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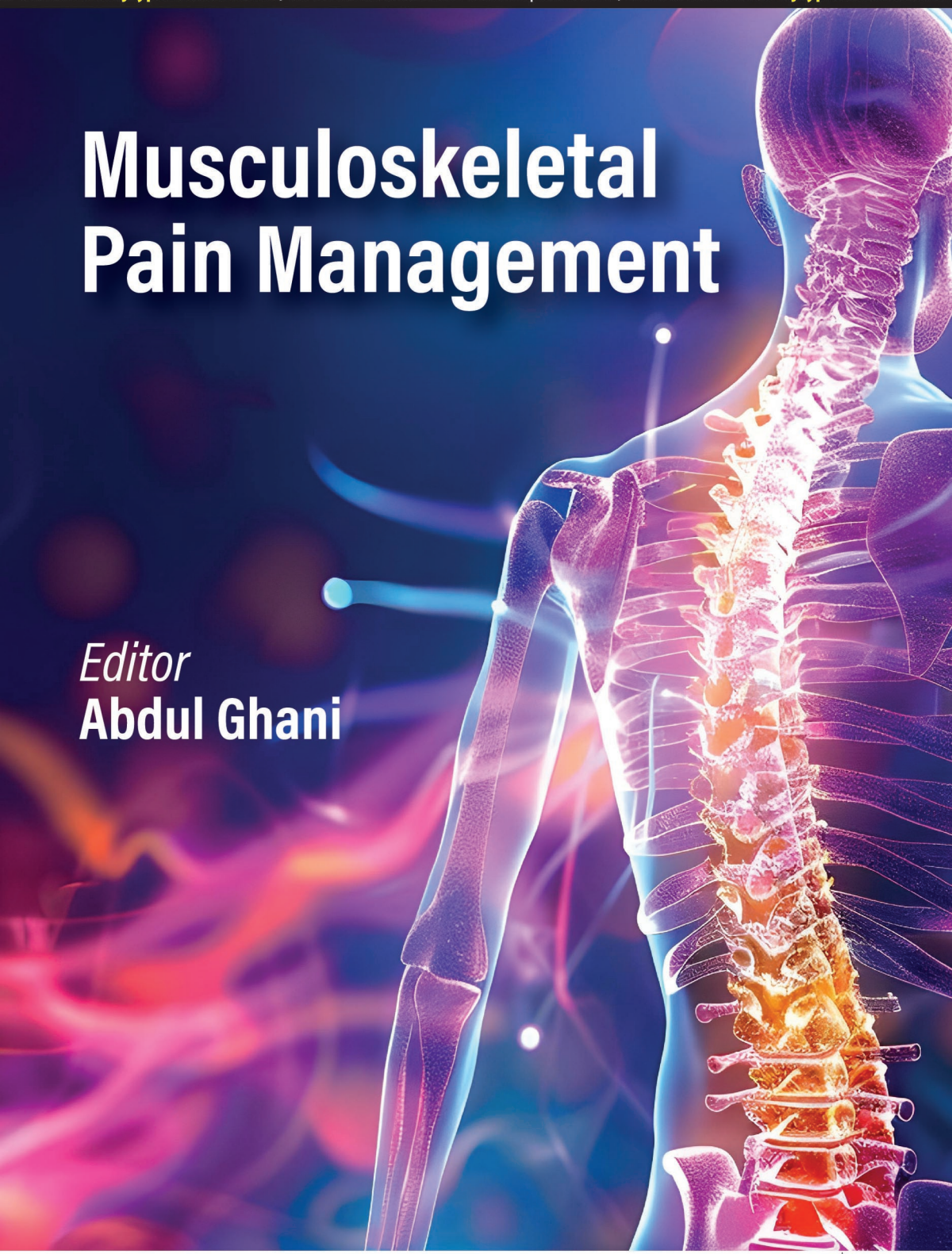
# Musculoskeletal Pain Management



