
Good Morning

PHARMACOTHERAPY FOR ORO-FACIAL PAIN USING NSAIDs

CONTENTS

Introduction

Orofacial pain can result form two general pathological mechanism, tissue injury, and inflammation, or from a primary lesion or dysfuntion of nervous system.

The first step in management of orofacial pain is the determination if the pain is primarily nociceptive or neuropathic or combination of both.

The NSAIDs are a group of chemically dissimilar agents that differ in their antipyretic, analgesic, and anti-inflammatory activities.

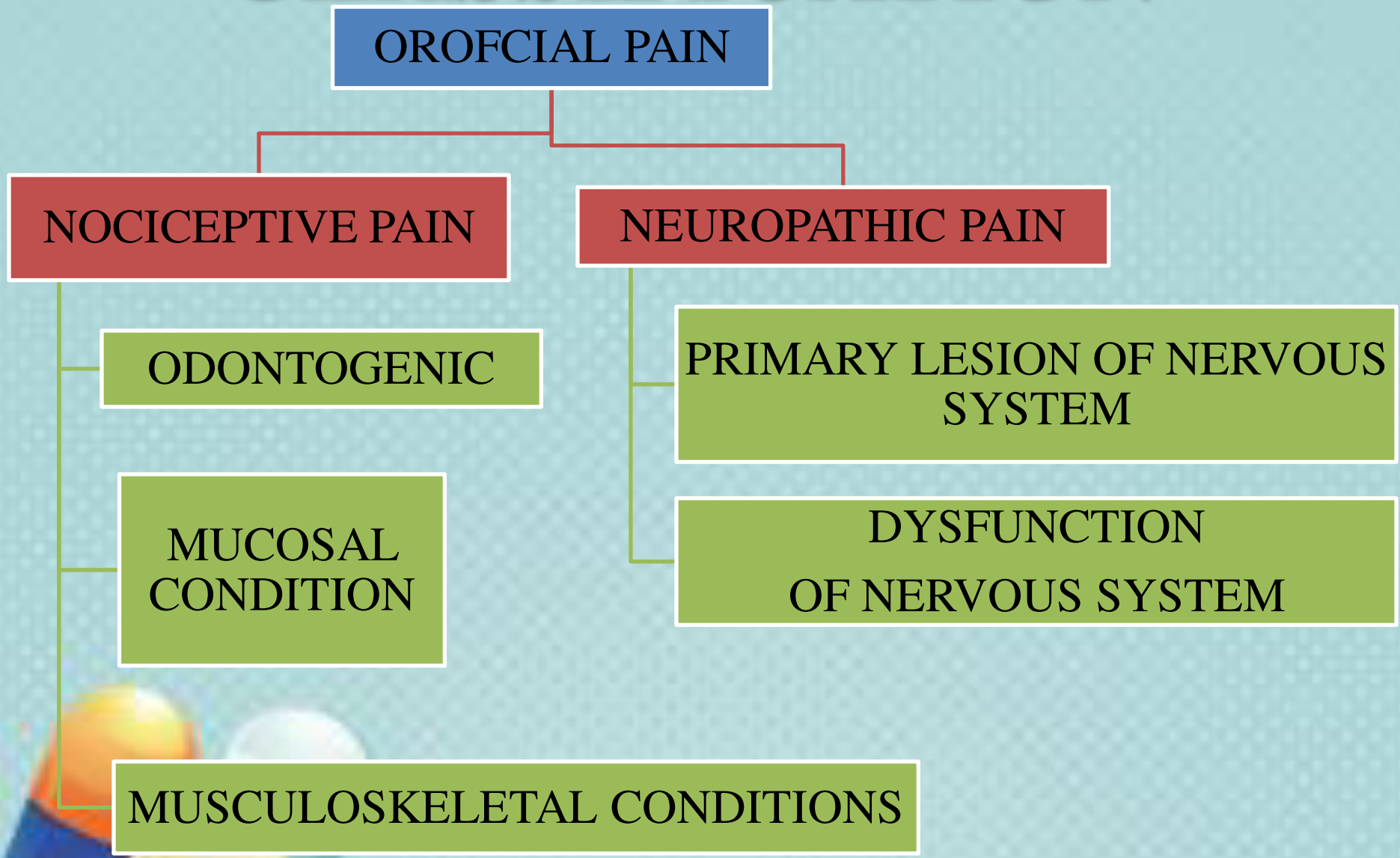
Terminology

Pain:

An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

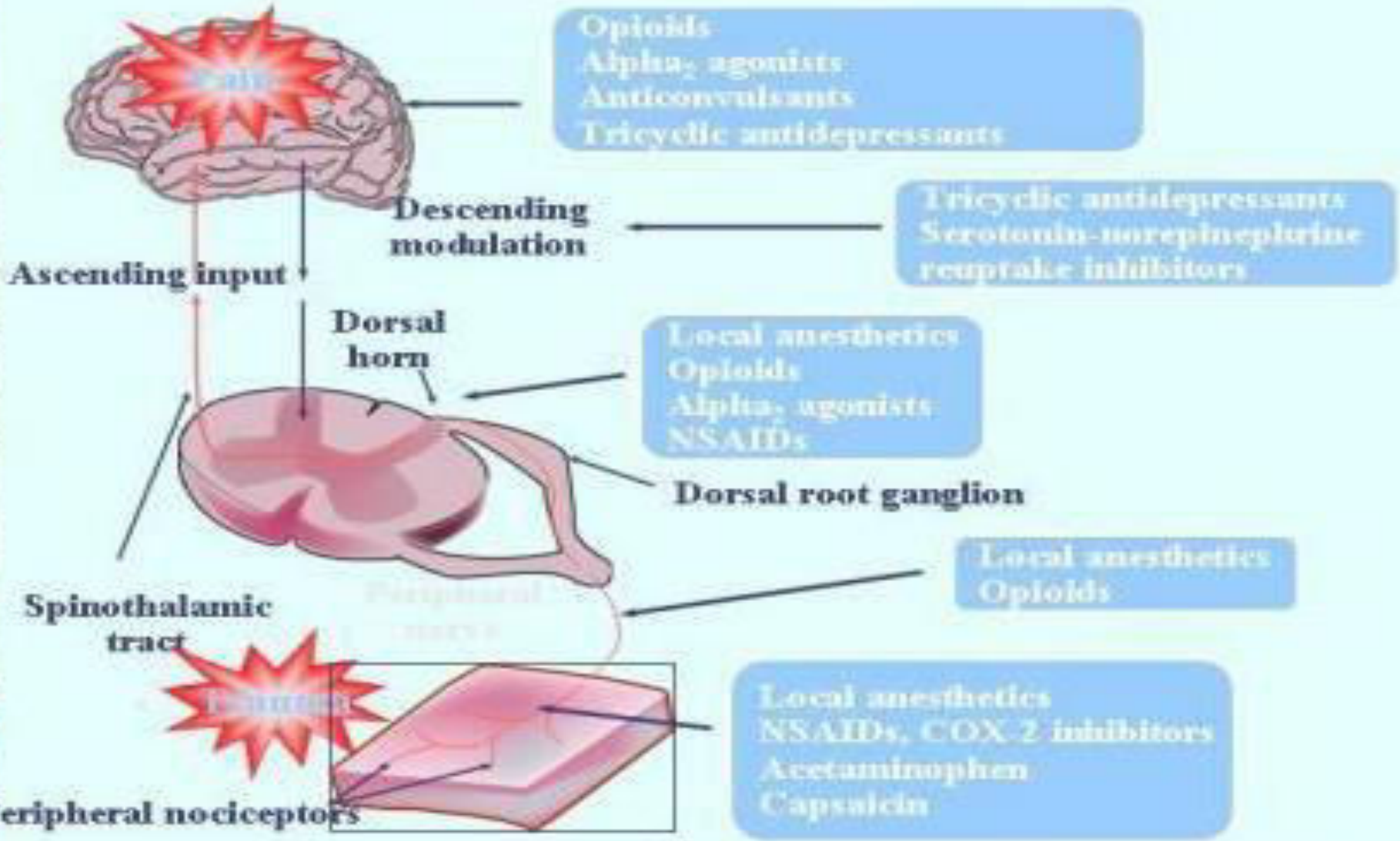
(Task force on Taxonomy of the international association for the study of pain.)

