

Central Nervous System (CNS) Drugs

1. Analgesics

Mechanism of Action (MOA):

- **Opioid Analgesics** (e.g., Morphine, Oxycodone, Fentanyl, Codeine, Hydromorphone): Bind to mu-opioid receptors in the CNS, inhibiting pain transmission and modulating perception.
- **Non-Opioid Analgesics** (e.g., Paracetamol/Acetaminophen): Inhibits cyclooxygenase (COX) in the CNS, reducing prostaglandin synthesis and decreasing pain and fever.
- **NSAIDs (Non-Steroidal Anti-Inflammatory Drugs):**
 - **Non-Selective COX Inhibitors** (e.g., Ibuprofen, Naproxen, Diclofenac, Ketorolac): Inhibit both COX-1 and COX-2 enzymes, reducing prostaglandin synthesis and inflammation.
 - **Selective COX-2 Inhibitors** (e.g., Celecoxib, Etoricoxib): Specifically inhibit COX-2, reducing inflammation with fewer gastrointestinal side effects.
- **Adjuvant Analgesics:**
 - **Anticonvulsants** (e.g., Gabapentin, Pregabalin): Modulate calcium channels, reducing neuropathic pain transmission.
 - **Tricyclic Antidepressants** (e.g., Amitriptyline, Nortriptyline): Block serotonin and norepinephrine reuptake, enhancing pain modulation.
 - **SNRIs** (e.g., Duloxetine, Venlafaxine): Inhibit serotonin and norepinephrine reuptake, useful in chronic pain conditions.

- **NMDA Receptor Antagonists (e.g., Ketamine, Dextromethorphan):** Block NMDA receptors involved in pain sensitization and chronic pain.
- **Topical Agents (e.g., Capsaicin, Lidocaine Patches):** Act locally to reduce pain perception by desensitizing nociceptive neurons.

Important Side Effects (SE):

- **Opioids:** Respiratory depression, constipation, sedation, nausea, vomiting, tolerance, and addiction potential.
- **Paracetamol:** Hepatotoxicity in overdose, particularly at high doses or with alcohol use.
- **Non-Selective NSAIDs:** Gastrointestinal bleeding, peptic ulcer disease, nephrotoxicity, increased cardiovascular risks, fluid retention.
- **Selective COX-2 Inhibitors:** Increased risk of cardiovascular events (myocardial infarction, stroke), but lower GI toxicity.
- **Adjuvant Analgesics:**
 - **Gabapentin/Pregabalin:** Drowsiness, dizziness, peripheral edema, weight gain.
 - **TCAs (Amitriptyline, Nortriptyline):** Anticholinergic effects (dry mouth, urinary retention, constipation), sedation, orthostatic hypotension.
 - **SNRIs (Duloxetine, Venlafaxine):** Increased blood pressure, nausea, insomnia.
 - **Ketamine:** Hallucinations, dissociation, increased blood pressure.
 - **Capsaicin:** Local burning sensation.
 - **Lidocaine Patches:** Local skin irritation.

Key Notes:

- **Opioid overdose** is treated with **Naloxone**, a competitive opioid antagonist.
 - **NSAIDs should be avoided in renal impairment and cardiovascular disease** due to increased risks.
 - **Paracetamol toxicity is treated with N-acetylcysteine (NAC)** as an antidote.
 - **COX-2 inhibitors have fewer GI side effects** but should be used cautiously in patients with cardiovascular risk.
 - **Adjuvant analgesics are useful for neuropathic pain and chronic pain conditions.**
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2. Antidepressants

Mechanism of Action (MOA):

- **Selective Serotonin Reuptake Inhibitors (SSRIs)** (e.g., Fluoxetine, Sertraline, Escitalopram): Inhibit serotonin reuptake, increasing serotonin levels in the synapse.
- **Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs)** (e.g., Venlafaxine, Duloxetine): Inhibit both serotonin and norepinephrine reuptake.
- **Tricyclic Antidepressants (TCAs)** (e.g., Amitriptyline, Nortriptyline): Block serotonin and norepinephrine reuptake but have significant anticholinergic effects.
- **Monoamine Oxidase Inhibitors (MAOIs)** (e.g., Phenelzine, Tranylcypromine): Inhibit monoamine oxidase enzymes, increasing levels of serotonin, norepinephrine, and dopamine.

Important Side Effects (SE):

- **SSRIs:** Sexual dysfunction, GI upset, insomnia, serotonin syndrome.
- **SNRIs:** Hypertension, increased heart rate, GI upset.
- **TCAs:** Sedation, orthostatic hypotension, anticholinergic effects, cardiotoxicity in overdose.
- **MAOIs:** Hypertensive crisis (when taken with tyramine-containing foods), serotonin syndrome.

Key Notes:

- SSRIs are first-line for depression and anxiety disorders.
 - MAOIs require dietary restrictions due to interactions with tyramine.
 - TCAs are used for neuropathic pain but have a high risk of overdose toxicity.
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3. Antipsychotics

Mechanism of Action (MOA):

- **Typical Antipsychotics (First-Generation)** (e.g., Haloperidol, Chlorpromazine): Block dopamine (D2) receptors in the mesolimbic pathway.
- **Atypical Antipsychotics (Second-Generation)** (e.g., Risperidone, Quetiapine, Clozapine): Block dopamine (D2) and serotonin (5-HT_{2A}) receptors, reducing both positive and negative symptoms of schizophrenia.

Important Side Effects (SE):

- **Typical Antipsychotics:** Extrapyrarnidal symptoms (EPS), tardive dyskinesia, neuroleptic malignant syndrome (NMS), sedation.
- **Atypical Antipsychotics:** Weight gain, metabolic syndrome, QT prolongation, agranulocytosis (Clozapine).

Key Notes:

- Clozapine requires regular monitoring of white blood cell count due to the risk of agranulocytosis.
 - Typical antipsychotics are more likely to cause EPS and tardive dyskinesia.
 - Atypical antipsychotics are preferred for long-term treatment due to fewer motor side effects.
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