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CHAPTER 9

# Nonsteroidal Anti-inflammatory Drug and Acetaminophen

**Arti Mahajan**

## INTRODUCTION

Nonsteroidal anti-inflammatory drugs (NSAIDs) are a cornerstone in the management of musculoskeletal pain, commonly used for their analgesic, anti-inflammatory, and antipyretic properties. This chapter explores the role of NSAIDs in treating conditions such as osteoarthritis, rheumatoid arthritis, and acute injuries, highlighting their mechanisms of action, efficacy, and safety profile. By inhibiting cyclooxygenase enzymes (COX-1 and COX-2), NSAIDs reduce the production of prostaglandins, which are mediators of pain and inflammation. Understanding the benefits, limitations, and appropriate use of NSAIDs is crucial for optimizing pain management strategies while minimizing potential risks.

## PHARMACOLOGICAL THERAPY

Commonly used drugs to treat mild-to-moderate pain are:

- Acetaminophen (paracetamol)
- Nonsteroidal anti-inflammatory drugs
  - Nonspecific
  - Cyclooxygenase (COX)-2 inhibitors

### Acetaminophen (Paracetamol)

Acetaminophen is a centrally acting nonopioid analgesic. It is the first-line treatment for mild-to-moderate pain because of its relative effectiveness in many pain conditions, high tolerability, and minimum side effects. It has an analgesic and antipyretic activity similar to NSAIDs, but it does not possess significant anti-inflammatory activity. Its molecules do not present the peripheral action on prostaglandin, so it is better tolerated than NSAIDs, especially at the gastrointestinal level, and does not include platelet activity.

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Paracetamol should be considered in combination with NSAIDs in the management of musculoskeletal pain (MSP) and alone in patients in whom NSAIDs are contraindicated.

- *Route*: PO/IV
- *Dose*: 10–15 mg/kg (average 1 g)
- *Duration*: 6–8 hours

Monitoring the patients who receive acetaminophen regularly and beyond the maximum dosage of 3–4 g/day (in adults) is required.

Paracetamol (also known as acetaminophen) is generally considered safe when used as directed, but it can interact with other medications, leading to potential adverse effects. Some notable drug interactions are as follows:

- *Alcohol*:
  - *Interaction*: Increased risk of liver damage
  - *Mechanism*: Both alcohol and paracetamol are metabolized by the liver, and concurrent use can exacerbate liver toxicity.
- *Warfarin and other anticoagulants*:
  - *Interaction*: Increased risk of bleeding
  - *Mechanism*: Chronic use of paracetamol may enhance the anti-coagulant effect of warfarin, although this interaction is generally considered to be minor and occurs primarily with prolonged use.
- *Carbamazepine*:
  - *Interaction*: Reduced effectiveness of paracetamol
  - *Mechanism*: Carbamazepine induces hepatic enzymes that can increase the metabolism of paracetamol, potentially reducing its analgesic effect.
- *Rifampin*:
  - *Interaction*: Reduced effectiveness of paracetamol
  - *Mechanism*: Rifampin induces liver enzymes, which can increase the metabolism of paracetamol.
- *Phenytoin*:
  - *Interaction*: Increased risk of liver toxicity
  - *Mechanism*: Phenytoin can induce liver enzymes, leading to increased formation of toxic metabolites of paracetamol.
- *Isoniazid*:
  - *Interaction*: Increased risk of liver damage
  - *Mechanism*: Isoniazid inhibits the metabolism of paracetamol, increasing the risk of hepatotoxicity.
- *Lamotrigine*:
  - *Interaction*: Reduced effectiveness of lamotrigine
  - *Mechanism*: Paracetamol can increase the clearance of lamotrigine, potentially reducing its effectiveness.
- *Cholestyramine*:
  - *Interaction*: Reduced absorption of paracetamol
  - *Mechanism*: Cholestyramine binds to paracetamol in the gastro-intestinal tract, reducing its absorption and effectiveness.

