT GIVE WITHIN 24 HOURS OF TRIPTAN Additive vasoconstriction --> coronary vasospasm Mostly used if triptans fail

Migraine Monitoring:
History of migraines, triggers, and previous treatment, focus on prevention
Effectiveness of treatment (assess pain level)
Blood pressure and pulse
Watch for chest pain, palpitations, confusion, tingling in extremities, or sudden change of headache status (Fever? Rash? Stiff neck?)
Headaches are usually a symptom

Nervous system

Branches of peripheral nervous system	1.Somatic nervous system	Voluntary control over skeletal muscles
	2. Autonomic nervous system	Involuntary control over smooth and cardiac muscle and glands Divided into sympathetic and parasympathetic
Autonomic nervous system	1. Sympat-hetic	Activated under stress Fight-or-flight response Primitive response to avoid harm
	2. Parasy-mpathetic	Activated under non-stressful conditions Rest-and-digest response

Nervous system (cont)

Primary neurotransmitters in the periphery

Norepi-nep-hrine (NE)	Binds with adrenergic receptors	
	Alpha (α) receptors (α1 & α2)	α1-adrenergic Receptors In sympathetic target organs except heart α2-adrenergic Receptors At presynaptic adrenergic neuron terminals
	Beta (β) receptors (β1 & β2)	β1-adrenergic Receptors Mostly in heart muscle β2-adrenergic Receptors Mostly in the lungs
Acetyl-choline (Ach)	Binds with cholinergic receptors	
	Muscarinic receptors	Binding to muscarinic receptor varies between stimulatory and inhibitory action, depending on site
	Nicotinic receptors	Skeletal muscle, smooth muscle, glands Not many useful drugs affect nicotinic receptors

Adverse effects of autonomic drugs

Cholinergics	salivation, sweating, abdominal cramping and hypotension
Anticholinergics	dry mouth, constipation, urinary retention, confusion, tachycardia Less mucous production = dry mouth, eyes, nose Pupil dilation, blurred/double vision, increased intraocular pressure Less sweating = ↑ in body temp Urinary retention = ↑ risk of infection CNS = Agitation, inability to concentrate, confusion -> delirium, hallucinations, illogical thinking, incoherent speech
α-adrenergics	Oral - anxiety, restlessness, tremor, hypertension, tachycardia Nasal – burning of mucosa, rebound congestion if used for long periods
adrenergic antagonist β1 - blocking	bradycardia, hypotension, headache, fatigue, dizziness, sleep disturbances, nausea; most are dose-related and appear early in therapy Rebound tachycardia, arrhythmias and infarction if discontinued suddenly

Anxiety and Sleep Disorders

Anxiety Disorders	Generalized anxiety disorder (GAD) ,Phobias, Panic disorders, Obsessive-compulsive disorder (OCD), Post-traumatic stress disorder (PTSD)

Anxiety and Sleep Disorders (cont)

Sleep Disorders	Either an inability to: Fall asleep, Stay asleep, or Both

In both anxiety and sleep disorders, nonpharmacological management is more effective LONG TERM

Medications provide relief but should be used for SHORT TERM if possible in addition to non-pharmacological management

CNS depressants

1.Benzodi-azepines	Intensify GABA (bind to benzodiazepine receptors on a GABA receptor)
2.Barbitu-rates	Enhance GABA (bind to barbiturate receptor on GABA receptor)
3.Hypnoti-cs/Sed-atives	Commonly also use a benzodiazepine receptor to potentiate GABA, but much more specific
	Bind only to GABA1 for sleep Only cause sedation no anxiolytic or anticonvulsant properties
4.Miscell-aneous	Can act on any neurotransmitter any drug that causes sedation can potentially be used to induce or prolong sleep even if it is an adverse effect
	Includes antihistamines such as diphenhydramine (Benadryl®), dimenhydrinate (Gravol®) or hydroxyzine (Atarax®)
CNS depression is a continuum	muscle relaxation>sedation>induce sleep>anesthesi-a>coma>death

Slow down neural activity in the brain, May or may not be specific for certain neurotransmitters

Classes of Medication for Psychosis

Typical antipsychotics	conventional, 1st generation - good at managing positive symptoms, no dependence D > 5HT More side effects (especially EPS) than atypical

Classes of Medication for Psychosis (cont)

A. Phenothiazines **Chlorpromazine**	Blocks post-synaptic dopamine receptors; also blocks histamine and muscarinic receptors Used to manage mania and psychosis, prevention and treatment of nausea and vomiting
other phenothiazines:Fluphenazine, Methotrimeprazine, Perphenazine, Promazine, Trifluoperazine	
B. Non-Phenothiazines **Haloperidol**	Blocks post-synaptic dopamine receptors Used to manage psychotic disorders, Tourette's, manic states; also an antiemetic
other non-phenothiazines: Flupentixol, Loxapine, Pimozide, Thiothixene, Zuclopenthixol	
Atypical anti-psychotics	unconventional, 2nd generation Newer class – now drugs of choice No dependence More specific for serotonin than dopamine receptors, with different affinities Also bind to α-receptors in periphery Less side effects (especially EPS) than typicals/1st Gen
A.Clozapine	Blocks dopamine receptors; also blocks serotonin, muscarinic, and histamine receptors Reserved only for treatment-resistant schizophrenia because of adverse effects does not have EPS

