Pain Assessment and Management

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Determination of Pain

Pain Management Approach

NEUROANATOMY AND PATHOPHYSIOLOGY OF PAIN

- OPIOID ANALGESICS
- LOCAL ANESTHETICS
- NONOPIOID MEDIATED ANALGESIA

Perioperative pain control considerations

Pain Management Policy

- All patients must have effective pain management
  - Appropriate screening and pain assessment
  - Documentation
  - Care and treatment
  - Pain education
- Patient self report of pain must be source of assessment whenever possible
  - Patient’s acceptable level of pain must guide treatment

Determination of Pain

Patient self report of pain must be source of assessment whenever possible

- Patient's acceptable level of pain must guide treatment

If the patient is unable to self report, assessment strategies should include

- Observable behaviors (facial expressions, body movements, crying)
- physiological measures (heart rate and blood pressure).

What is Pain?

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

International Association for the Study of Pain (Merskey, 1979)

Pain is always subjective.

The patient's self-report of pain is the single most reliable indicator of pain.

The clinician must accept the patient's self report of pain.

Acute Pain

Acute pain presents most often with a clear cause, relatively brief in duration and subsides as healing takes place.

Acute pain is often accompanied by observable objective signs of pain

- *increased pulse rate*
- *increased blood pressure*
- *Non-verbal signs and symptoms such as facial expressions and tense muscles.*

Chronic Pain

Pain that is persistent and recurrent.

When pain persists, it serves no useful purpose and may dramatically decrease the quality of life and function.

Chronic pain rarely has any observable or behavioral signs although persons may appear anxious or depressed.

Pain Assessment

Initial Pain Assessment should include:

- Location(s)
- Intensity
- Sensory quality
- Alleviating and aggravating factors

Any new onset of pain requires a new comprehensive pain assessment.

Pain Management Approach

Should be interdisciplinary and multimodal.

Care is individualized and may depend on:

- Pain source and intensity
- Patient's age
- Developmental, physical, emotional and cognitive status
- Cultural beliefs
- Treatment preferences
- Concurrent medical conditions

Multimodal Analgesia

This term describes the use of multiple modalities that are used to provide pain relief with various parts of the pain pathway targeted.

Decreased dependence on single modality agents decreases the risk of side effects.

May include

- *Pharmacological (opioids, NSAIDS, gabapentanoids)*
- *Relaxation techniques (biofeedback, deep breathing)*
- *Regional analgesia (nerve blocks, epidural catheters)*

Treatments May Include

NON-PHARMACOLOGIC METHODS

- Heat/cold
- Relaxation
- Distraction
- Acupressure/acupuncture

PHARMACOLOGIC METHODS

NSAIDS

Anti-seizure medications

Anti-depressants

Opioid analgesics

Local anesthetics

Neurolytics

Acute vs. Chronic Pain Management

ACUTE PAIN

Most often treated with:

- NSAIDS
- Opioids
- Local anesthetics
- Splinting
- Positioning changes
- Ice

CHRONIC PAIN

Most often treated with:

- Anti-seizure medications
- Anti-depressant medications
- NSAIDS
- Implantable devices
- Psychological therapy
- Acupuncture

When all else fails and benefits outweigh risks

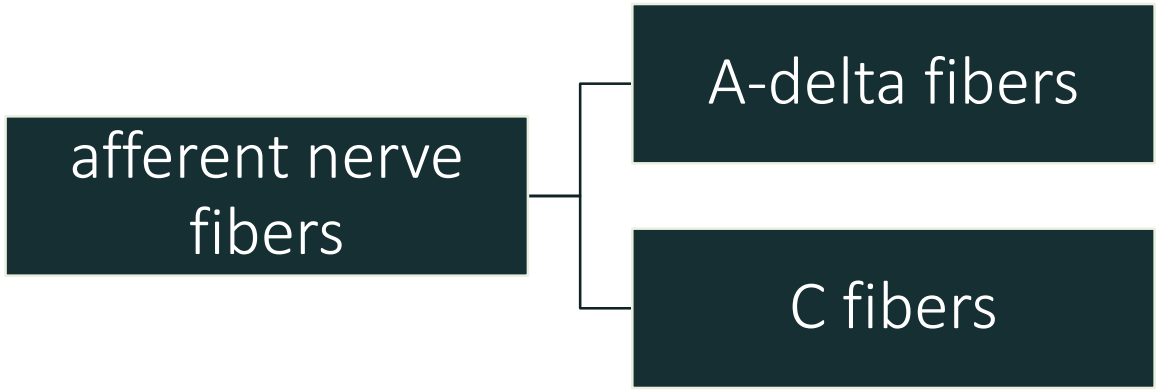
- Opioids

Management of acute pain

NEUROANATOMY AND PATHOPHYSIOLOGY OF PAIN

Nociception is the electrical transmission of a noxious stimulus from a site of injury to higher brain centers.

Following tissue damage outside of the craniofacial region, free nerve endings present in peripheral tissue are stimulated by mechanical, thermal, and chemical means.



NEUROANATOMY AND PATHOPHYSIOLOGY OF PAIN

A-delta fibers:

are lightly myelinated

have a larger diameter

are fast conducting

They are primarily activated by mechanical stimuli and are characterized by **sharp**, **stabbing**, and **shooting** sensations.

NEUROANATOMY AND PATHOPHYSIOLOGY OF PAIN

C fibers:

have a smaller diameter

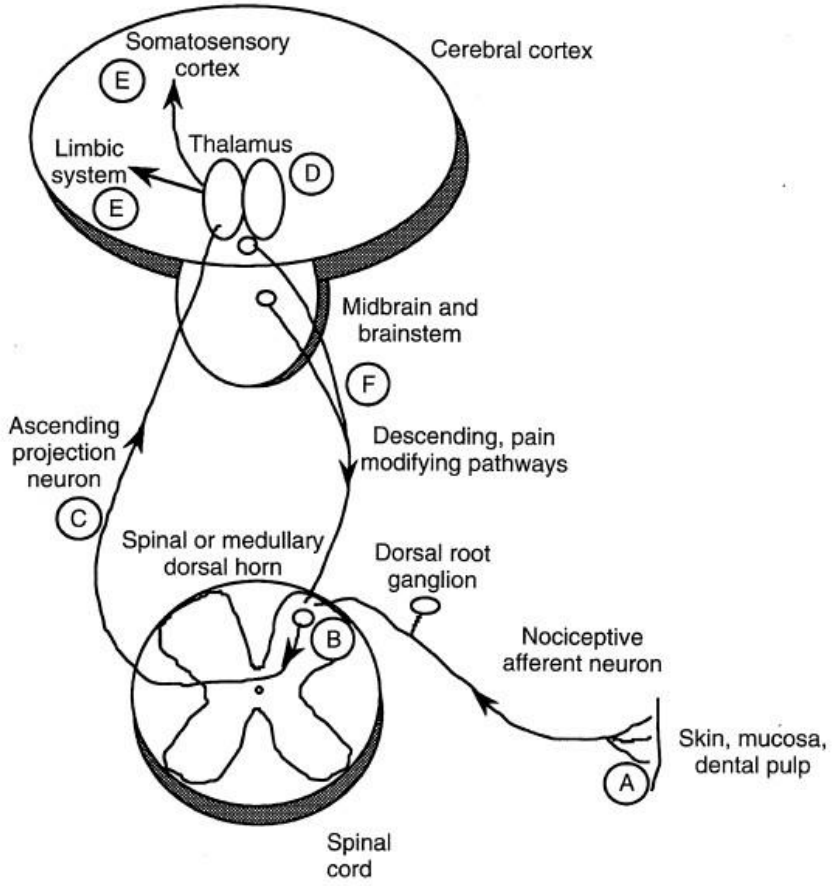
are unmyelinated

transmit impulses at a slower rate

They are activated by mechanical, chemical, and thermal stimuli, and are associated with **dull** pains.