CLASSIFICATION



Oral medicine, Burket's, 11th edition

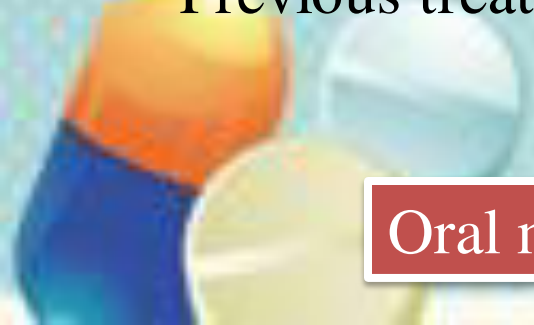
Mechanisms of Pain and Medication Activity Sites



Gottschalk A, et al. *Am Fam Physician*. 2001;63:1979-1984. Adapted from artist Klemm D.

Pain Characteristics

Intensity
Quality
Location
Onset
Associated events at onset
Duration and timing of pain
Course of symptoms since onset
Activities that increase pain
Activities that decrease pain
Associated symptoms
Previous treatment and their effects



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PAIN RATING SCALE

Simple category scales such as four-point "none, mild, moderate and severe" (verbal categorical scale).

1-10 numerical scale can be scored in several ways. The simplest, the method of equal appearing intervals assigns successive integers to verbal categories directly.

A Visual Analogue Scale (VAS) is a simple measure of subjective pain. It consists of a 10-cm horizontal or vertical line with two end points labeled "no pain" and "worst pain ever".

The subject is required to place a mark on the 10-cm line at a point, which corresponds to the level of pain intensity the subject presently feels. The distance in cm from the lower end of the VAS to the patients mark is used as a numerical index of the severity of pain.

Indian J Physiol Pharmacol 2003; 47(3)

Differential diagnosis of orofacial pain

Intracranial pain disorders	Neoplasm, aneurysm, abscess, hemorrhage, hematoma, edema.
Primary headache disorders	Migraine, Cluster headache
Neurogenic pain disorders	Paroxysmal neuralgia
Intraoral pain disorders	Dental pulp, periodontium, mucogingival tissues.
Temporomandibular disorders	Masticatory muscle, TMJ associated structures
Associated structure	Eyes, ears, nose, paranasal sinuses, throat, lymph nodes, salivary glands, neck

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NSAIDS

All of the NSAIDs act by inhibiting the synthesis of prostaglandins. Thus, an understanding of NSAIDs requires comprehension of the actions and biosynthesis of prostaglandins.

Compared to morphine they are weaker analgesics;

- Do not depress CNS
- Do not produce physical dependence
- No abuse liability

Other names:

- Non-narcotic
- Non-opoid
- Aspirin-like analgesics

Essentials of medical pharmacology, K.D.Tripathi. 5th edition.

HISTORY

The use of willow bark and leaves to relieve fever has been attributed to Hippocrates, but was most clearly documented by the Rev.

YEAR		HISTORY
1829	Leroux	Salicin was crystalized
1836	Pina	isolated salicylic acid
1859 1874	Kolbe	synthesized salicylic acid being produced industrially
1899	Hoffmann	Sought to improve the adverse-effect profile of salicylic acid.
1899	Bayer	that a drug was tested on animals in an industrial setting and proceeded soon thereafter to human studies and the marketing of aspirin.

Goodman Gillman Pharmacology 11th Ed, 2006.

CLASSIFICATION

Non selective COX inhibitors(Conventional NSAIDs):

1.Salicylates: Aspirin, Diflunisal.
2.Pyrazolone derivatives: Phenylbutazone, Oxyphenbutazone.
3.Indole derivatives: Indomethacin, Sulindac.
4.Propionic acid derivatives: Ibuprofen, Naproxen, Ketoprofen, Flurbiprofen.
5.Anthranilic acid derivtive: Diclofenac.
6.Oxicam derivatives: Piroxicam, Tenoxicam.
7.Pyrrolo-pyrrole derivative: Ketorolac.

Preferential COX-2 inhibitors:

Nimesulide, Meloxicam, Nabumetone.

Selective COX-2 inhibitors

Celecoxib, Rofecoxib, Valdecoxib.

Analgesic-antipyretics with poor anti-inflammatory action:

1. Paraaminophenol derivative: Paracetamol (Acetaminophen)

2. Pyrazolone derivatives: Metamizol, Propiphenazone

3. Benzoxazocine derivative: Nefopam

Essentials of medical pharmacology, K.D.Tripathi. 5th edition.

ANALGESIC EFFECT

The NSAIDs are effective against mild or moderate pain, especially that arising from inflammation or tissue damage.
Two sites of action have been identified.

1. Peripherally, they decrease production of the prostaglandins that sensitise nociceptors to inflammatory mediators such as bradykinin and they are therefore effective in arthritis, pain of muscular and vascular origin, toothache, and the pain of cancer metastases in bone-all conditions that are associated with increased local prostaglandin synthesis

2. Central action, possibly in the spinal cord. Inflammatory lesions increase prostaglandin release within the cord, causing facilitation of transmission from afferent pain fibres to relay neurons in the dorsal horn.

Rang and Dale's pharmacology, 6th edition

ANTIPYRETIC EFFECT

Normal body temperature is regulated by a centre in the hypothalamus that controls the balance between heat loss and heat production.

Fever occurs when there is a disturbance of this hypothalamic 'thermostat', which leads to the set point of body temperature being raised. NSAIDs 'reset' this thermostat.

Once there has been a return to the normal set point, the temperature-regulating mechanisms then operate to reduce temperature. Normal body temperature in humans is not affected by NSAIDs.

Rang and Dale's pharmacology, 6th edition

ANTI-INFLAMMATORY EFFECTS

Many mediators coordinate inflammatory and allergic reactions. While some are produced in response to specific stimuli.