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- *Metoclopramide and domperidone*:
  - *Interaction*: Increased absorption of paracetamol
  - *Mechanism*: Both drugs increase gastrointestinal motility, potentially leading to faster absorption of paracetamol.
- *Probenecid*:
  - *Interaction*: Increased levels of paracetamol
  - *Mechanism*: Probenecid inhibits the conjugation of paracetamol, which can increase its plasma concentration and prolong its effects.
- *Other hepatotoxic medications (e.g., methotrexate, amiodarone)*:
  - *Interaction*: Increased risk of liver damage
  - *Mechanism*: Concurrent use of other hepatotoxic drugs can exacerbate the risk of liver injury associated with paracetamol.

Paracetamol (acetaminophen) is generally considered safe when used as directed, but there are certain contraindications and situations where its use should be avoided or used with caution. The key contraindications and precautions are as follows:

- *Contraindications*:
  - Hypersensitivity
  - Severe liver disease
  - Severe renal impairment
    - While not an absolute contraindication, caution is advised in patients with severe renal impairment, and dosage adjustments may be necessary.
- *Precautions*:
  - Chronic alcoholism
  - Malnutrition
    - Malnourished individuals may have depleted glutathione stores, which are necessary for the detoxification of paracetamol metabolites, increasing the risk of toxicity.
  - Concurrent use with other medications
    - Caution is needed when paracetamol is used in combination with other hepatotoxic drugs or drugs that interact with its metabolism (e.g., isoniazid, phenytoin).
  - Glucose-6-phosphate dehydrogenase (G6PD) deficiency
    - Individuals with G6PD deficiency may have an increased risk of hemolysis when using paracetamol.
  - Special populations:
    - Elderly:
      - Older adults may be more susceptible to adverse effects due to concurrent illnesses or polypharmacy. Dose adjustments may be necessary based on liver and kidney function.
    - Children:
      - Paracetamol is commonly used in pediatric populations, but dosages must be carefully calculated based on weight to avoid overdose.

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## Nonsteroidal Anti-inflammatory Drugs

Nonsteroidal anti-inflammatory drugs are a class of medication used for the treatment of pain, fever, and other inflammatory processes.

It has a peripheral action and exerts anti-inflammatory action by inhibiting prostaglandin through the COX pathway. COX is required to convert arachidonic acid into thromboxane, prostaglandin, and prostacyclin. These substances are involved in inflammation, pain, and fever. A more detailed explanation of the mechanism of action of NSAIDs is given in the following text.

There are two main isoforms of the COX enzyme:

1. *COX-1*:
   a. Constitutively expressed in most tissues
   b. Involved in the production of prostaglandins that protect the stomach lining, maintain renal blood flow, and support platelet function.
2. *COX-2*:
   a. Inducible enzyme, usually expressed at sites of inflammation
   b. Produces prostaglandins that mediate inflammation, pain, and fever

### *Effects of Cyclooxygenase Inhibition*

- Anti-inflammatory
- Analgesic
- Antipyretic

### *Nonspecific Nonsteroidal Anti-inflammatory Drugs (Inhibit both COX-1 and -2)*

Commonly used nonspecific NSAIDs include diclofenac, ibuprofen, indomethacin, ketorolac, mefenamic acid, meloxicam, naproxen, and piroxicam.

They are used to treat mild-to-moderate pain, starting from the lowest effective dose and for the shortest possible duration of treatment.

*COX-2 selective* inhibitors include drugs like celecoxib, rofecoxib, and valdecoxib (the last two were withdrawn from the market in 2004 and 2005, respectively). They are as effective as nonselective NSAIDs for the treatment of mild-to-moderate pain. They exhibit analgesic and anti-inflammatory actions by inhibiting the COX-2 which is present in the inflammatory cells and damaged tissues. However, they are associated with less gastrointestinal side effects.

Selective COX-2 inhibitors should not be prescribed in patients with ischemic heart disease, cerebrovascular disease, and peripheral artery disease and should be prescribed cautiously in patients with hypertension, diabetes mellitus, hyperlipidemia, smokers, and those who are hypersensitive to the drug.