NEUROANATOMY AND PATHOPHYSIOLOGY OF PAIN

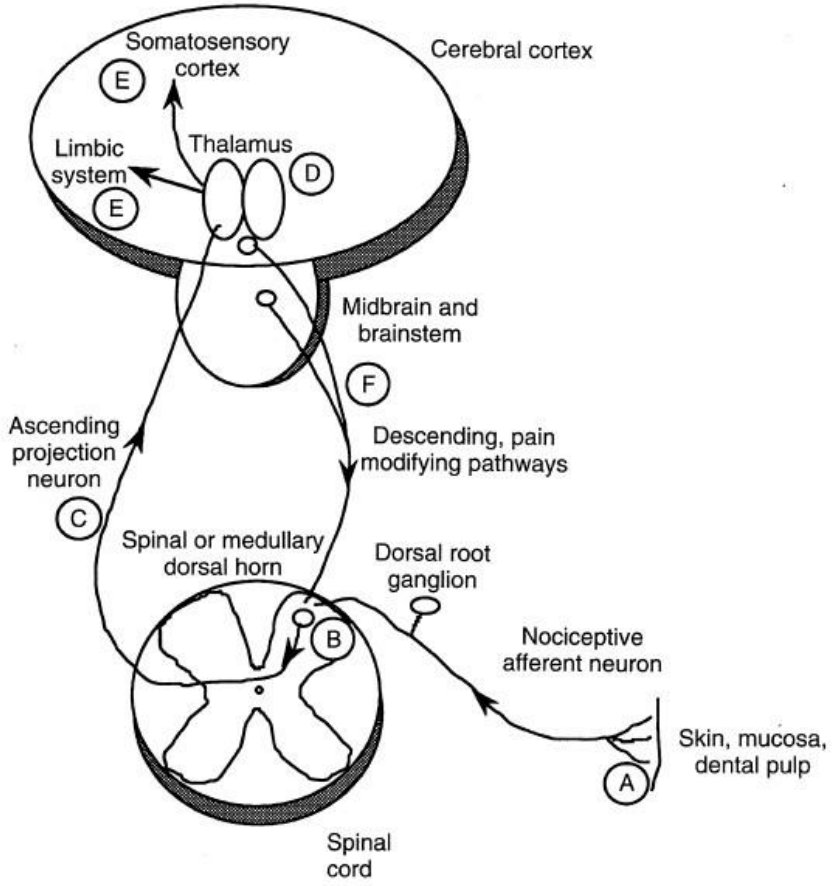
After synapsing with second order neurons in the dorsal horn, the impulse then crosses to the contralateral ascending spinothalamic tract.



NEUROANATOMY AND PATHOPHYSIOLOGY OF PAIN

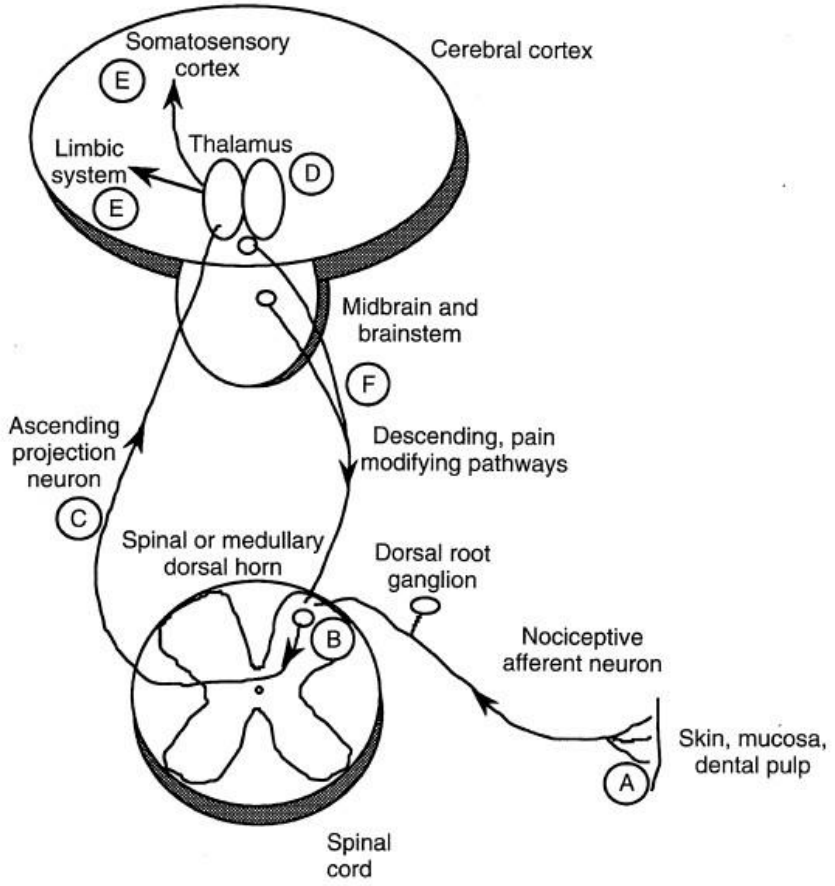
Then it travels to the thalamus.

From the thalamus, the impulse travels to somatosensory cortex and limbic system.



NEUROANATOMY AND PATHOPHYSIOLOGY OF PAIN

The transmission of this impulse may also be affected by descending pain-modifying pathways from the cerebral cortex, midbrain, and brainstem.



NEUROANATOMY AND PATHOPHYSIOLOGY OF PAIN

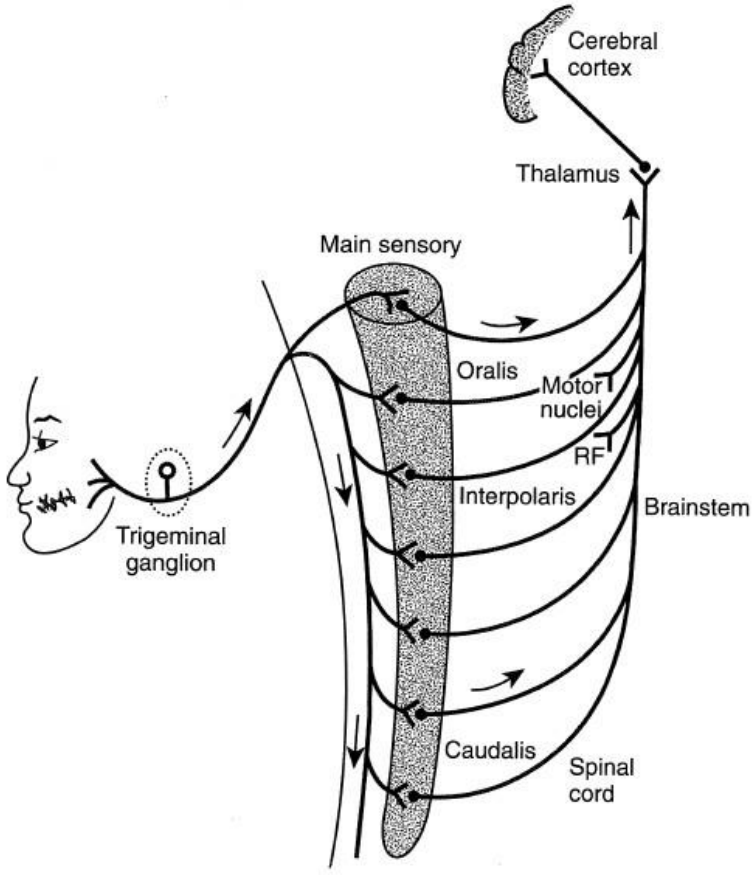
The main sensory nerve of the orofacial region is the trigeminal nerve (cranial nerve V).

There are also some tissues in the craniofacial region receiving afferent sensory innervation from other cranial nerves (cranial nerves VII, IX, X, and XII) and branches of the upper cervical spinal nerves (C1, C2, C3).

NEUROANATOMY AND PATHOPHYSIOLOGY OF PAIN

After synapsing in the VBSNC, the impulse travels to the thalamus where it can be relayed to somatosensory cortex and the limbic system.

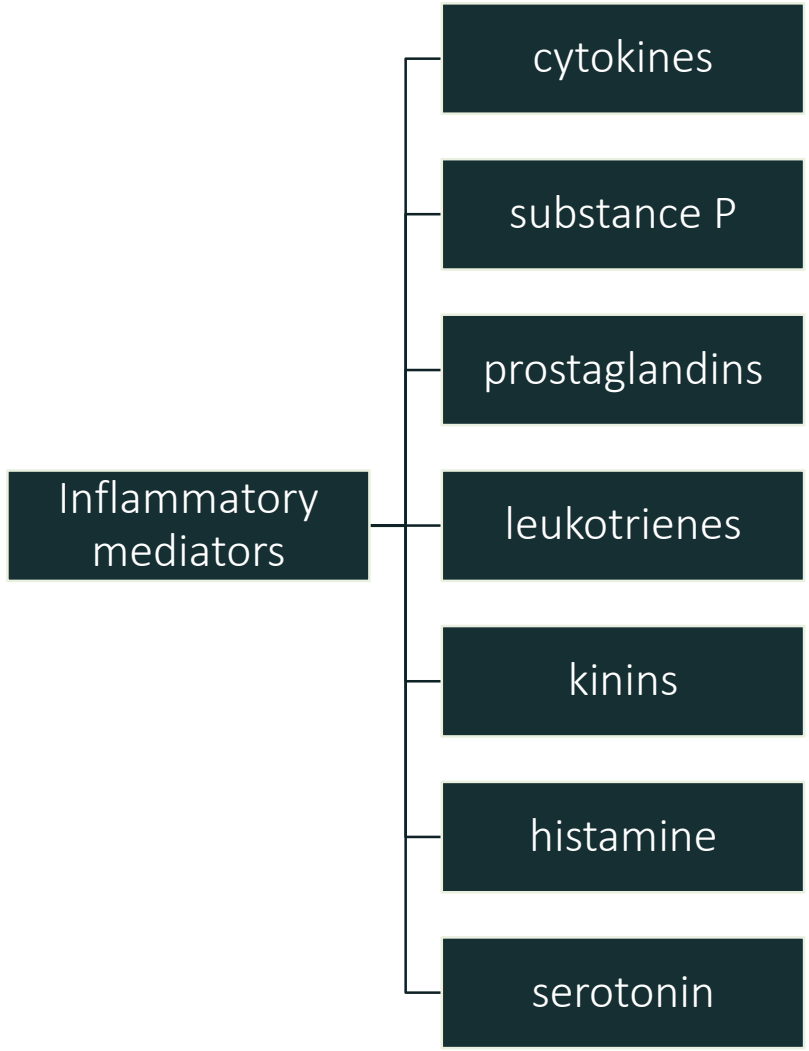
Descending modifying pathways are also present that can affect the transmission and sensation of pain.