The NSAIDs reduce mainly the components of the inflammatory and immune response in which prostaglandins, mainly derived from COX-2, play a significant part.

These include:
Vasodilatation, oedema, pain , again potentiating other mediators, such as bradykinin.
The NSAIDs suppress the pain, swelling and increased blood flow associated with inflammation but have little or no action on the actual progress of the underlying chronic disease itself.

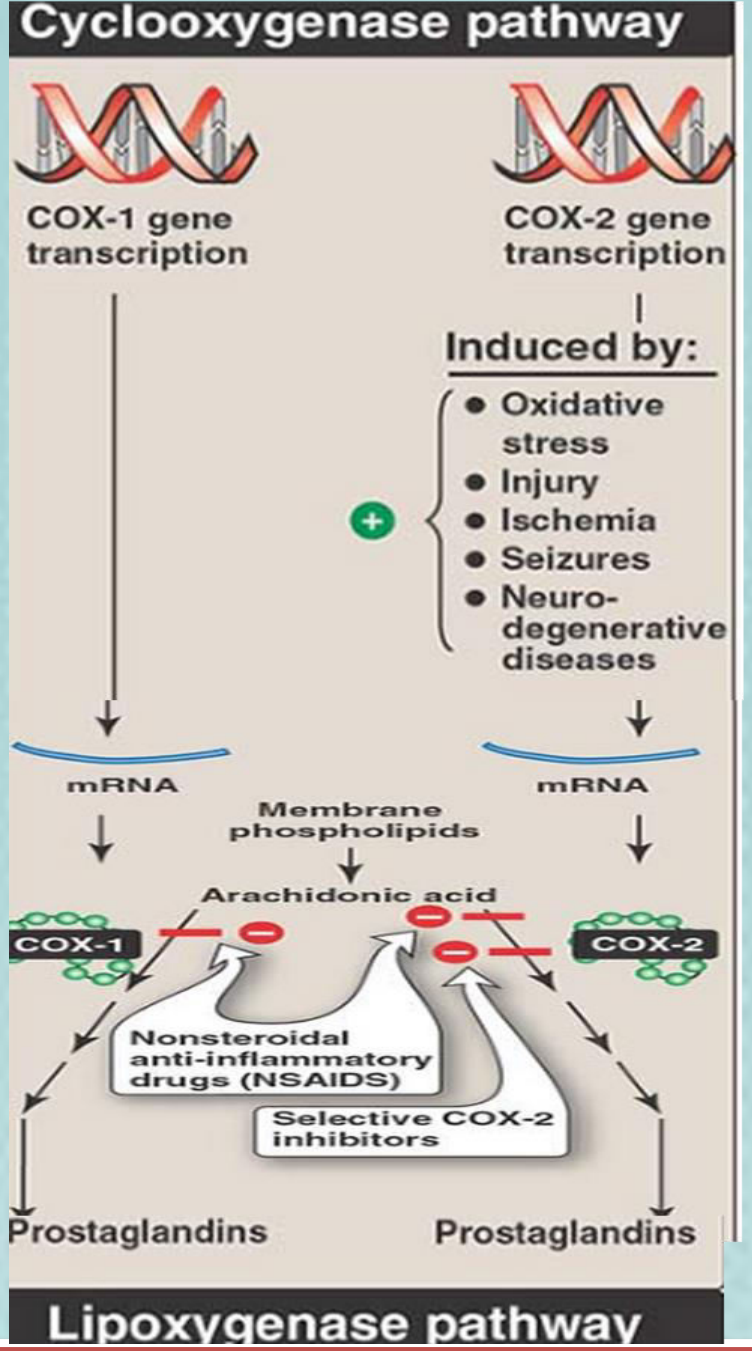
Rang and Dale's pharmacology, 6th edition

MECHANISM OF ACTION

NSAIDs block cycloxygenase and prostaglandin synthesis, thereby reducing pain and inflammation due to nociceptor sensitization.

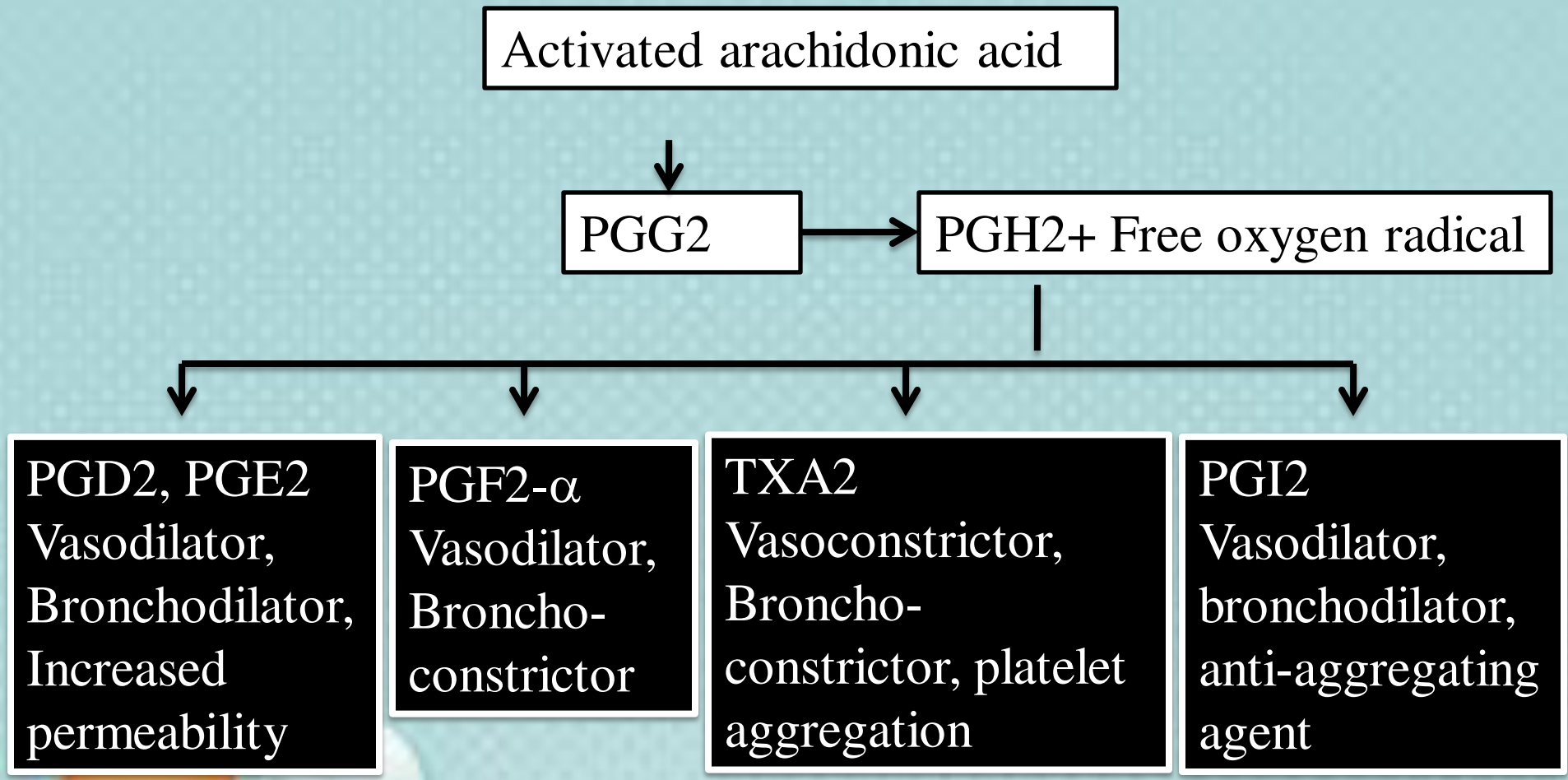
Nonselective NSAIDs block both isoforms of cycloxygenase, namely COX 1 and COX 2.