Classes of Medication for Psychosis (cont)

B. Quetiapine (Seroquel®)	Blocks serotonin receptors; also slightly blocks dopamine receptors Used to treat schizophrenia and bipolar disorder; also used in the behavioural and psychological symptoms of dementia (BPSD)
Others atypicals:Olanzapine (Zyprexa®) Risperidone (Risperdal®) Paliperidone (Invega®) Ziprasidone (Zeldox®)	
Miscellaneous	
A.Aripiprazole (Abilify®)	Partial dopamine and serotonin agonist; also serotonin antagonist at other sites Used for schizophrenia, bipolar, and depression (as an add-on) Fewer side effects but not as effective as others Will also see combinations of antidepressants, mood stabilizers, and benzodiazepines

Antipsychotics are not a cure for schizophrenia – but they are effective if continued

Medications are only effective for as long as the client takes the medication – no dependence

They often have multiple undesirable side effects: Agranulocytosis, EPS, weight gain, sedation, dyskinesias, anticholinergic effects

Effectiveness can lead to discontinuation

Seizure disorders

Seizure	a disturbance of electrical activity in the brain that can affect consciousness, motor activity, and sensation Not every seizure consists of convulsions Many types starting with local (one section) or generalized (whole brain)

Seizure disorders (cont)

Convulsions	involuntary, violent spasms of the large skeletal muscles of face, neck, arms, and legs
Epilepsy	a disorder characterized by recurrent seizures Those seizures can be any type You can experience a seizure without having epilepsy
Causes of seizures	Infectious diseases Trauma to head Metabolic disorders like dehydration, hypoglycemia, kidney disease, electrolyte imbalances Vascular diseases causing lack of oxygen Pediatric disorders febrile seizures Tumours
Seizure Threshold	the balance between excitatory and inhibitory forces in the brain which affect how susceptible a person is to seizures Important: many drugs that alter CNS activity can lower the seizure threshold – this leads to many potential drug interactions

Classes of Medication for seizure disorders

Drugs that potentiate GABA

a.Barbiturates	Potentiate GABA (inhibitory) and suppress the firing ability of neurons by stimulating an influx of Cl- CNS depressants Takes several weeks for control May be used as monotherapy
Phenobarbital	Causes least sedation Follows CNS depression spectrum Dependence and withdrawal occur

Classes of Medication for seizure disorders (cont)

b.Benzodiazepines	Intensify GABA by binding to benzodiazepine receptors, which stimulates an influx of Cl- Work very quickly if injected (used in status epilepticus) Usually an adjunct to other drugs because of dependence and tolerance – reason to use short-term only Follow CNS depression spectrum
Diazepam	As an anti-convulsant, used for short-term seizure control, calming and relaxation
c.Miscellaneous	*Primidone* – some classify as a barbiturate *Topiramate* – a combo of mechanisms (blocks Na+ influx, enhances GABA at some receptors - different from benzodiazepines, and more)

Drugs that suppress Na+ influx

Desensitize Na+ channels, which prevents influx of Na+ (different from blocking or antagonizing)
Sodium movement is a main factor that determines whether neuron will undergo an action potential (excitation)
No dependence or tolerance
Not all require lab monitoring
In CNS action potentials Na+ > Ca+

Classes of Medication for seizure disorders (cont)

a.Hydantoins (***phenytoin*** *and fosphenytoin)*	Very common, treats many types of seizures Very narrow therapeutic range – requires monitoring LOTS of drug interactions with anticoagulants, corticosteroids, supplements; impairs oral contraceptives and some antibiotics
b.Miscellaneous (phenytoin-like) *carbamazepine, lamotrigine,* ***valproic acid*** *(& divalproex*)	Still desensitizes sodium channels, which prevents influx of Na+ Used for absence and mixed-type seizures

Drugs that suppress Ca+ influx

a.Succinimides *Ethosuximide and methsuximide*	Block calcium channels, which delays Ca+ influx, which depresses the activity of neurons in the motor cortex Calcium influx is not as dominant as sodium influx In CNS action potentials Na+ > Ca+
b. Gabapentin	– unknown mechanism for anticonvulsant activity Is shaped like GABA (hence the name), but does NOT bind to GABA receptors Binds to calcium channels to reduce calcium influx Used mostly for neuropathic pain and migraines now

We use drugs that can:
a.Stimulate an influx of Cl- ions, which potentiates GABA
b.Delay an influx of Na+
c.Delay an influx of Ca+
In CNS action potentials Na+ > Ca+