NEUROANATOMY AND PATHOPHYSIOLOGY OF PAIN

Inflammation plays an important role in the development of postoperative pain.

As cells are disrupted, inflammatory mediators and metabolites are released into surrounding tissue beds.



NEUROANATOMY AND PATHOPHYSIOLOGY OF PAIN

Some of these factors can directly stimulate a nerve impulse in afferent pain fibers.

Prostaglandins, serve to sensitize nociceptors by lowering their activation thresholds. This leads to the hyperalgesia found in surgically altered tissue.

OPIOID ANALGESICS

The natural by-products of **opium**, including morphine, codeine, and their semisynthetic derivatives, are known as opiates.

The activity of all opioids is related to their binding to a special set of receptors.

These receptors have been categorized into three groups: **mu**, **kappa**, and **delta**.

OPIOID ANALGESICS

Agonists, such as morphine and codeine, elicit their analgesic effects by binding **mu** and **kappa** receptors.

Antagonists bind opioid receptors, but do not stimulate them.

Naloxone, a common narcotic reversal agent, is an opioid antagonist. Its ability to reverse the effects of opioid agonists is based on its higher binding affinity for opioid receptors.

Agonist-antagonist drugs act as agonists at one type of receptor and antagonists at others.

Pentazocine acts as an **agonist** at **kappa** receptors and an **antagonist** at **mu** receptors.

OPIOID ANALGESICS

Their analgesic effect is generated mainly by the inhibition of nociceptive impulses in the CNS.

They have been found at various levels of the ascending pain pathway.

They have also been found in the midbrain and medulla, where they activate descending inhibitory pathways.

OPIOID ANALGESICS

Binding at opioid receptors may lead to one of several biochemical results:

1) decrease in calcium influx at afferent nerve terminals leading to decreased presynaptic neurotransmitter release

2) increased potassium efflux leading to hyperpolarization of postsynaptic neurons and inhibition of impulse propagation

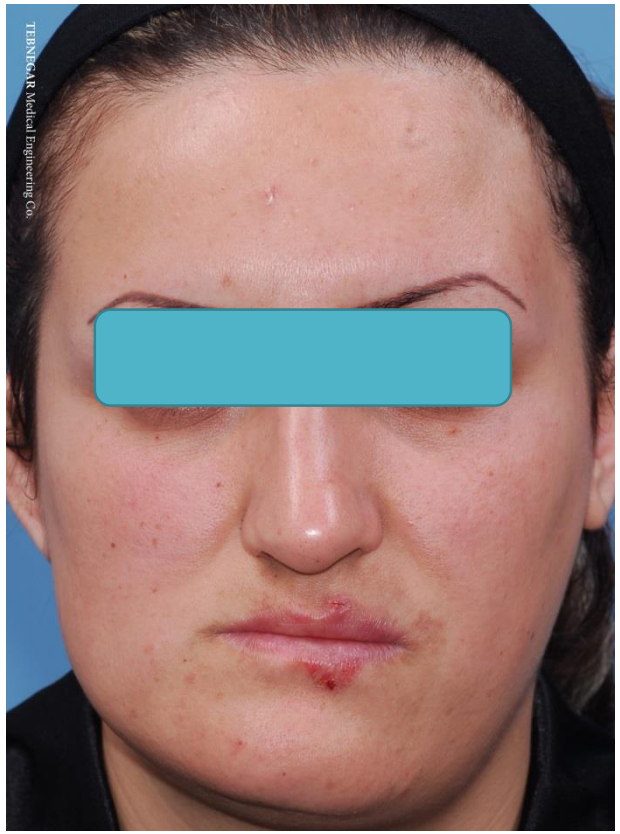
3) inhibition of GABAergic transmission in the brain-stem leading to the excitation of descending modulating circuits.

OPIOID ANALGESICS

In addition to analgesia, activation of opioid receptors can lead to several undesirable effects.