Selective COX 2 inhibitor, blocks only COX2, thus overcomes the GI side effects of COX 1 inhibition.



Lippincott'sIllustratedReviews Pharmacology 4thEdition.

Cyclo-oxygenase pathway



GENE	TISSUE EXPRESSION	FUNCTIONS	INHIBITORS
COX 1	Constitutively expressed in most tissues Physiological house keeping function.	Platelet aggregation, gastrointestinal protection, some pain, production of vascular prostacyclin	Most classic NSAIDs, some selective inhibitors
COX 2	Induced in many tissues by many stimuli, including growth factors, cytokines, oxidative stress, brain hypoxia or seizures, and other forms of injury or stress; constitutively present in brain, kidney and elsewhere	Inflammation, other pathological changes.	Many NSAIDs, COX-2- selective drugs such as the coxibs and others

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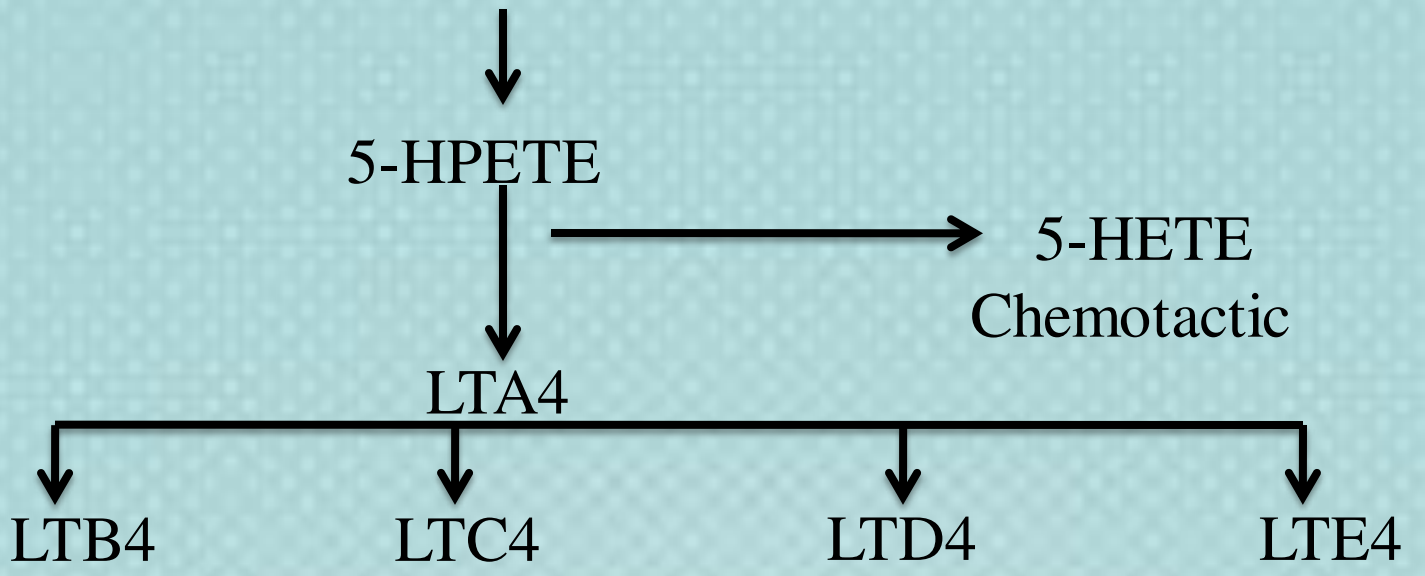
Lipoxygenase pathway

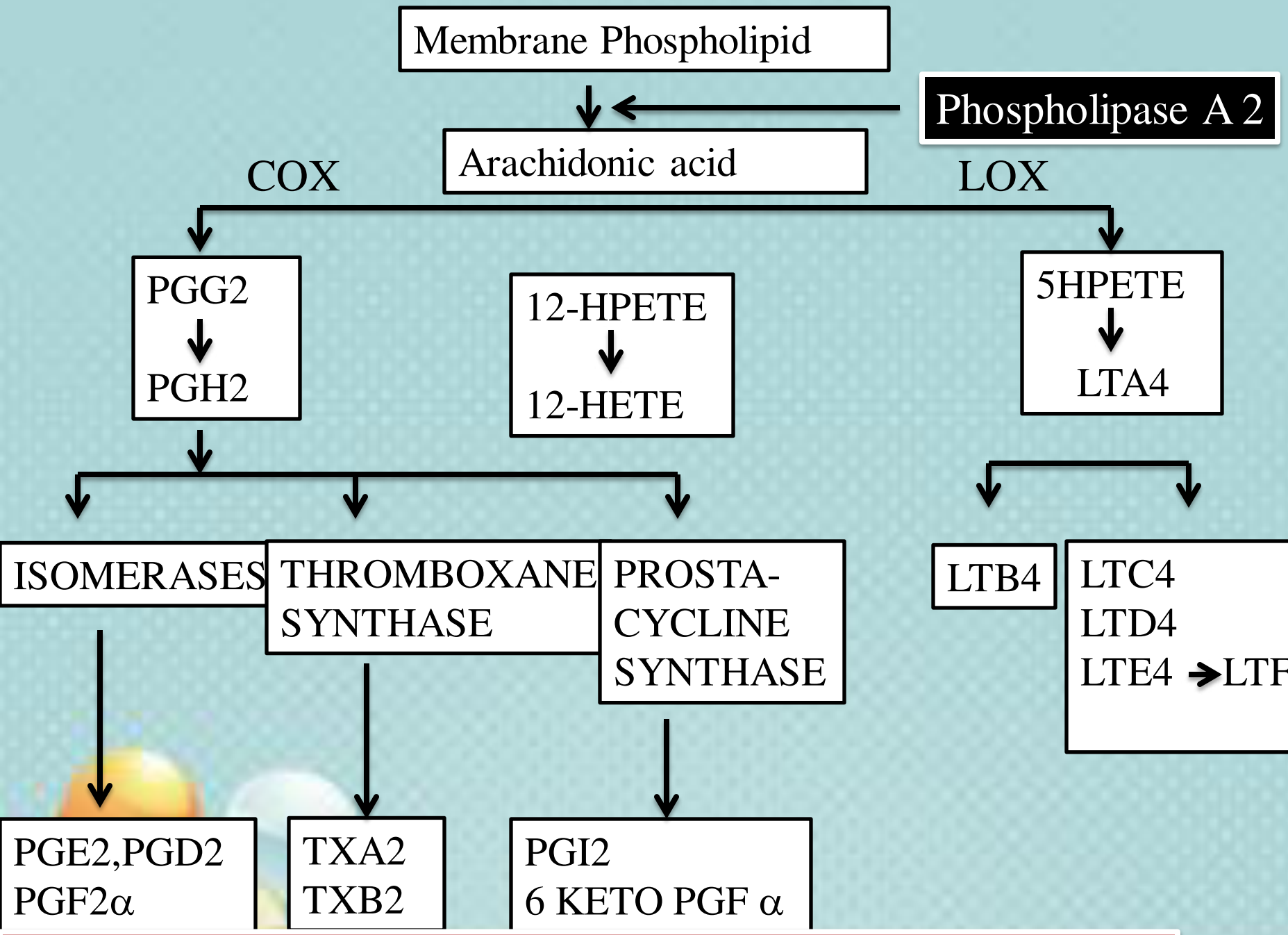
Operate mainly in lung, WBC, platelets.

