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Drugs Acting on the Central Nervous System

Pharmacology

Analgesics, anaesthetics, sedatives, hypnotics, antiepileptics, antipsychotics, antidepressants, anxiolytics and antiparkinsonian drugs.

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CHAPTER 07 / PHARMACOLOGY 07. Drugs Acting on the Central Nervous System

1. Introduction

CNS medicines modify neuronal signalling, neurotransmitter concentrations, ion channels or neural circuit activity. They are used to relieve pain, produce anaesthesia, control seizures and treat psychiatric and neurodegenerative conditions. Many affect alertness, respiration and coordination; monitoring and safe deprescribing matter.

2. Analgesics

Class	Examples / mechanism	Uses and major precautions
Non-opioid	Paracetamol; central analgesic and antipyretic action	Pain and fever; overdose can cause severe hepatic injury. Check combination products.
NSAIDs	Ibuprofen, diclofenac; cyclooxygenase inhibition	Inflammatory pain; GI bleeding, renal injury and cardiovascular risks.
Opioids	Morphine, fentanyl; mainly mu-receptor agonism	Selected moderate-severe pain and anaesthesia; respiratory depression, sedation, constipation, dependence.
Neuropathic pain agents	Gabapentin, pregabalin, duloxetine	Selected neuropathic pain; dizziness, sedation or drug-specific adverse effects.

Opioids require assessment of pain, sedation and respiratory status; additive CNS depressants increase risk. Naloxone is an opioid antagonist used for overdose with respiratory compromise; ventilation support and observation remain important because re-sedation can occur.

3. Anaesthetics, sedatives and hypnotics

General anaesthetics produce a controlled state of unconsciousness and other components of anaesthesia. Local anaesthetics block voltage-gated sodium channels and prevent impulse conduction in a defined area. See Chapter 24 for detailed classification.

Group	Examples	Mechanism / risks
Benzodiazepines	Diazepam, lorazepam, midazolam	Enhance GABA-A signalling; anxiolysis, sedation, anticonvulsant action; falls, respiratory suppression with other depressants, withdrawal.

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Z-drugs	Zolpidem	GABA-A receptor modulation; insomnia; next-day impairment, complex sleep behaviours.
Barbiturates	Phenobarbital	GABA-related inhibition; selected seizure treatment; sedation, respiratory depression, interactions.
Melatonin-related agents	Melatonin	Circadian rhythm effects; product and indication dependent.

Do not abruptly stop regular benzodiazepine or barbiturate therapy without a planned withdrawal regimen. Flumazenil may reverse benzodiazepine effects but can precipitate seizures in selected overdose or dependent patients; specialist use only.

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4. Antiepileptic / antiseizure drugs

Drug	Main action / use	Important adverse effects and nursing points
Phenytoin	Sodium-channel modulation; selected focal seizures and emergency protocols	Nystagmus, ataxia, gingival overgrowth, rash; IV formulation and rate precautions, interactions.
Carbamazepine	Sodium-channel modulation; focal seizures, trigeminal neuralgia	Hyponatraemia, rash, blood dyscrasias; monitor CBC, sodium, interactions.
Sodium valproate	Multiple mechanisms including GABA-related effects	Hepatotoxicity, pancreatitis, thrombocytopenia, major fetal risks; strict pregnancy-related precautions.
Levetiracetam	SV2A modulation; multiple seizure types	Somnolence, behavioural or mood changes; renal dose review.
Lamotrigine	Sodium-channel and glutamate-related effects	Serious rash; slow prescribed titration and interaction review.
Ethosuximide	T-type calcium-channel modulation	Absence seizures; GI effects, fatigue, rare blood disorders.

Benzodiazepines are commonly used first in emergency treatment of prolonged convulsive seizures. Protect the airway, time the seizure, avoid putting objects in the mouth and do not restrain convulsions. Persistent seizures require urgent protocol-based treatment. Monitor adherence and avoid abrupt stopping of antiseizure drugs.

5. Antipsychotics

Class	Examples	Major considerations
First-generation	Haloperidol, chlorpromazine; D2 antagonism	Extrapyramidal symptoms, prolactin elevation, QT effects, sedation or hypotension.
Second-generation	Risperidone, olanzapine, quetiapine, clozapine; variable dopamine/serotonin actions	Metabolic effects, sedation, orthostasis; drug-specific movement and cardiac risks.

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Partial dopamine agonist	Aripiprazole	Akathisia, nausea, impulse-control problems in some patients.

Acute dystonia involves sustained involuntary muscle contractions; akathisia is motor restlessness; parkinsonism includes rigidity and bradykinesia; tardive dyskinesia may involve persistent involuntary movements. Neuroleptic malignant syndrome (fever, rigidity, altered mental state, autonomic instability) requires emergency escalation. Clozapine requires blood monitoring for severe neutropenia and vigilance for myocarditis, seizures and severe constipation.