BOX 6-1 **Opioid Adverse Side Effects**

Respiratory depression
Nausea and vomiting
Mental clouding
Sedation
Euphoria
Constipation
Hypotension
Urinary retention
Pruritus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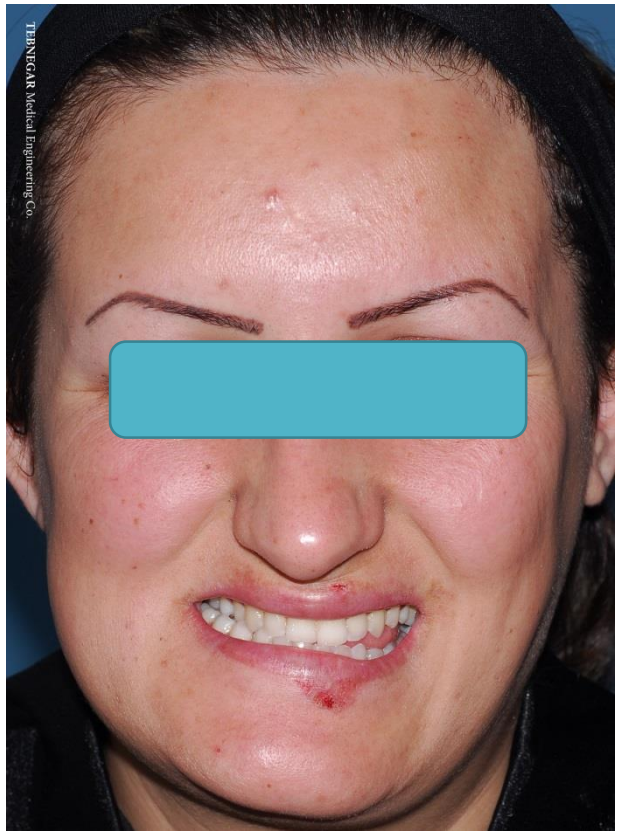
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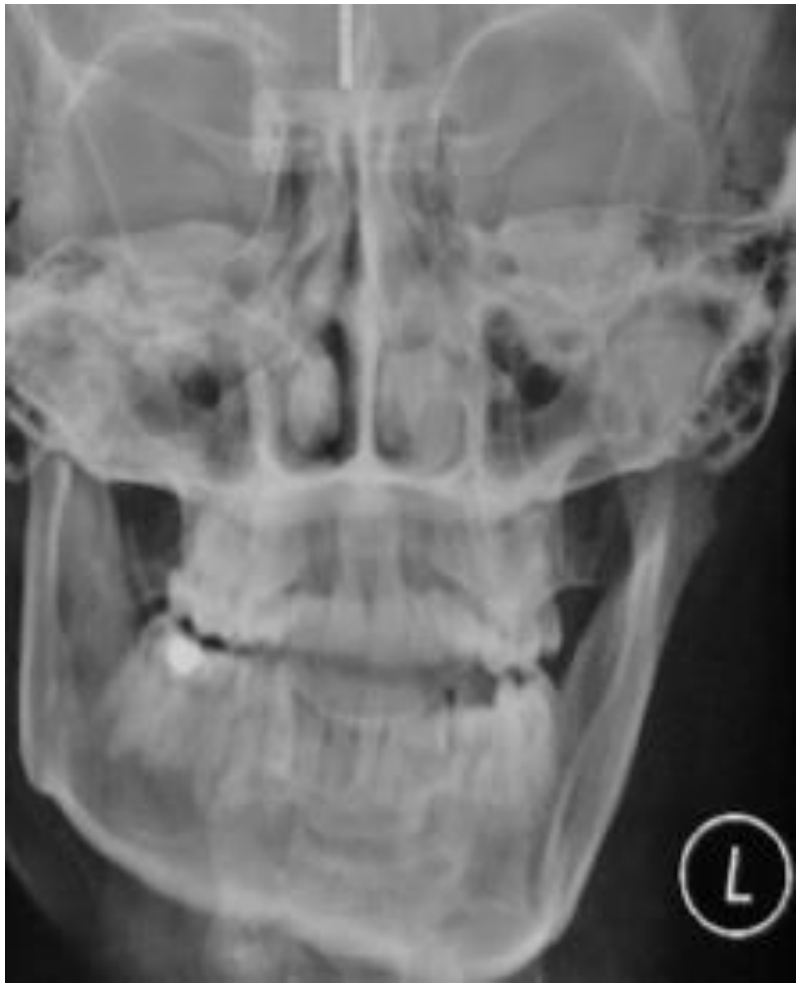
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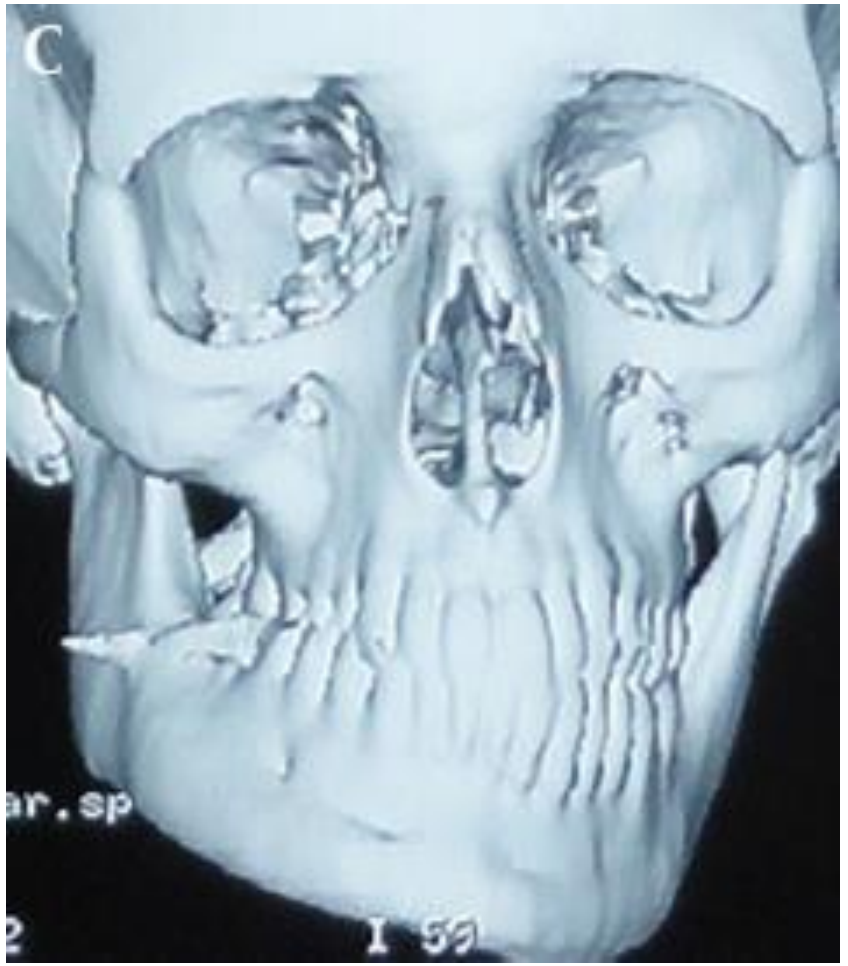
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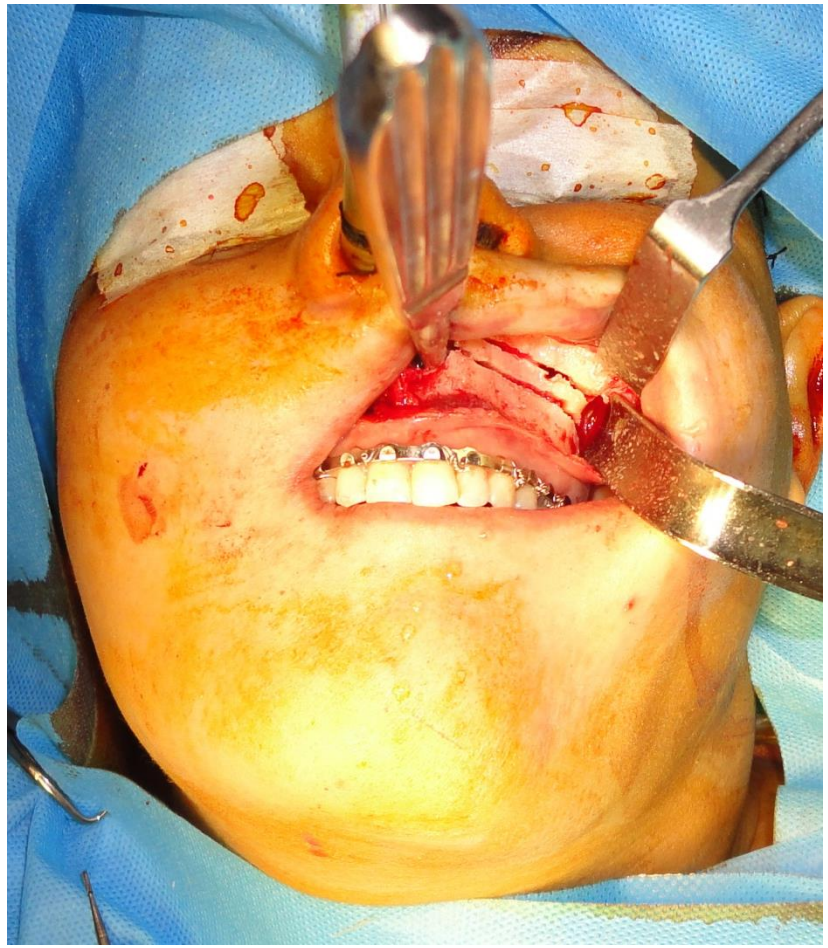