Activated arachidonic acid



Chemotactic
Cell adherence

Smooth muscle constrictor
Vasoconstrictor
Bronchoconstrictor
Increase vascular permeability



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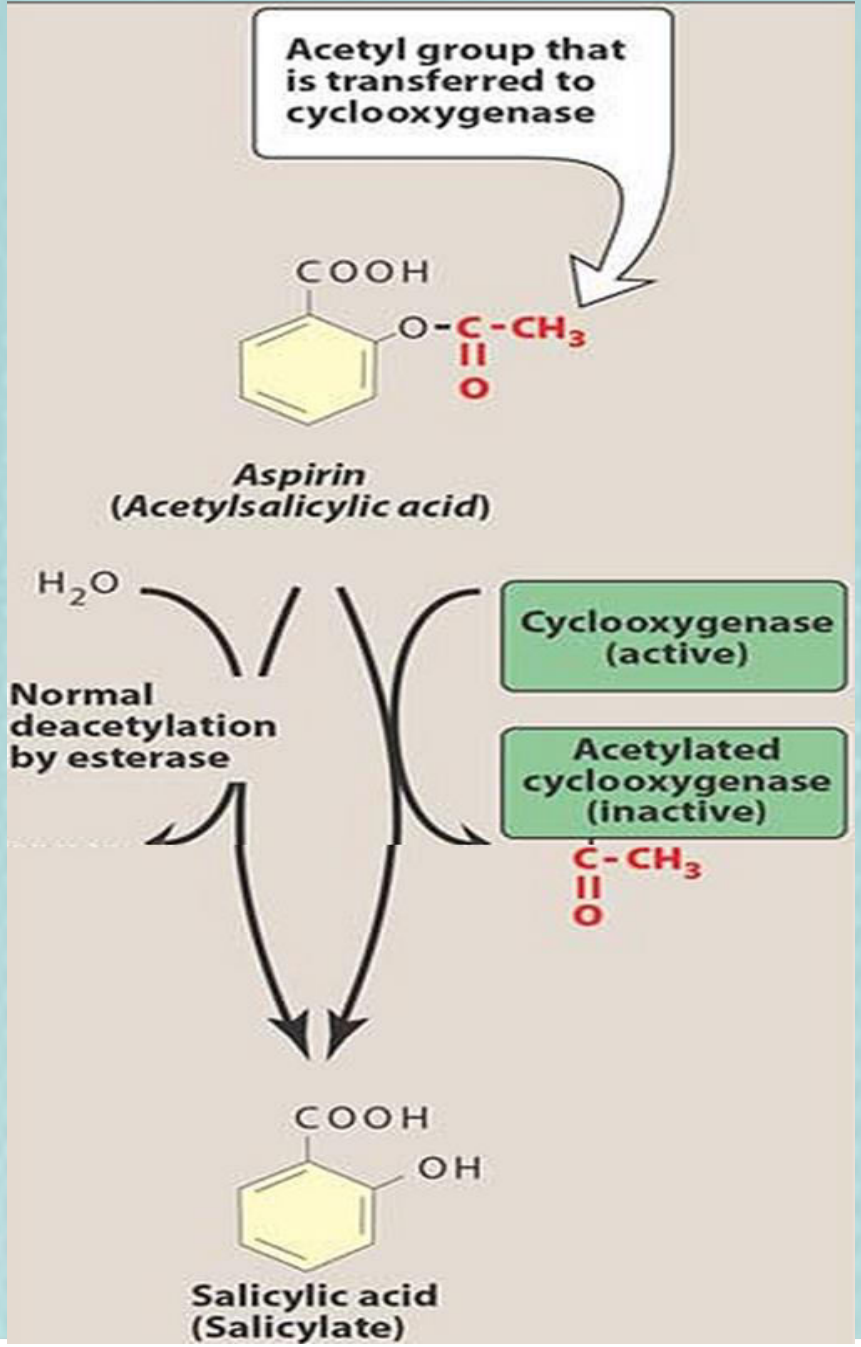
COX: Cycloxygenase
LOX: Lipoxygenase
PG: Prostaglandin
PGI2: Prostacyclin
TXA2: Thromboxane
LT: Leukotriene
HPETE: Hydroperoxyeicosatetraenoic acid
HETE: Hydroxyeicosatetraenoic acid

Salicylates



Aspirin is acetyl salicylic acid.
MECHANISM

LippincottsIllustratedReviews Pharmacology 4thEdition.

Pharmacological actions

- Aspirin is a weaker analgesic than morphine.
- Obtunding of peripheral pain receptors and prevention of PG mediated sensitization of nerve endings.

Analgesic

- Aspirin resets the hypothalamic thermostat and reduces fever by promoting heat loss.

Antipyretic

- Anti-inflammatory action (3-6g/day) Signs of inflammation like vasodilatation, leukocyte infiltration are suppressed.

Anti-inflammatroy

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- **At anti-inflammatory doses,**
- Cellular metabolism is increased.
- Increased utilization of glucose.

Metabolic effects

- Hyperventilation is prominent in salicylate poisoning.
- Later respiratory depression and death due to respiratory failure.

Respiration

- **Anti-inflammatory dose,**
- Respiratory stimulation predominates and tends to wash out CO2 despite increased production leading to ***respiratory alkalosis.***
- Still higher doses causes respiratory depression with CO2 retention, while excess CO2 production continues leads to ***respiratory acidosis.***

Acid-base electrolyte balance

- occurs in poisoning due to increased water loss in urine, increased sweating and hyperventilation

Dehydration

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- Large doses increase cardiac output to meet increased peripheral O2 demand and cause vasodilatation.
- Toxic doses cause fall in BP.

CVS

- Irritation of gastric mucosa leading to epigastric distress, nausea and vomiting.

GIT

- Dose related effect is seen:
- **< 2g/day:** Urate retention and antagonism of all other uricosuric drugs
- **2-5g/day:** Variable effects, often no change
- **>5g/day**: Increased urate excretion.

