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Anti-inflammatory and Musculoskeletal Drugs

Pharmacology

NSAIDs, corticosteroids, antigout drugs, muscle relaxants, drugs used in rheumatoid arthritis and medicines used for musculoskeletal disorders.

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CHAPTER 16 / PHARMACOLOGY 16. Anti-inflammatory and Musculoskeletal Drugs

1. Introduction

Inflammation is a protective response to tissue injury, infection or immune activation. It can cause redness, heat, swelling, pain and loss of function. Anti-inflammatory medicines reduce components of this response. Musculoskeletal pharmacotherapy also includes drugs that modify immune disease, lower urate, reduce muscle spasm or improve bone health.

2. Non-steroidal anti-inflammatory drugs (NSAIDs)

NSAIDs inhibit cyclooxygenase (COX) enzymes, reducing prostaglandin synthesis. COX-1 contributes to gastric mucosal protection and platelet function; COX-2 is involved in inflammation and other physiological functions. Inhibition can provide analgesic, antipyretic and anti-inflammatory effects but also cause adverse reactions.

Group	Examples	Major precautions
Nonselective NSAIDs	Ibuprofen, naproxen, diclofenac, indomethacin	GI ulceration/bleeding, renal injury, fluid retention and cardiovascular risk.
Preferential/selective COX-2 inhibition	Celecoxib, etoricoxib	May reduce some GI risks but does not eliminate them; renal and cardiovascular risks persist.
Salicylate	Aspirin	Antiplatelet effect at low doses; bleeding, bronchospasm in susceptible patients, salicylate toxicity.
Topical NSAIDs	Diclofenac gel	Local skin irritation; systemic absorption still possible.

Avoid duplicate NSAIDs. Assess GI bleeding history, renal function, cardiovascular disease, anticoagulant or steroid use, asthma sensitivity and pregnancy before use. NSAIDs are generally avoided in later pregnancy and require particular caution from 20 weeks onward. Aspirin is not routinely used for fever in children with viral illness because of Reye syndrome risk. Paracetamol is an analgesic and antipyretic, not a conventional anti-inflammatory NSAID.

3. Corticosteroids

Prednisolone, dexamethasone, hydrocortisone and methylprednisolone act through intracellular glucocorticoid receptors, modifying gene expression and inflammatory responses. They are used for selected inflammatory and autoimmune disorders through oral, IV, local or intra-articular routes when indicated. Risks include hyperglycaemia, infection, osteoporosis, hypertension, mood changes, muscle weakness and adrenal suppression. Monitor glucose, BP, infection and bone health when relevant. Prolonged systemic courses must

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not be stopped abruptly without appropriate review.

4. Antigout drugs

Gout develops through deposition of monosodium urate crystals, producing inflammatory arthritis. Acute flare treatment and long-term urate reduction have different goals.

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Drug / group	Mechanism and indication	Nursing concerns
Colchicine	Alters microtubule-dependent neutrophil activity; acute flare or prophylaxis in selected cases	Diarrhoea, marrow/neuromuscular toxicity; severe interactions with strong CYP3A4/P-gp inhibitors, renal/hepatic review.
NSAIDs / glucocorticoids	Reduce inflammation during selected flares	Screen comorbidities and contraindications.
Allopurinol	Xanthine oxidase inhibitor; lowers urate production	Rash, severe hypersensitivity; renal assessment, prescribed titration.
Febuxostat	Xanthine oxidase inhibitor	Liver monitoring; cardiovascular risk review.
Probenecid	Increases urinary urate excretion	Kidney stones, interactions; renal function matters.

Urate-lowering treatment is generally long term, with clinical and serum urate monitoring. It is not an immediate analgesic. A flare can occur during initiation; use prescribed prophylaxis when indicated. Fever, severe joint pain and systemic illness may indicate septic arthritis and need urgent evaluation.