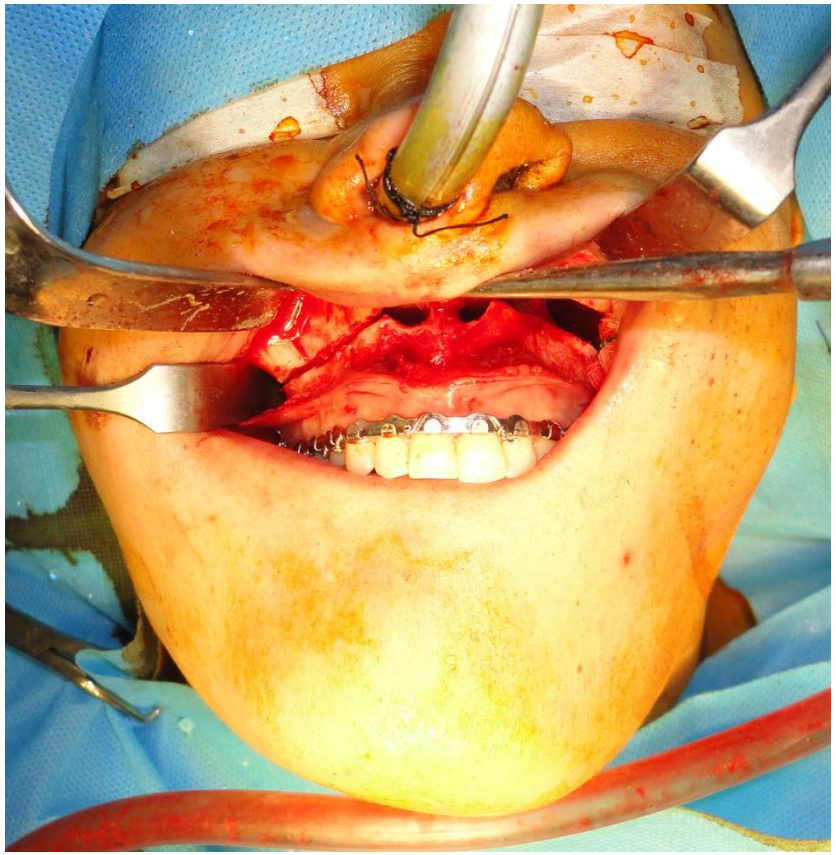

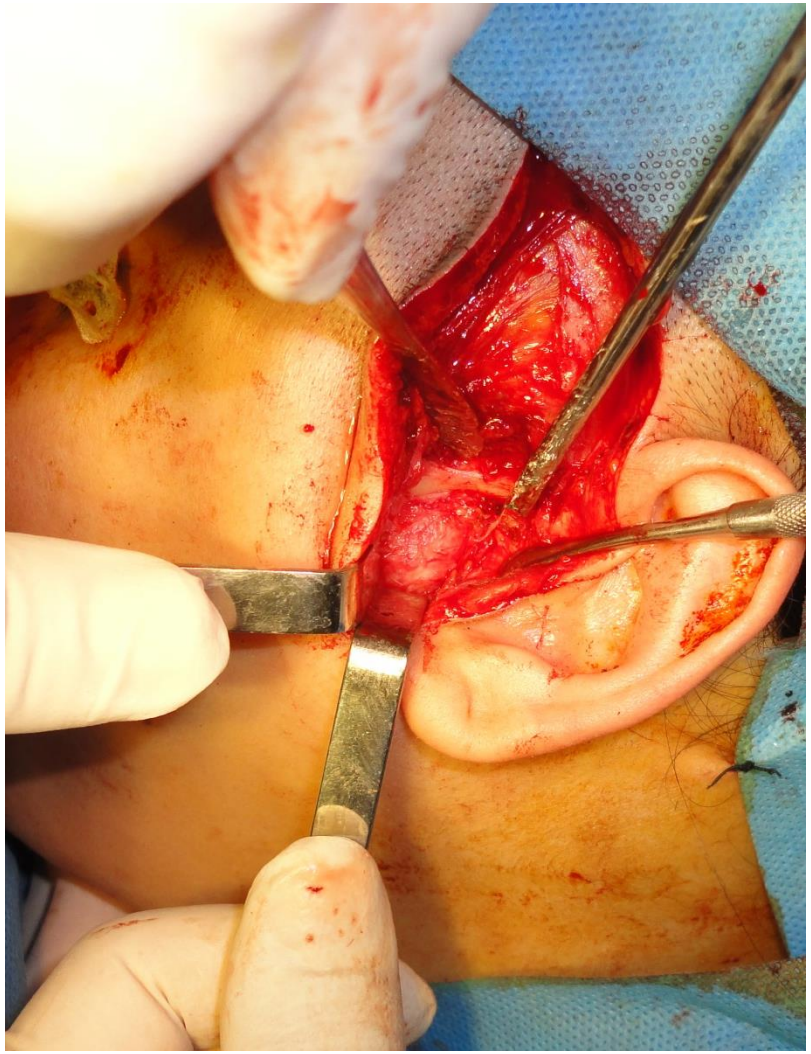

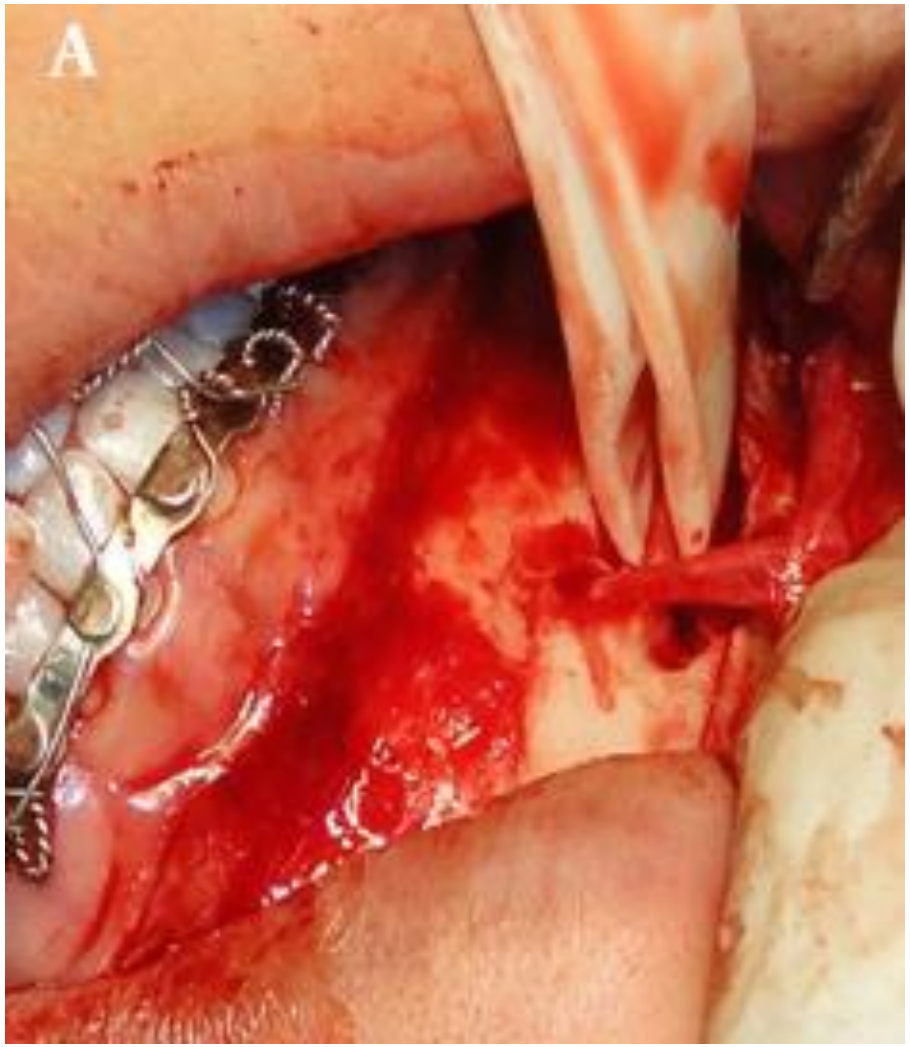
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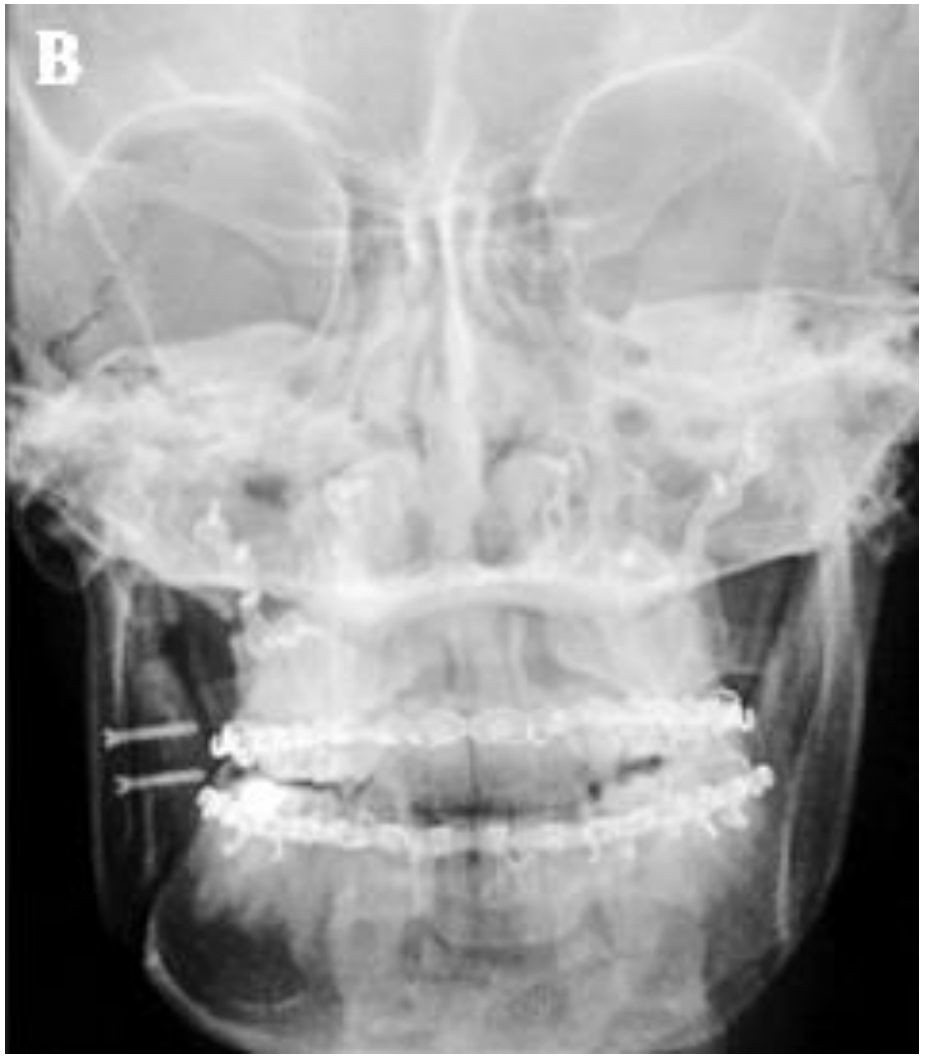
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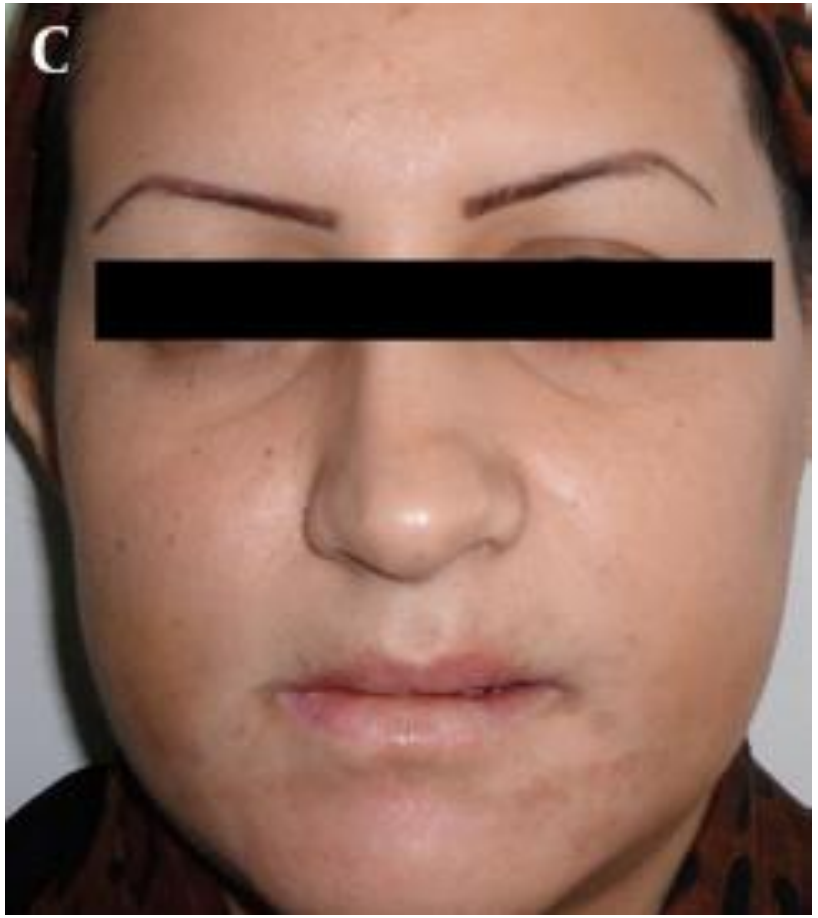
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OPIOID ANALGESICS