Urate excretion

- irreversibly inhibits TXA2 synthesis by platelets. Interferes with platelet aggregation and bleeding time is prolonged.

Blood

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INDICATION

- For headache, back ache, myalgia, joint pain , pulled muscle, tooth ache, neuralgias. → ***As analgesic***
- Effective in fever of any origin. → ***As antipyretic***
- aspirin is the first drug of choice
- Dose: 4-6g/day, brings relief in 1-3 days. → ***Acute rheumatic fever***
- Dose of 3-5g/day, relief of pain, swelling, morning stiffness. → ***Rheumatoid arthritis***
- symptomatic relief → ***Osteoarthritis***
- by inhibiting platelet aggregation, lowers the incidence of reinfarction. TXA2 synthesis in platelets is inhibited at low doses. → ***Postmyocardial infarction and post stroke patient***

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Pharmacokinetics:

•Absorbed from stomach and small intestine.
•Rapidly deacetylated in the gut wall, liver, plasma and other tissues to release salicylic acid which is the major active form.
•Conjugated in liver with glycine to salicyluric acid.
•Plasma half life: 15-20 min.

Adverse effects:

•***At analgesic dose:*** Nausea, vomiting, epigastric distress.
•***Hypersensitivity and idiosyncracy:*** Rashes, urticaria, rhinorrhoea, angioedema, asthma, anaphylactoid reaction.
•***At anti-inflammatroy doses:*** **Salicylism:** Dizziness, tinnitus, vertigo, reversible impairment of hearing and vision, mental confusion, hyperventilation, electrolyte imbalance.

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Dose: Aspirin 350mg tab, Colsprin 100, 325, 650 mg tab, Ecosprin 75, 150, 325 mg tab, Disprin 350mg tab.
Injection: Biospirin: Lysine acetylsalicylate 900mg + glycine 100mg/ vial for dissolving in 5ml water and IV injection.

Acute salicylate poisoning

Serious toxicity seen at serum salicylate levels > 50mg/dl.

Manifestations are:

Vomiting, dehydration, electrolyte imbalance, hyper/hypoglycemia, petechial hemorrhages, restlessness, hallucinations, hyperpyrexia, convulsions, coma and death due to respiratory failure+ cardiovascular collapse.

Treatment:

IV fluids with sodium, potassium, bicarbonate, glucose.
Gastric lavage.
Hemodialysis, blood transfusion, if needed.

Pyrazolones

Phenylbutazone:

Inhibits COX and is a potent anti-inflammatory drug

Pharmacokinetics:

Metabolized in liver by hydroxylation and glucuronidation.

Plasma half life is 60 hrs.

Adverse effects: Nausea, Vomiting, distress and epigastric ulceration, hypersensitivity reactions such as rashes, serum sickness, hepatitis.

Oxyphenbutazone:

- Metabolite of phenybutazone.
- *Dose:* 100-200mg BD.
- Phenabid 100mg tab

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Metamizole:

- Derivative of amidopyrine.
- Potent analgesic and antipyretic.
- Route: Oral, IM, IV
- Dose: 0.5-1.5g
- Trade name: Analgin 0.5g tab.
- Novalgin 0.5g tab.

Propiphenazone:

- Similar to metamizole.
- Dose: 300-600mg
- Trade name: Sardion
- Dart: Propiphenazone 150mg+ Paracetamol 300mg+ Caffeine 50mg

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Indomethacin

Analgesic, anti-pyretic, Anti-inflammatory drug.

Inhibits PG synthesis and suppresses neutrophil motility

- Partly meabolized in liver to inactive products and excreted by kidney.
- Plasma half life: 2-5 hrs.

Pharmacokinetics

- Rheumatoid arthritis, ankylosing spondylitis, acute gout.

Indications

- Gastric effects: Nausea, anorexia, gastric bleeding, diarrhoea.
- Headache, dizziness, ataxia, mental confusion.

Adverse effects

- Idicin, Indocap 25 mg, 75 mg SR cap

Dose

- Prodrug, converted in the body into active sulfide metabolite
- Anti-inflammatory

Sulindac

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Propionic acid derivatives

IBUPROFEN

Analgesic, anti-pyretic, anti-inflammatory

Inhibit platelet aggregation and prolong bleeding time.

Pharmacokinetics

- Metabolized in liver by hydroxylation and glucuronide conjugation and excreted in urine.
- Plama half life: 2 hrs.

Indications

- Analgesic, anti-pyretic
- Rheumatoid arthritis, osteoarthritis
- Soft tissue injuries, tooth extraction

Adverse effects

- GI effects: Nausea, vomiting, gastric erosion.
- CNS effects: headache, dizziness, blurring of vision, tinnitus, depression.

Contraindications

- Pregnancy, peptic ulcer patient.

Trade name

- Brufen, 400to 800mg TDS

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NAPROXEN: Potent in inhibiting leukocyte migration

Indication: acute gout, ankylosing spondylitis

Dose: 750mg.

Plasma half life: 12-16 hr.

Dose: 250mg BD-TDS

KETOPROFEN: Stabilize lysosomes.

Dose: 50-100mg BD-TDS

FLURBIPROFEN: More effective than ibuprofen.

Gastric side effects are more.

Ocuflur 0.03% eyedrops, 1 drop 6 hourly.

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Anthranilic acid derivative

MEPHENAMIC ACID:

Analgesic, antipyretic, anti-inflammatory drug.

Inhibits COX as well as antagonises certain actions of PGs.

- Partly metabolized and excreted in urine as well as bile.
- Plasma half life is 2 to 4 hrs.

Pharmacokinetics

- Analgesic in muscle, Joint and soft tissue pain

Indications

- Diarrhoea, epigastric distress, skin rashes.

Adverse effects

- Medol 250, 500mg capsule
- Meftal 250, 500mg tab.

Trade name

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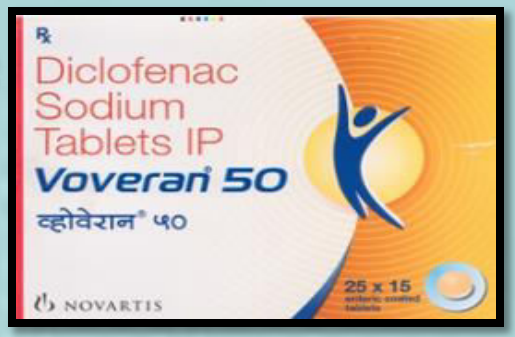
Aryl-acetic acid derivative



DICLOFENAC SODIUM

Analgesic, antipyretic, anti-inflammatory drug

Inhibits PG synthesis and has short lasting anti-platelet action.

Pharmacokinetics

- Well absorbed orally,
- Metabolized and excreted both in urine & bile.
- Plasma half life: 2 hrs.

Indications

- Post-operative/ post-inflammatory conditions, rheumatoid and osteo arthritis.

Adverse effects

- Epigastric pain, Nausea, headache, dizziness, rashes, Gastric ulceration.

