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Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) are Members of a Drug Class that Reduces Pain, Decreases Fever, Prevents Blood Clots, and Higher Doses, Decreases Inflammation

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Description

Nonsteroidal anti-inflammatory drugs (NSAIDs) are members of a drug class that reduces pain, decreases fever, prevents blood clots, and in higher doses, decreases inflammation. Side effects depend on the specific drug but largely include an increased risk of gastrointestinal ulcers and bleeds, heart attack, and kidney disease. The term nonsteroidal distinguishes these drugs from steroids, which while having a similar eicosanoid-depressing, anti-inflammatory action, have a broad range of other effects. First used in 1960, the term served to distance these medications from steroids, which were particularly stigmatised at the time due to the connotations with anabolic steroid abuse.

NSAIDs are useful in the management of post-operative dental pain following invasive dental procedures such as dental extraction. When not contra-indicated they are favoured over the use of paracetamol alone due to the anti-inflammatory effect they provide. When used in combination with paracetamol the analgesic effect has been proven to be improved. There is weak evidence suggesting that taking pre-operative analgesia can reduce the length of post-operative pain associated with placing orthodontic spacers under local anaesthetic. Combination of NSAIDs with pregabalin as pre-emptive analgesia has shown promising results for decreasing post-operative pain intensity.

There is an argument over the benefits and risks of NSAIDs for treating chronic musculoskeletal pain. Each drug has a benefit-risk profile and balancing the risk of no treatment with the competing potential risks of various therapies should be considered. For people over the age of 65 years old, the balance between the benefits of pain-relief medications such as NSAIDS and the potential for adverse effects has not been well determined. The main adverse drug reactions (ADRs) associated with NSAID use relate to direct and indirect irritation of the gastrointestinal (GI) tract. NSAIDs cause a dual assault on the GI tract: the acidic molecules directly irritate the gastric mucosa, and inhibition of COX-1 and COX-2 reduces the levels of protective prostaglandins.

Certain NSAIDs, such as aspirin, have been marketed in enteric-coated formulations that manufacturers claim reduces the incidence of gastrointestinal ADRs. Similarly, some believe that rectal formulations may reduce gastrointestinal ADRs. However, consistent with the systemic mechanism of such ADRs, and in clinical practice, these formulations have not demonstrated a reduced risk of GI ulceration. Hydrogen sulphide NSAID hybrids prevent the gastric ulceration/bleeding associated with taking the NSAIDs alone. Hydrogen sulphide is known to have a protective effect on the cardiovascular and gastrointestinal system.

NSAIDs are also associated with a fairly high incidence of adverse drug reactions (ADRs) on the kidney and over time can lead to chronic kidney disease. The mechanism of these kidney ADRs is due to changes in kidney blood flow. Prostaglandins normally dilate the afferent arterioles of the glomeruli. This helps maintain normal glomerular perfusion and glomerular filtration rate (GFR), an indicator of kidney function. This is particularly important in kidney failure where the kidney is trying to maintain renal perfusion pressure by elevated angiotensin II levels.