The activation of mu receptors in respiratory centers of the brainstem leads to **respiratory depression**.

This response is dose dependent and causes reductions in respiratory rate and minute volume.

This is the **most serious side effect** of opioid administration, and overdose can lead to respiratory arrest and death.

OPIOID ANALGESICS

One of the more common unwanted effects of opioid use is **nausea** and **vomiting**.

This response is produced by the activation of neurons in the chemoreceptor trigger zone of the medulla.

OPIOID ANALGESICS

Other CNS effects include mental clouding, sedation, and euphoria.

In the gastrointestinal (GI) system, opioids decrease gastric and intestinal motility leading to constipation.

OPIOID ANALGESICS

Opioids cause arteriolar **vasodilation** and can result in orthostatic hypotension.

However, the decrease in cardiac demand caused by opioids can also be a benefit in patients experiencing angina or myocardial infarction.

OPIOID ANALGESICS

Opioids also cause an increase in smooth muscle tone of the bladder and urinary sphincter leading to **urinary retention**.

Finally, **itching** may occur during opioid use as a result of the release of histamine from mast cells.

OPIOID ANALGESICS

Oral administration is the most common route of administration.

Opioids absorbed in the GI tract are subject to first-pass hepatic metabolism. This limits their efficacy and increases inter-patient variability when administered orally.

OPIOID ANALGESICS

Morphine is considered the prototype for all opioids.

Administration of morphine is indicated for severe pain, and thus its use in oral and maxillofacial surgery is limited.

Opioids have no ceiling effect for analgesia. Their administration is limited by the undesirable effects that occur as doses are increased.

OPIOID ANALGESICS

Fentanyl is a fast-acting opioid that is approximately 100 times more potent than morphine.

It is indicated for severe pain and is a common agent used for conscious sedation in oral and maxillofacial surgical procedures.

OPIOID ANALGESICS

Meperidine is 10 times less potent than morphine, has a slower onset, and lasts about twice as long as morphine and fentanyl.

Its use has generally fallen out of favor because of its undesirable effects (tachycardia, decreased myocardial contractility, mydriasis).

OPIOID ANALGESICS

Normeperidine is a neurotoxic metabolite of meperidine.

It has been shown to cause dysphoria, tremors, and generalized seizures.

In addition, use of meperidine with monoamine oxidase inhibitors (MAOIs) can cause **hypertensive** crisis, **hyperpyrexia**, and **cardiovascular** collapse.

OPIOID ANALGESICS

TABLE 3-6 Antidepressant Medication Classification

Antidepressant Classification	Generic Name	Trade Name
Monoamine oxidase inhibitors (MAOIs)	Isocarboxazid	Marplan
	Phenelzine	Nardil
	Selegiline transcutaneous	Emsam
	Tranylcypromine	Parnate
Tricyclic antidepressant (TCAs)	Amitriptyline	Elavil
	Clomipramine	Anafranil
	Desipramine	Norpramin
	Doxepin	Sinequan
	Imipramine	Tofranil
	Nortriptyline	Aventyl HCL, Pamelor
	Protriptyline	Vivactil
	Trimipramine	Surmontil
Selective serotonin reuptake inhibitors (SSRIs)	Citalopram	Celexa
	Escitalopram	Lexapro
	Fluoxetine	Prozac, Sarafem
	Fluvoxamine	Fluvox
	Paroxetine	Paxil, Pexeva
	Sertraline	Zoloft

OPIOID ANALGESICS

Codeine is another naturally occurring alkaloid that is structurally similar to morphine. It is typically given in the oral form. Codeine is a weak mu agonist.

A small percentage of codeine is converted to morphine after administration.

In the oral form, codeine is about four times less potent than morphine.

Doses greater than 60 mg are not recommended because the side effects above this dose outweigh the analgesic benefits.

OPIOID ANALGESICS

Hydrocodone and **oxycodone** are semi-synthetic opioids that are similar to morphine and codeine.

These drugs are available only in the oral formulation and are about 6.5 and 10 times more potent than codeine, respectively.

OPIOID ANALGESICS

Propoxyphene is structurally similar to methadone, a compound typically prescribed to treat heroin dependence and addiction.

It has about two-thirds of the potency of codeine.

It also causes cardiotoxicity, pulmonary edema, and arrythmias.

It is therefore not generally considered a first-line choice for opioid analgesia. It may be of some use in patients that exhibit sensitivities to those medications.

OPIOID ANALGESICS

Pentazocine is an opioid agonist-antagonist.

It exhibits mu antagonist activity and kappa agonist activity.

It has less respiratory depressive effects than the pure opioid agonists.

Because it antagonizes mu receptors, it can precipitate opioid withdrawal symptoms in patients concurrently taking mu agonists.

OPIOID ANALGESICS

Tramadol is an analgesic drug marketed for treatment of moderate to severe pain.

It is a weak mu agonist.

Respiratory effects and abuse potential are minimal.

The efficacy of tramadol appears to be comparable with codeine and its oral combinations.

It has been associated with side effects such as headache, nausea, vomiting, somnolence, and seizures.

OPIOID ANALGESICS

TABLE 6-1 Commonly Used Opioid Analgesics

Analgesic	Dose (mg)	Dosing Interval (hr)	Comments
AGONIST			
Morphine	10-30	4	Standard analgesic dose used for potency comparisons
Codeine	60	3-4	Can be administered as a single agent or in combination with acetaminophen or aspirin
Hydrocodone	5-10	4-5	Only available in a combination formulation with either acetaminophen, aspirin, or ibuprofen
Oxycodone	5-10	4-5	Only available in a combination formulation with either acetaminophen or aspirin
Propoxyphene	65-130	4-6	Weak analgesic properties
MIXED AGONIST-ANTAGONIST			
Pentazocine	50-100	4-6	Will precipitate an abstinence syndrome in patients dependent on opioids
OTHER			
Tramadol	50-100	4-6	Not available in a combination formulation; although currently unscheduled by the Drug Enforcement Administration, abuse potential exists

LOCAL ANESTHETICS

Local anesthesia is a loss of sensation in an area of the body caused by the depression of nerve ending excitation or the inhibition of nerve impulse conduction.

Amide local anesthetics are primarily metabolized in the liver.

Caution should be exercised when administering these drugs to patients with liver disease.

LOCAL ANESTHETICS

Lidocaine and mepivacaine, the most commonly used local anesthetics, are short-acting.

Used in conjunction with epinephrine, they block conduction for 3 to 5 hours.

Their use for postoperative pain is therefore limited.

The long-acting amide local anesthetics include bupivacaine and etidocaine.

The dental formulations of these drugs can block conduction for up to 8 to 12 hours.

LOCAL ANESTHETICS

Whenever using local anesthetics, the clinician should be cognizant of the maximum doses for each agent.