6. Antidepressants, anxiolytics and mood stabilizers

Class	Examples	Nursing considerations
SSRIs	Sertraline, fluoxetine, escitalopram	GI symptoms, sexual dysfunction, hyponatraemia, bleeding risk, serotonin toxicity.
SNRIs	Venlafaxine, duloxetine	Similar serotonergic effects; monitor BP and drug-specific risks.
TCAs	Amitriptyline	Anticholinergic effects, orthostasis, dangerous overdose and conduction effects.
MAO inhibitors	Selected specialist agents	Major food and drug interactions; require appropriate washout.
Other antidepressants	Mirtazapine, bupropion	Drug-specific sedation/weight effects or seizure risk.
Non-benzodiazepine anxiolytic	Buspirone	Delayed benefit; dizziness; not a rapid rescue sedative.
Mood stabilizers	Lithium, valproate, selected antipsychotics	Lithium has narrow therapeutic window; monitor levels, renal and thyroid function.

Antidepressant benefits may develop over weeks. Monitor clinical worsening, suicidal thoughts, activation or manic symptoms, especially after initiation or dose changes. Serotonin toxicity can cause agitation, clonus, hyperreflexia, sweating and fever. Lithium toxicity may cause vomiting, tremor, ataxia and confusion, worsened by dehydration or interactions such as NSAIDs, ACE inhibitors and some diuretics.

7. Antiparkinsonian drugs

Levodopa with carbidopa replaces dopamine precursor while reducing peripheral conversion. Dopamine agonists (pramipexole), MAO-B inhibitors (rasagiline), COMT inhibitors (entacapone) and amantadine have selected roles. Antimuscarinic agents may be used in limited circumstances. Assess dyskinesia, hallucinations, orthostasis, sleep attacks and impulse-control symptoms. Parkinson medicines are time-critical; delayed or abrupt withdrawal can cause severe deterioration. Review interactions with dopamine-blocking antiemetics and antipsychotics.

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8. Nursing responsibilities

Assess mental status, pain, seizure pattern, respiratory status, fall risk and baseline metabolic parameters where indicated. Verify interactions, monitor drug-specific laboratory tests, maintain seizure safety and counsel about driving, alcohol and adherence. Report sudden mental status changes or severe reactions promptly.

9. Examination questions

Long answer questions

1. 1. Classify CNS drugs and discuss analgesics and antiseizure drugs.
2. 2. Explain antipsychotic and antidepressant drugs with nursing care.

Short answer questions

- Benzodiazepines; lithium toxicity; extrapyramidal symptoms; levodopa/carbidopa; opioid monitoring; serotonin toxicity.

Very short answer questions

- Opioid antagonist? - Naloxone.
- Drug commonly associated with gingival overgrowth? - Phenytoin.
- Main precursor used in Parkinson disease? - Levodopa.

10. Quick revision

CNS depressants may impair breathing. Seizure medicines must not be stopped abruptly. Monitor antipsychotic movement and metabolic effects; lithium requires level monitoring. Levodopa timing is clinically important.

Conclusion

CNS pharmacotherapy requires individualized assessment of consciousness, mobility, breathing, behaviour and therapeutic response, with early recognition of severe adverse effects.

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Practice questions

Choose one best answer. The answer key and explanations follow the questions.

1. Morphine and fentanyl produce their main opioid effects through which mechanism?

A. Mu-receptor agonism
B. Cyclooxygenase inhibition
C. SV2A modulation
D. T-type calcium-channel modulation

2. Why is continued observation needed after naloxone reverses opioid-associated respiratory compromise?

A. All absorbed opioids are immediately excreted
B. Re-sedation can occur

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C. Naloxone always produces permanent opioid blockade

D. Respiratory status becomes irrelevant after one dose

3. Which adverse-effect pattern is associated with phenytoin?

A. Dry mouth, urinary retention and rebound congestion

B. Hypophosphataemia and iron-related dark stool

C. Nystagmus, ataxia and gingival overgrowth

D. Persistent diarrhoea and oral candidiasis

4. After an opioid dose, a patient becomes difficult to rouse with slow shallow breathing. What should the nurse prioritise?

A. Urgent airway and breathing support, escalation and the prescribed opioid-reversal protocol

B. Reassess pain before assessing breathing because analgesia is the main goal

C. Treat the reduced alertness as normal sleep and review at the next medicine round

D. Administer another prescribed sedative before checking respiratory status

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Answer key and explanations

1. A - Mu-receptor agonism

Morphine and fentanyl act mainly as mu-opioid receptor agonists.

2. B - Re-sedation can occur

Naloxone reversal does not remove the need for ventilation support and observation.

3. C - Nystagmus, ataxia and gingival overgrowth

Phenytoin can cause nystagmus, ataxia, gingival overgrowth and rash.

4. A - Urgent airway and breathing support, escalation and the prescribed opioid-reversal protocol

Reduced consciousness and slow breathing can indicate opioid toxicity. Ventilation and airway support are immediate priorities alongside escalation and protocol-directed reversal.

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