Trade name

- Voveran 50mg, Diclomax25 mg, 50mg tab; 3ml inj.

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Oxicam derivative

PIROXICAM:

Anti-inflammatory, analgesic, antipyretic action.

Reversible inhibitor of COX, lowers PG concentration in synovial fluid and inhibits platelet aggregation.

- Metabolized in liver by hydroxylation and glucuronide conjugation; excreted in urine and bile. Plasma half life: 2 days

Pharmacokinetics

- Short term analgesic, long term anti-inflammatory. Dentistry, musculoskeletal injuries, rheumatoid arthritis, ankylosing spondylitis, acute gout.

Indications

- heart burn, nausea, anorexia

Adverse effects

- Dolonex 10, 20mg cap, 20ml injection

Trade name

- Tobitil 20mg tab. OD

Tenoxicam

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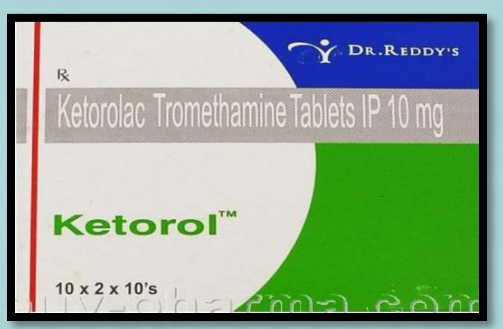
Pyrrolo-Pyrrole derivative



KETOROLAC: Potent analgesic , moderate anti-inflammatory activity.

- highly plasma bound and excreted unchanged in urine.
- Major metabolic pathway is glucouronidation.
- Plasma half life: 5-7 hrs.

Pharmacokinetics

- Post-operative pain, musculoskeletal pain, migraine, renal colic.

Indications

- nausea, abdominal pain, dyspepsia, ulceration, loose stools drowsiness, dizziness, headache.

Adverse effects

- Ketorol, ketonov 10mg tab, 30 mg in 1ml amp.

Trade name

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Preferential COX-2 inhibitors

NIMESULIDE:

Weak inhibitor of PG synthesis.

Relative COX-2 selectivity.

Analgesic, antipyretic, anti-inflammatroy.

Pharmacokinetics

- Completely absorbed orally, extensively Metabolized and excreted in urine.
- Plasma half life: 2-5 hrs

Indications

- Dental surgery, sports injury, sinusitis, fever, postoperative, osteoarthritis.

Adverse effects

- Nausea, rash, pruritis, somnolence, dizziness, diarrhoea.

Trade name

- Nimulid, nimugesic, Nimodol 100mg BD

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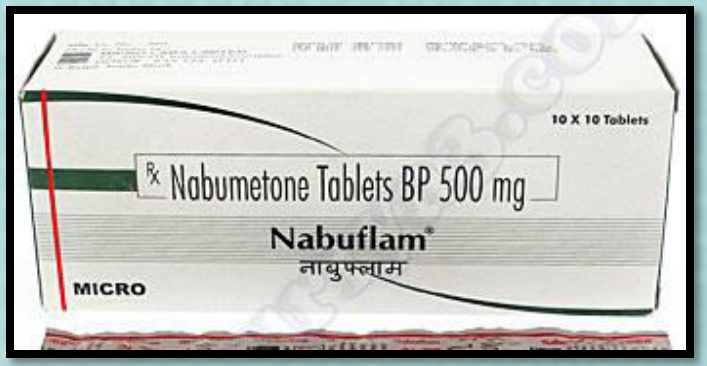
MELOXICAM:
Has COX2: COX 1 Selectivity ratio of 10:14.
Inhibits platelet TXA2 production

- Melflam, Muvik, 7.5mg, 15mg Tab.

Trade name

NABUMTONE: Prodrug-generates an active metabolite which is more potent COX 2 than Cox 1.
Analgesic, antipyretic, antiinflammmatory



- Nabuflam 500mg tab, OD

Trade name

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Selective COX-2 inhibitors

CELECOXIB:

Antiinflammatory, analgesic, antipyretic.

Platelet aggregation in response to collagen exposure remained intact.

TXB2 levels were not reduced.

- Slowly absorbed
- Plasma half life: 11hrs.

Pharmacokinetics

- Osteoarthritis, rheumatoid arthritis

Indications

- Abdominal pain, diarrhoea, still less than conventional NSAIDs

Adverse effects

- Celact, Revibra, Colcibra 100,200mg Cap.

Trade name

Essentials of medical pharmacology, K.D.Tripathi. 5th edition.

ROFECOXIB:

Most potent COX-2 selective inhibitor

- Well absorbed orally, Extensively metabolized.
- Plasma half life: 17hrs.

Pharmacokinetics

- Dental pain, Postoperative pain, musculoskeletal pain, Osteoarthritis, rheumatoid arthritis

Indications

- Headache, dizziness.

Adverse effects

- Rofact, Rofegesic, 12.5, 25 mg tab. OD

Trade name

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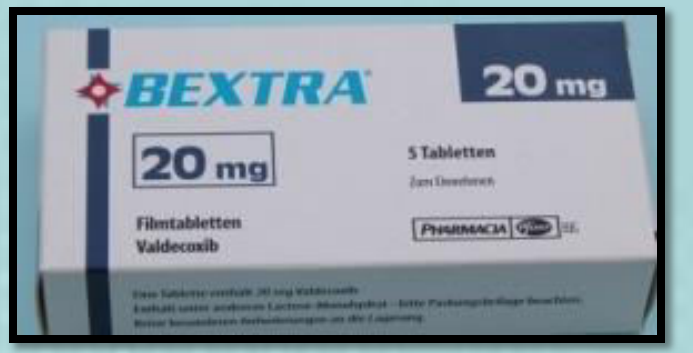
VALDECOXIB:



Recently marketed selective COX-2
Efficacy similar to rofecoxib.

- Well absorbed orally.
- Plasma half life: 8-11hrs.

Pharmacokinetics

- Postoperative pain, Osteoarthritis, rheumatoid arthritis

Indications

- Vorth, Valus 10mg tab.

Trade name

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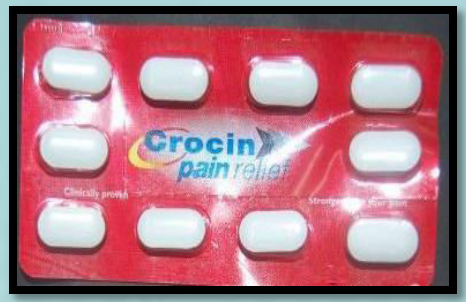
Para-amino phenol derivatives



- Well absorbed orally. Conjugated with glucuronic acid and sulfate and excreted rapidly in urine
- Plasma half life: 2-3hrs

Pharmacokinetics

- Dental pain, Postoperative pain, musculoskeletal pain, Osteoarthritis, rheumatoid arthritis

Indications

- Nausea, rashes occur rarely.

Adverse effects

- Crocin, 0.5, 1.0g tab, metacin 500mg, 500-1000mg- 3 to 4 times a day.
- Routes: Oral, IM, Rectal suppository

Trade name

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Acute Paracetamol Poisoning

Occurs in patients who have low hepatic glucuronide conjugation ability.