CNS and cardiovascular toxicity can result from overdose.

CNS depression from local anesthetic overdose typically manifests with slurred speech, light-headed-ness, headache, blurred vision, and drowsiness.

Generalized tonic-clonic seizures can also occur.

LOCAL ANESTHETICS

In the **cardiovascular system**, initial signs may include elevations in heart rate, blood pressure, and respiratory rate.

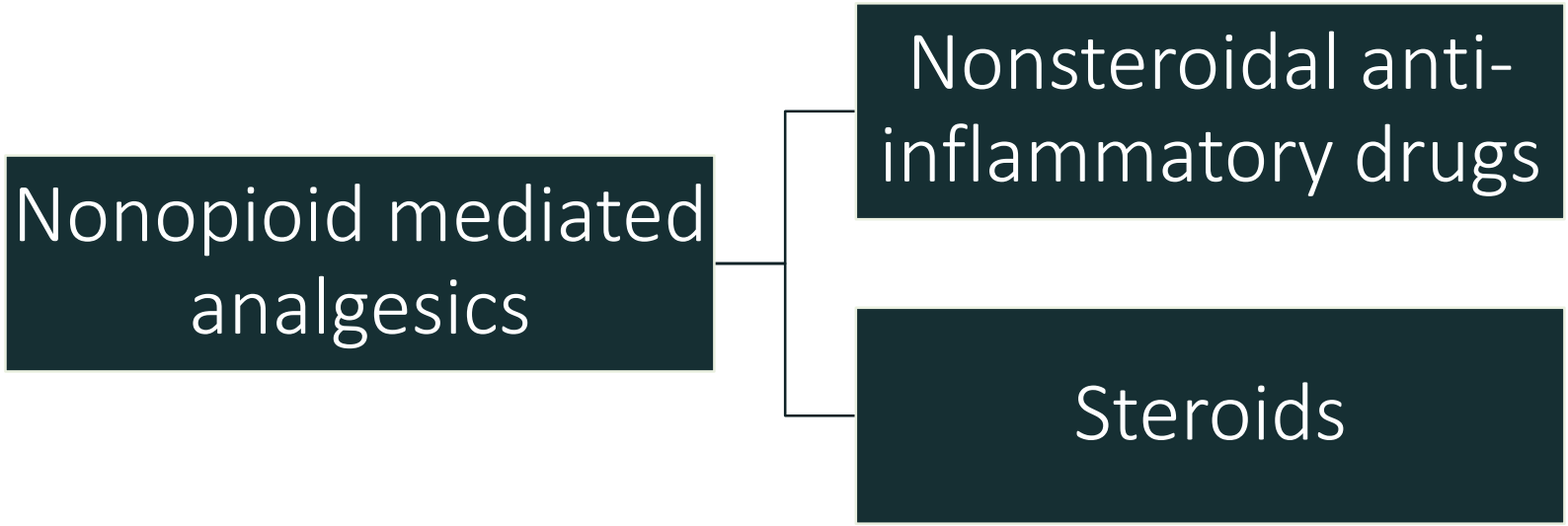
As levels of systemic local anesthetic increase, myocardial contractility is decreased, heart rate, blood pressure, and respiratory rate become depressed, and circulatory collapse can occur.

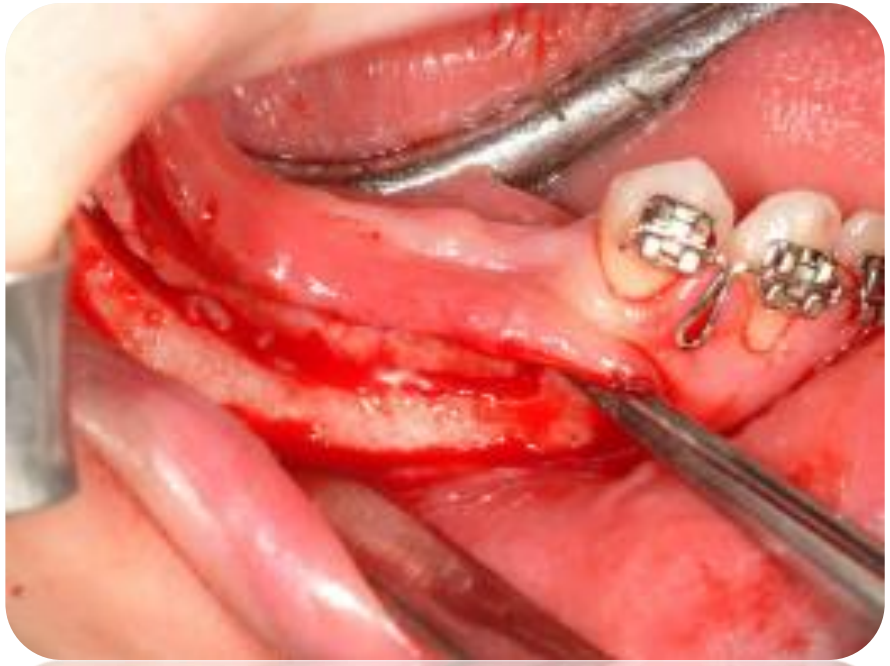
NONOPIOID MEDIATED ANALGESIA

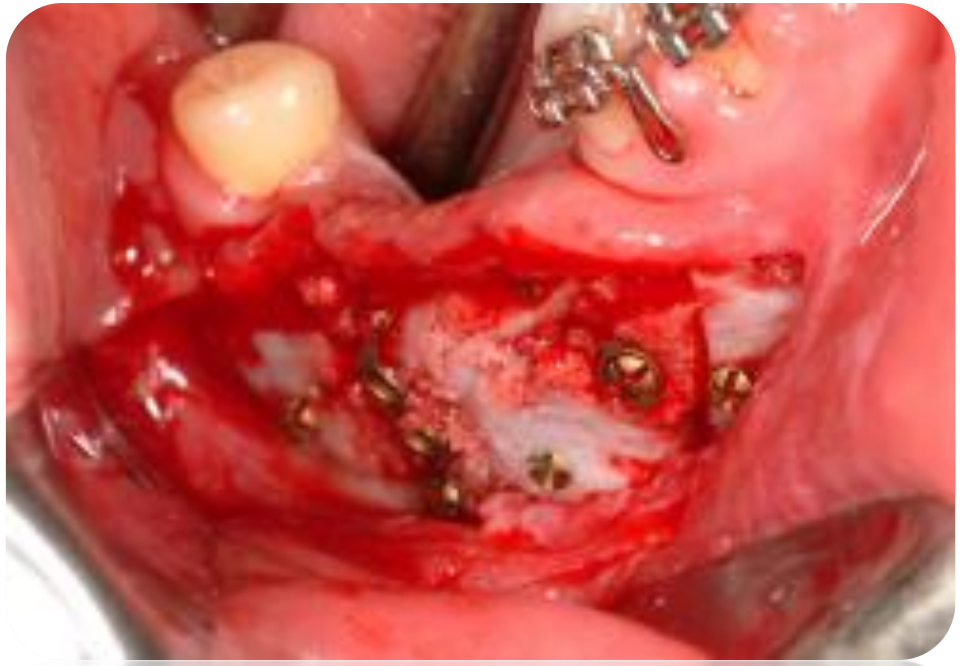
The **inflammatory phase** of wound healing consists of release of prostaglandins, histamine, kinins, leukotrienes, and substance P.

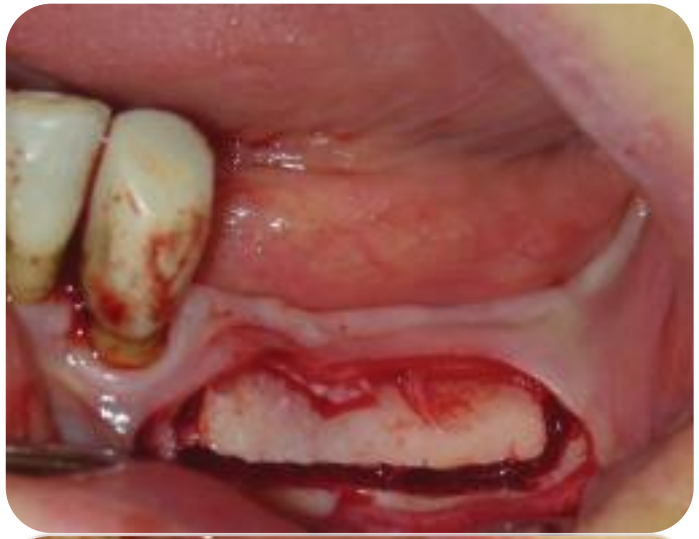
Prostaglandin synthesis at the site of tissue injury enhances pain by increasing the sensitization and reducing the activation threshold of nociceptors, which is called **hyper-algesic** effect.