5. Rheumatoid arthritis and disease-modifying antirheumatic drugs (DMARDs)

Group	Representative drug	Important monitoring
Conventional synthetic DMARD	Methotrexate	CBC, liver, renal tests; usually WEEKLY for inflammatory arthritis, not daily; pregnancy and interaction precautions.
Other conventional DMARDs	Hydroxychloroquine, sulfasalazine, leflunomide	Retinal screening for hydroxychloroquine; CBC/liver tests for several agents; reproductive precautions.
Biological DMARDs	Adalimumab, etanercept, tocilizumab, rituximab	Infection risk; appropriate TB/hepatitis screening and infusion/injection monitoring.
Targeted synthetic DMARDs	Tofacitinib and other JAK inhibitors	Infection, thrombosis, cardiovascular and malignancy risks; specialist risk assessment.

DMARDs aim to modify disease activity and reduce structural damage, unlike simple analgesics. Clinical benefit may take weeks or months. Methotrexate dosing errors can be fatal: verify indication and designated weekly day, and clarify daily orders for rheumatoid arthritis. Folic acid may be prescribed to reduce selected adverse effects.

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6. Muscle relaxants and other musculoskeletal agents

Baclofen is a GABA-B agonist used for spasticity; sedation, weakness and withdrawal risk are important, especially with intrathecal therapy. Tizanidine is an alpha-2 agonist that reduces spasticity; monitor sedation, hypotension and interactions. Cyclobenzaprine and similar agents may have selected short-term roles in acute muscle spasm, with sedation and anticholinergic effects. Neuromuscular blockers used during anaesthesia are different: they cause paralysis without analgesia or unconsciousness (Chapter 24). For osteoporosis, calcium/vitamin D when indicated, bisphosphonates, denosumab and selected anabolic agents may be prescribed (Chapter 12).

7. Nursing responsibilities

Assess pain and function, inflammation, GI symptoms, renal/hepatic status, infection and medication history. Monitor laboratory tests for DMARDs; teach weekly dosing, report rash or fever, and reinforce vaccination and infection precautions. Encourage prescribed exercise, physiotherapy, joint protection and adherence. Do not automatically combine analgesics without checking ingredients and risks.

8. Examination questions

Long answer questions

1. 1. Classify NSAIDs and explain mechanism, adverse effects and nursing precautions.
2. 2. Discuss antigout drugs and DMARDs, with a comparison of acute and chronic gout treatment.

Short answer questions

- Colchicine; allopurinol; methotrexate safety; corticosteroids; baclofen; COX inhibitors.

Very short answer questions

- Which enzyme is inhibited by allopurinol? - Xanthine oxidase.
- Methotrexate for rheumatoid arthritis is usually given how often? - Weekly per prescription.
- Which class inhibits cyclooxygenase? - NSAIDs.

9. Quick revision

NSAIDs reduce prostaglandins but may harm the GI tract, kidney and cardiovascular system. Colchicine controls gout inflammation; allopurinol lowers urate. DMARDs modify disease rather than merely relieving pain. Distinguish skeletal muscle antispasticity drugs from anaesthetic neuromuscular blockers.

Conclusion

Musculoskeletal pharmacology combines symptom control and disease modification, supported by organ-function monitoring, safe dose schedules and rehabilitation.

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Practice questions

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

1. What is the principal anti-inflammatory mechanism of NSAIDs?

A. Mu-opioid receptor activation

B. Direct replacement of cortisol

C. Xanthine oxidase inhibition

D. Cyclooxygenase inhibition reduces prostaglandin synthesis

2. Through which type of receptor do glucocorticoids modify inflammatory gene expression?

A. Intracellular glucocorticoid receptors

B. Beta-2 adrenergic receptors

C. Muscarinic M2 receptors

D. Platelet ADP receptors

3. How does allopurinol lower urate?

A. Inhibits COX to reduce inflammation during a flare

B. Inhibits xanthine oxidase to reduce urate production

C. Increases urinary urate excretion

D. Alters microtubule-dependent neutrophil activity

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Answer key and explanations

1. D - Cyclooxygenase inhibition reduces prostaglandin synthesis

NSAIDs inhibit COX enzymes, reducing prostaglandin production.

2. A - Intracellular glucocorticoid receptors

Corticosteroids act through intracellular glucocorticoid receptors to modify gene expression.

3. B - Inhibits xanthine oxidase to reduce urate production

Allopurinol reduces urate production through xanthine oxidase inhibition.

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