Nonsteroidal anti-inflammatory drugs are contraindicated in patients who are allergic to them, who have undergone coronary artery bypass graft

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surgery, during the thirdtrimester of pregnancy, and patients with asthma, especially children.

*Adverse effects of NSAIDs* include *gastric* (it prevents the creation of prostaglandin that protects gastric mucosa), *cardiovascular* [myocardial infarction (MI), thromboembolic event, atrial fibrillation (AF)], *renal* (acute renal dysfunction, fluid and electrolyte disorders), *hematological* (antiplatelet activity), and *anaphylactoid* reactions.

The exact dose of NSAIDs can vary based on the specific medication, the condition being treated, patient factors, and other considerations. The commonly prescribed doses for some widely used NSAIDs are as follows:

- *Ibuprofen*:
  - *Adult dosage*: 400–800 mg orally every 6–8 hours as needed
  - *Maximum daily dosage*: 3,200 mg
- *Naproxen*:
  - *Adult dosage*: 250–500 mg orally every 12 hours as needed
  - *Maximum daily dosage*: 1,000 mg (1,500 mg for the first day of treatment)
- *Diclofenac*:
  - *Adult dosage*: 50 mg orally every 8 hours as needed
  - *Maximum daily dosage*: 150 mg
- *Celecoxib (a COX-2 inhibitor)*:
  - *Adult dosage*: 100–200 mg orally once or twice daily
  - *Maximum daily dosage*: 400 mg
- *Meloxicam*:
  - *Adult dosage*: 7.5–15 mg orally once daily
  - *Maximum daily dosage*: 15 mg
- *Ketorolac*:
  - *Adult dosage*: 10 mg orally every 4–6 hours as needed
  - *Maximum daily dosage*: 40 mg
  - *Note*: Use is typically limited to a maximum of 5 days due to the risk of gastrointestinal and renal side effects.
- *Indomethacin*:
  - *Adult dosage*: 25–50 mg orally two to three times daily
  - *Maximum daily dosage*: 200 mg
- *Aceclofenac*:
  - *Adults*: The usual recommended dose is 100 mg taken twice daily.
  - *Elderly*: Same as adults, but with close monitoring for adverse effects due to increased susceptibility.
  - *Children*: Not recommended for use in children
- *Etoricoxib*:
  - *Dosage*:
    - Osteoarthritis: 30 or 60 mg once daily
    - Rheumatoid arthritis: 60 or 90 mg once daily
    - Ankylosing spondylitis: 60 or 90 mg once daily
    - Acute pain (e.g., gout): 120 mg once daily, limited to a maximum of 8 days

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### *Drug Interaction in Case of Nonsteroidal Anti-inflammatory Drug Usage*

Nonsteroidal anti-inflammatory drugs can interact with a variety of other medications, potentially leading to increased risks of adverse effects or decreased effectiveness of treatments. Some common and significant drug interactions with NSAIDs are as follows:

- *Anticoagulants* (e.g., warfarin, heparin, direct oral anticoagulants)
  - *Interaction*: Increased risk of bleeding
  - *Mechanism*: NSAIDs inhibit platelet aggregation and can cause gastrointestinal bleeding, which can be compounded by anticoagulants.
- *Antihypertensive medications* [e.g., angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), diuretic]
  - *Interaction*: Reduced effectiveness of antihypertensive medications
  - *Mechanism*: NSAIDs can cause sodium and water retention, leading to increased blood pressure.
- *Corticosteroids* (e.g., prednisone)
  - *Interaction*: Increased risk of gastrointestinal ulcers and bleeding
  - *Mechanism*: Both NSAIDs and corticosteroids can cause gastrointestinal irritation and ulceration.
- *Selective serotonin reuptake inhibitors* (SSRIs; e.g., fluoxetine, sertraline)
  - *Interaction*: Increased risk of gastrointestinal bleeding
  - *Mechanism*: SSRIs can inhibit platelet function, and NSAIDs can cause gastrointestinal irritation, leading to a higher risk of bleeding.
- *Lithium*:
  - *Interaction*: Increased lithium levels and potential toxicity
  - *Mechanism*: NSAIDs can reduce renal clearance of lithium.
- *Methotrexate*:
  - *Interaction*: Increased risk of methotrexate toxicity
  - *Mechanism*: NSAIDs can decrease renal clearance of methotrexate.
- *Digoxin*:
  - *Interaction*: Increased digoxin levels
  - *Mechanism*: NSAIDs can reduce renal clearance of digoxin.
- *Cyclosporine*:
  - *Interaction*: Increased risk of nephrotoxicity
  - *Mechanism*: Both NSAIDs and cyclosporine can have nephrotoxic effects.
- *Aspirin*:
  - *Interaction*: Reduced cardioprotective effect of low-dose aspirin; increased risk of gastrointestinal side effects.
  - *Mechanism*: NSAIDs can interfere with the antiplatelet effect of low-dose aspirin.

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**BOX 1 Potential high-risk patients.**

- >60 years of age
- Previous history of ulceration
- Concomitant steroid use
- Systemic disease, e.g., renal impairment, heart failure, carcinomatosis, increased risk of bleeding, e.g., on warfarin
- Patients on a higher dose of nonsteroidal anti-inflammatory drug

- *Alcohol*:
  - *Interaction*: Increased risk of gastrointestinal bleeding and ulcers
  - *Mechanism*: Both alcohol and NSAIDs can cause gastrointestinal irritation.

It is important to have the information about all the medications the patient is taking and to tailor the treatment to the individual's needs and medical condition. Long-term use of NSAIDs should be monitored due to potential side effects such as gastrointestinal bleeding, cardiovascular risks, and renal impairment.

### *Various Do's and Don'ts of Nonsteroidal Anti-inflammatory Drug Use*

- NSAIDs should be used cautiously in all patients and any alternative analgesics should always be tried first line.
- They should be used initially in normal-risk patients.
- Stop NSAIDs in suspected peptic ulcer.
- Do not initiate NSAIDs during active ulceration.
- If NSAIDs are essential in high-risk patients, add long-term lansoprazole 30 mg OD.
- Patients with more than one risk factor **(Box 1)** can be considered for treatment either with coxib alone or in combination with an established NSAID plus lansoprazole 30 mg OD.

Around 20% of patients prescribed coxibs report dyspeptic symptoms, and these side effects may limit use.

### *Who Needs Prophylaxis?*

- Give prophylaxis against peptic ulcer if the patient has a history of peptic ulcer or bleeding on NSAID.
- Consider prophylaxis in high-risk patients.

## MANAGING GASTROINTESTINAL SIDE EFFECTS OF NONSTEROIDAL ANTI-INFLAMMATORY DRUGS

Management of gastrointestinal side effects of nonsteroidal anti-inflammatory drugs is shown in **Flowchart 1**.