Early manifestation: Nausea, vomiting, abdominal pain, liver tenderness.

After 12-18hrs centrilobular hepatic necrosis may be accompanied by renal tubular necrosis and hypoglycemia, may progress to coma.

Jaundice starts after 2 days. Fulminating hepatic failure, death, if plasma levels is above 200µg/ml at 4hr.

Mechanism of toxicity: N-acetyl-p-benzoquinoneimine is a highly reactive minor metabolite of paracetamol which is detoxified by conjugation with glutathione.

Large doses of paracetamol, glucuronidation capacity is saturated.

Treatment: Induce vomiting, gastric lavage,

Specific: N-acetylcysteine (MUCOMIX 200mg/ml inj). 150mg/kg should be infuse I.V. over 15min, followed by I.V. over next 20 hrs.

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Benzoxazocine derivative

NEFOPAM:

Nonopiod analgesic which does not inhibit PG synthesis.

Contraindicated in epileptics

- Musculoskeletal pain

Indications

- Anti-cholinergic effects: Dry mouth, blurred vision, urinary retention,
- Sympathomemetic effects: Tachycardia,

Adverse effects

- Nefomax 30mg tab, 20mg in 1ml amp.

Trade name

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Topical NSAIDs

Indication: Osteoarthritis, sprains, sport injuries, tensinovitis, backache, spondylitis.
The drug will penetrate to the subjacent tissues attaining high concentrations in the affected muscles/ joints maintaining low blood levels.

Preparations:
Diclofenac gel 1%
Ibuprofen gel 10%
Naproxen gel 10%
Ketoprofn gel 2.5%
Flurbiprofen gel 5 %
Piroxicam gel 0.5%

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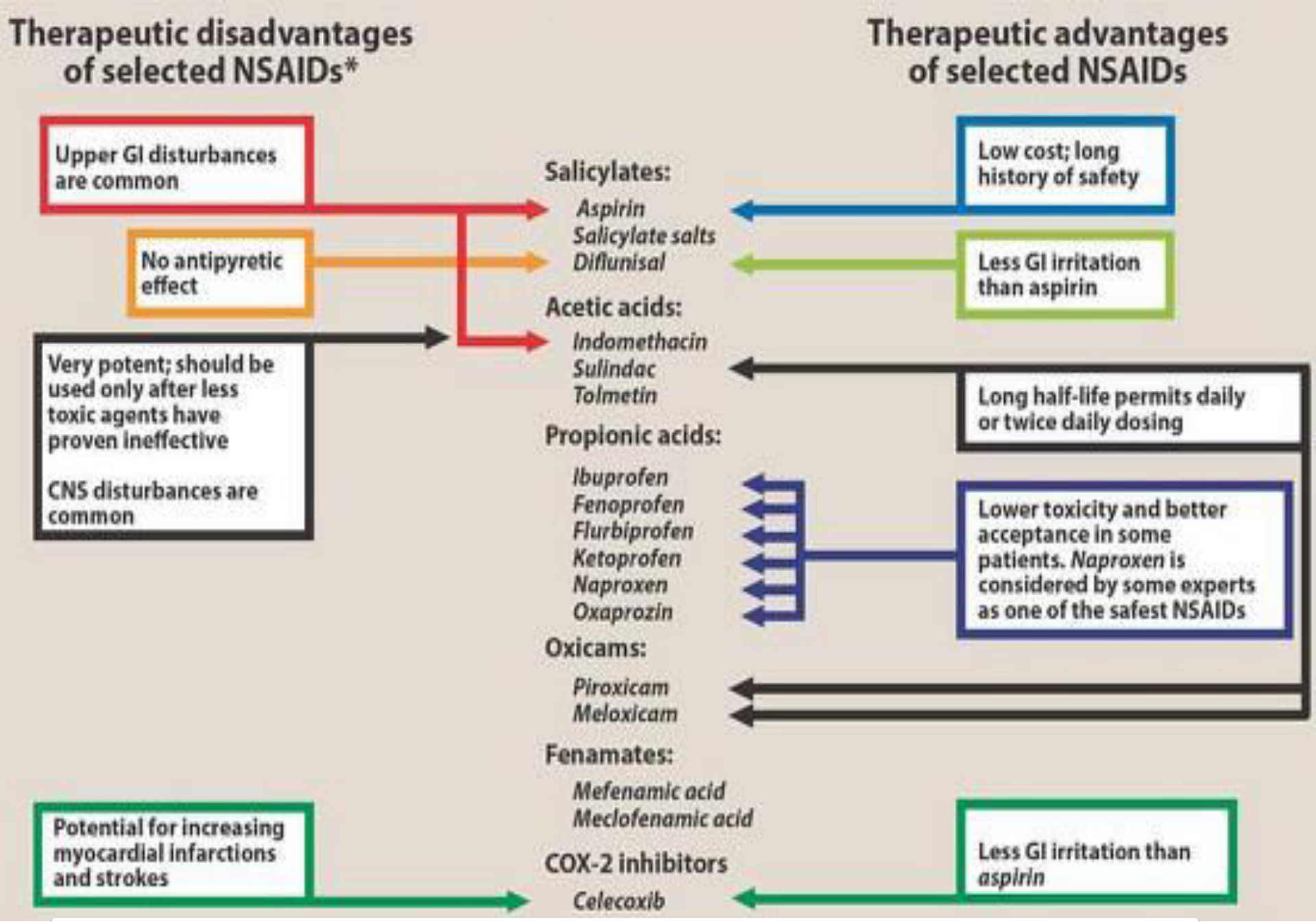
Summary

Condition	Drug
Mild to moderate pain with little inflammation	paracetamol or ibuprofen low dose
Acute musculoskeletal, injury associated inflammation	Propionic acid derivative, diclofenac, rofecoxib
Postoperative	ketorolac, nefopam
Gastric intolerance to conventional NSAIDs and predisposed patients	Rofecoxib, Celecoxib.
Patient with history of asthma, anaphylactoid reaction to aspirin/other NSAIDs	Nimesulide

Essentials of medical pharmacology, K.D.Tripathi. 5th edition.



Lippincott'sIllustratedReviews Pharmacology 4thEdition.

Nonsteroidal anti-inflammatory drug (NSAID) adverse effects	
Gastrointestinal	Gastrointestinal ulceration and intolerance
Renal	Inhibition of prostaglandin-mediated renal function
Hemostatic	Blockade of platelet function
Pregnancy	Inhibition of uterine motility may prolong gestation
Immune	Hypersensitivity reactions
Cardiovascular	Increased blood pressure
Interactions	Warfarin: NSAIDs bind to plasma proteins and can displace from binding site

Pain Res Manage: Lynch and Watson;2006 Vol 11 No 1

Conclusion

The goals of management of orofacial pain include reducing or eliminating pain, halting the disease process when possible, normalizing function, improving quality of life, and reducing the need for long-term care.

A multidisciplinary approach to pain management has been demonstrated to be most efficacious

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Thank you!