Drug Classes for Pain

Analgesics

Drug Classes for Pain (cont)

a.Opioid analgesics	Work in spinal cord and brain (CNS) to alter perception of pain Moderate to severe pain Some used for anesthesia Different levels of potency/efficacy – all are compared to morphine (Gold Standard)
Routes for administration	Oral: Systemic effects all over the body at opioid receptors Parenteral: Localized or systemic – depends how we do it
Morphine	Routes: PO, IV, IM, SC, rectal, epidural, intrathecal Remember – 5mg PO ǂ 5mg IV Duration of action: PO – 4 to 7h IV – 4 to 5h Epidural – 4 to 24h
Opioid dependency	Physical dependence lasts 7 days Psychological dependence can last many months or years Often, patients switch from IV and inhalation forms to oral form called methadone
Methadone	A long lasting opioid that avoids withdrawal symptoms by stimulating receptors, with no euphoria Has a long t½ - most only need to dose once daily (still patient variation)

Drug Classes for Pain (cont)

opioid antagonist *Naloxone and naltrexone*	Competitively binds to and blocks mu and kappa receptors Blocking opioid receptors would only biologically change something in someone taking an opioid Used to reverse opioid effects Can be a diagnostic tool
naloxone	Opioid antagonist used to reverse opioid toxicity (i.e. respiratory depression is the lethal symptom) Higher affinity for opioid receptors, therefore displaces opioid (competitive antagonist) No euphoria, no dependence or tolerance Schedule II (for emergency purposes only) Effects = instant withdrawal symptoms: Pain, hypertension, sweating, anxiety, irritability + (very uncomfortable to patient, but not life-threatening) Not a substitute for ambulatory care, but can keep someone alive longer If opioid agonist is longer acting than naloxone (i.e. methadone), toxicity could return

Drug Classes for Pain (cont)

b.Non opioid analgesics	Work in peripheral tissues to prevent formation of pain impulses Most non-opioids are also effective for fever, inflammation, and analgesia Used for mild or moderate pain associated with inflammation *Acetaminophen vs. NSAIDs* Acetaminophen does not have anti-inflammatory properties Both have anti-pyretic and analgesic effects
Non-steroidal anti-inflammatory drugs *NSAIDs* *Aspirin (ASA), ibuprofen, naproxen (OTC)*	Primary drugs for the treatment of mild to moderate inflammation Inhibit cyclo-oxygenase (COX), a key enzyme in the biosynthesis of prostaglandins Prostaglandins promote inflammation Reducing prostaglandins effectively reduces inflammation NSAIDs can be selective for COX-2 or non-selective ALSO anticoagulant, antipyretic, anti-inflammatory Primary use: for fever, arthritis, mild to moderate musculoskeletal pain, dysmenorrhea Some drug interactions Caution in elderly due to poor kidney function No ASA in children – Reye's Syndrome

Drug Classes for Pain (cont)

Aceta-min-ophen	Reduce fever at level of hypothalamus and dilation of peripheral blood vessels Enables sweating and dissipation of heat Primary use is to relieve mild-moderate pain and reduce fever No anti-inflammatory actions
Miscell-aneous	Focus is the CNS used for neuropathic pain
a.Gaba-pentin	while shaped similarly to GABA, does not bind to GABA receptors; binds to calcium channels and reduces calcium influx
b.Preg-abalin (Lyrica®)	reduces calcium influx at nerve terminals, which may reduce transmission of nerve pain
Cortico-steroids	Cortisol is released by adrenal glands in response to stimuli to help restore body to normal Drugs synthetically made to mimic cortisol They are anti-inflammatory and immuno-suppressive Primary use: for severe inflammation or immuno-suppr-ession

Drug Classes for Pain (cont)

Muscle relaxa-nts *Methocarbamol, cyclobenzaprine, baclofen, hyoscine*	After sustaining an injury, muscle spasms may occur to stabilize the affected body part and prevent further damage - also generate pain Most work in brain to reduce tonic, somatic motor activity in alpha and gamma systems NOT on muscle cells NOT at neuromuscular junction
Anesthetics	A drug that causes anesthesia, reversible loss of sensation Stabilize the neuronal membrane, preventing initiation and conduction of impulses Primary use is surgery, epidurals General: a reversible loss of consciousness Local: a reversible loss of sensation for a limited region of the body while maintaining consci-ousness
Anti-depressants	
TCAs	Primary use is depression, moving towards chronic pain Migraines, nerve pain, fibromyalgia, etc. Neuropathic pain (due to effect on neurotran-smitters)

Drug Classes for Pain (cont)	
SSRI's *Citalopram, fluoxetine, sertraline, paroxetine*	Selective for serotonin, less side effects than TCAs Also treat concurrent depression and anxiety disorders May be effective for chronic fatigue, hot flashes, mostly used off-label for other pathologically similar conditions
Duloxetine (Cymbalta®)	serotonin and norepinephrine reuptake inhibitor Now indicated for pain associated with diabetic peripheral neuropathy, fibromyalgia, chronic low back pain, and osteoarthritis of the knee Also depression and generalized anxiety disorder
Anti-anxiety meds	*Benzodiazepines* Not a direct MOA, more of a co-morbidity of anxiety along with pain Worry about tolerance and dependence with long term use Encourage PRN (as needed) use, other coping mechanisms, counselling

Pain management is subjective and difficult to manage due to consistent change of condition, tolerance, and dependence – and racism
Patient is guide to treatment
Difficult to know when to encourage more or less use of analgesics

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