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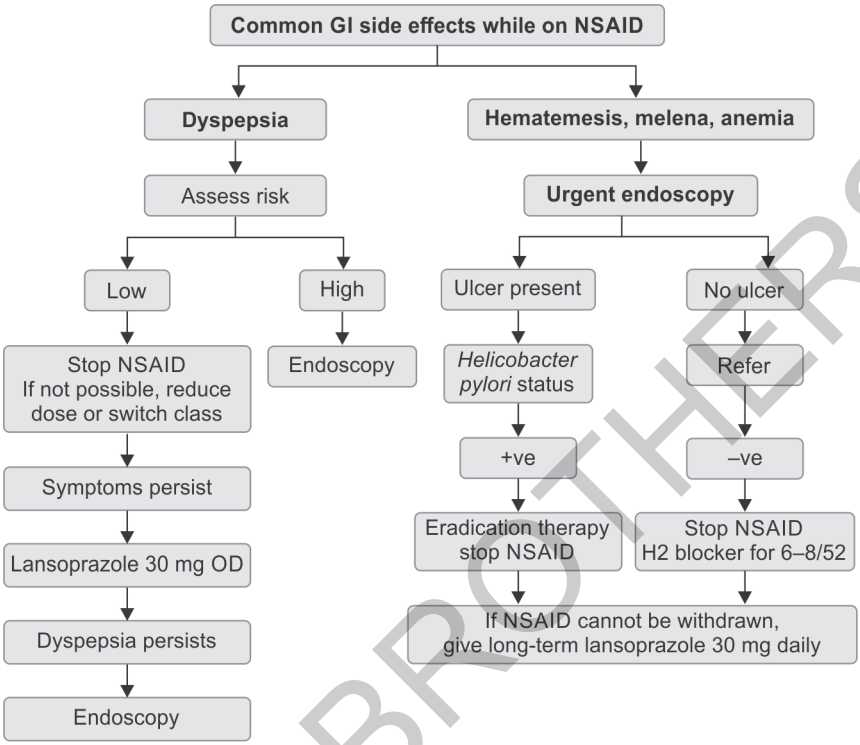


**Flowchart 1:** Algorithm management of side effects of NSAID.

Note: If gastric ulcer is proven, endoscopy must be repeated after 8–12 weeks of treatment to confirm healing.

(GI: gastrointestinal; NSAID: nonsteroidal anti-inflammatory drug)

## CONCLUSION

Nonsteroidal anti-inflammatory drugs play a vital role in managing musculoskeletal pain, providing effective relief for many patients. However, their use must be balanced with considerations of potential side effects, such as gastrointestinal, cardiovascular, and renal risks. Healthcare providers should tailor NSAID therapy to each patient's specific condition, risk factors, and overall health status, incorporating both pharmacologic and non-pharmacologic approaches. As research continues to evolve, a more nuanced understanding of NSAID therapy will help clinicians maximize their benefits while minimizing harm, ultimately improving patient outcomes in musculoskeletal pain management.

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# Musculoskeletal Pain Management

Musculoskeletal Pain Management is a comprehensive guide addressing the multifaceted realm of pain management within the orthopaedic and musculoskeletal domains. Authored by experts in the field, the book offers an in-depth exploration of various facets of pain, ranging from its physiological mechanisms to its clinical implications and treatment modalities.

Through a structured approach, the book delves into the anatomy and pathophysiology underlying orthopedic conditions and trauma-induced pain. It elucidates the intricacies of diagnosing different types of musculoskeletal pain, including acute injuries, chronic conditions, and postoperative discomfort.

Moreover, the text provides a thorough examination of contemporary pain management strategies, encompassing pharmacological interventions, interventional procedures, physical therapy modalities, and complementary therapies. It emphasizes a multidisciplinary approach, advocating for collaboration among orthopedic surgeons, pain specialists, physical therapists, and other healthcare professionals to optimize patient outcomes.

Furthermore, the book discusses the importance of patient education, shared decision-making, and holistic care in managing orthopedic pain effectively. It underscores the significance of addressing not only the physical but also the psychological and social aspects of pain to achieve comprehensive and sustainable pain relief.

In essence, this book on "Musculoskeletal Pain Management" serves as a valuable resource for healthcare professionals involved in the care of patients with orthopedic conditions, offering evidence-based insights and practical guidance for enhancing pain management practices and improving patient quality of life.

**Abdul Ghani** MBBS MS MRCS is a distinguished Orthopedic Surgeon and Professor of Orthopedics, with over 25 years of experience in patient care and teaching undergraduate and postgraduate medical students. After post-graduation from India's most prestigious medical institute (AIIMS Delhi), he did his MRCS in the United Kingdom, where he got working experience of more than 06 years. He is proactively involved in teaching and research with many national and international publications and presentations to his credit.

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