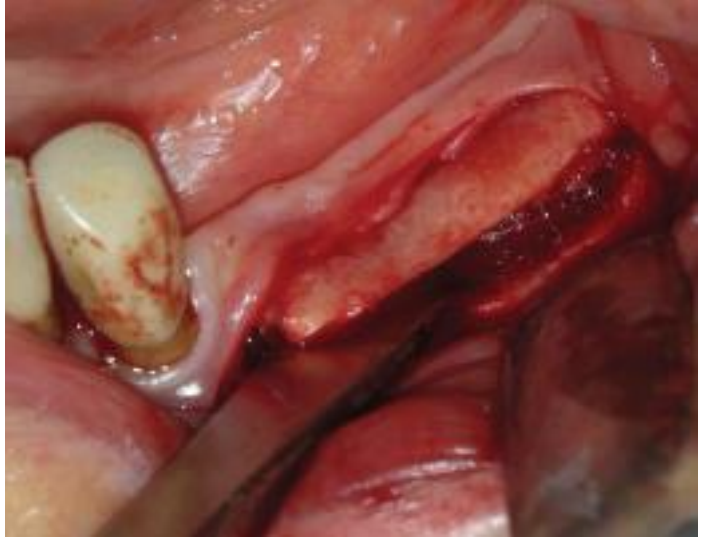
NONOPIOID MEDIATED ANALGESIA

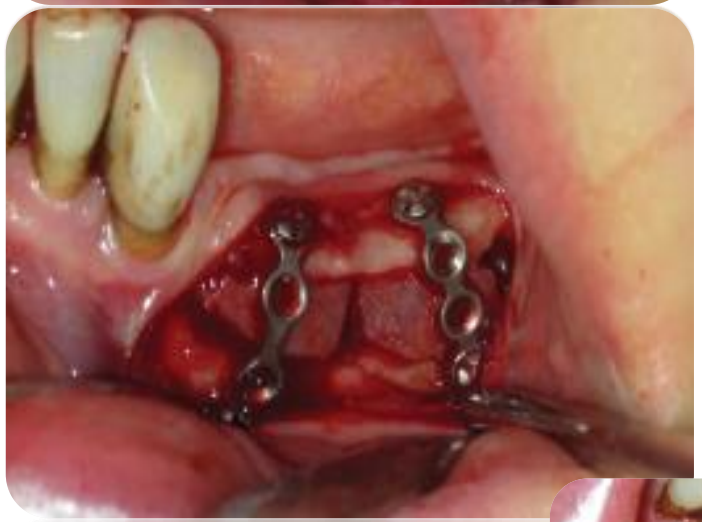


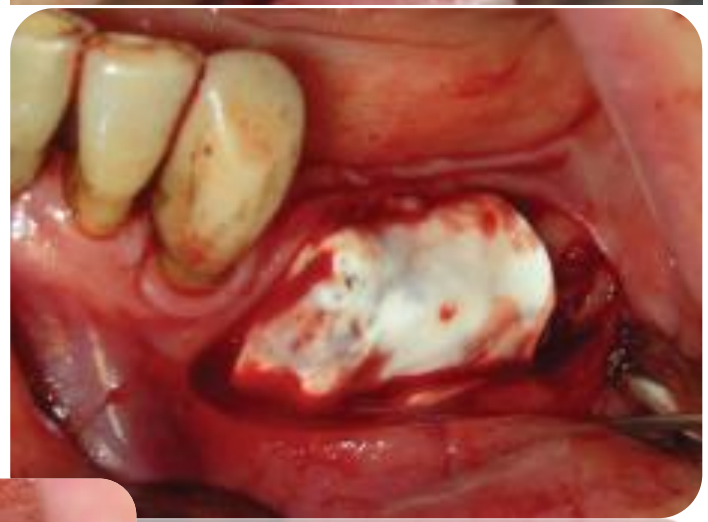


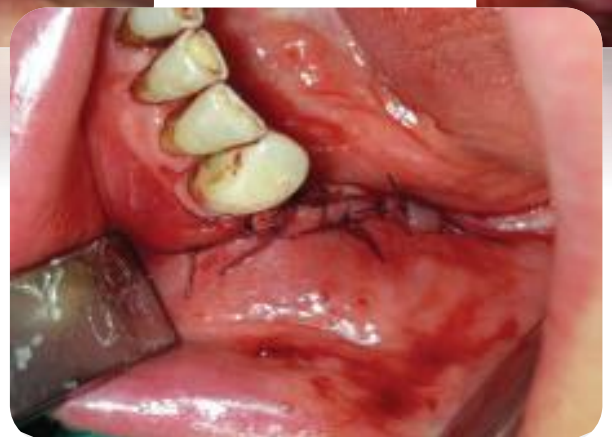


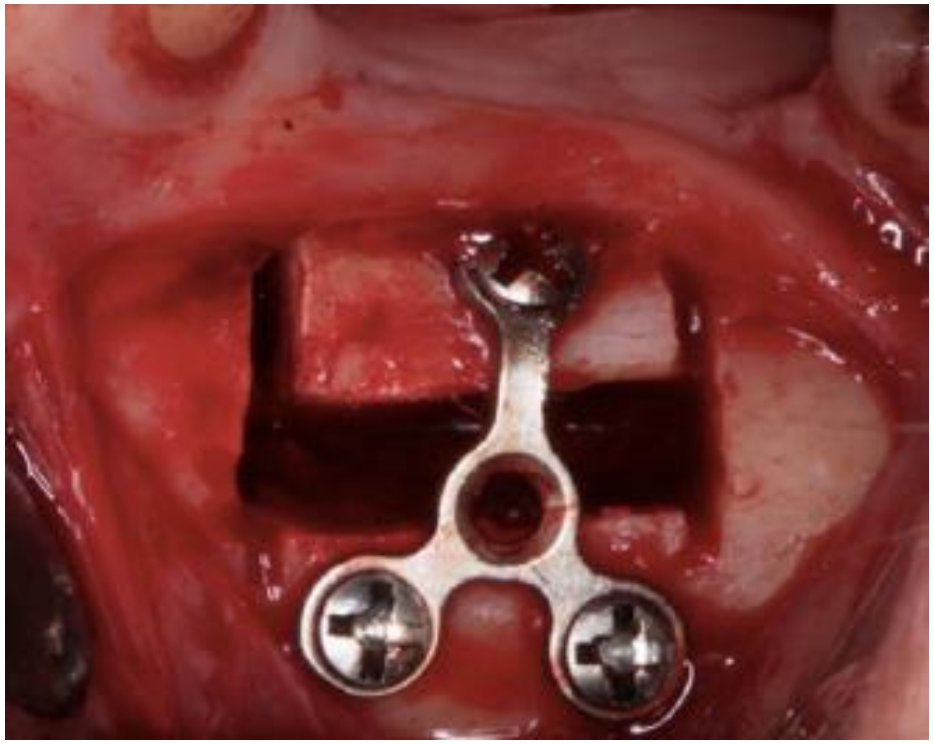


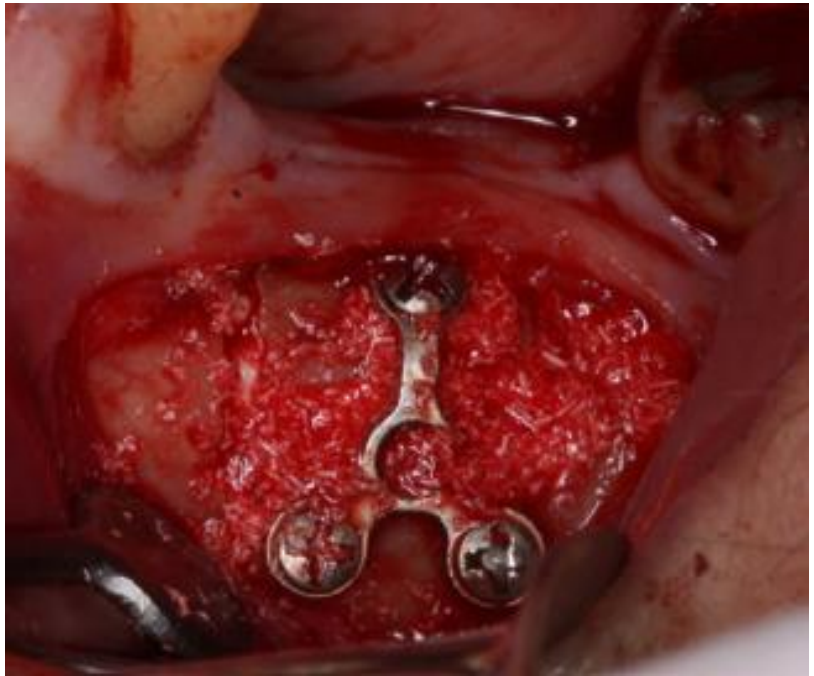


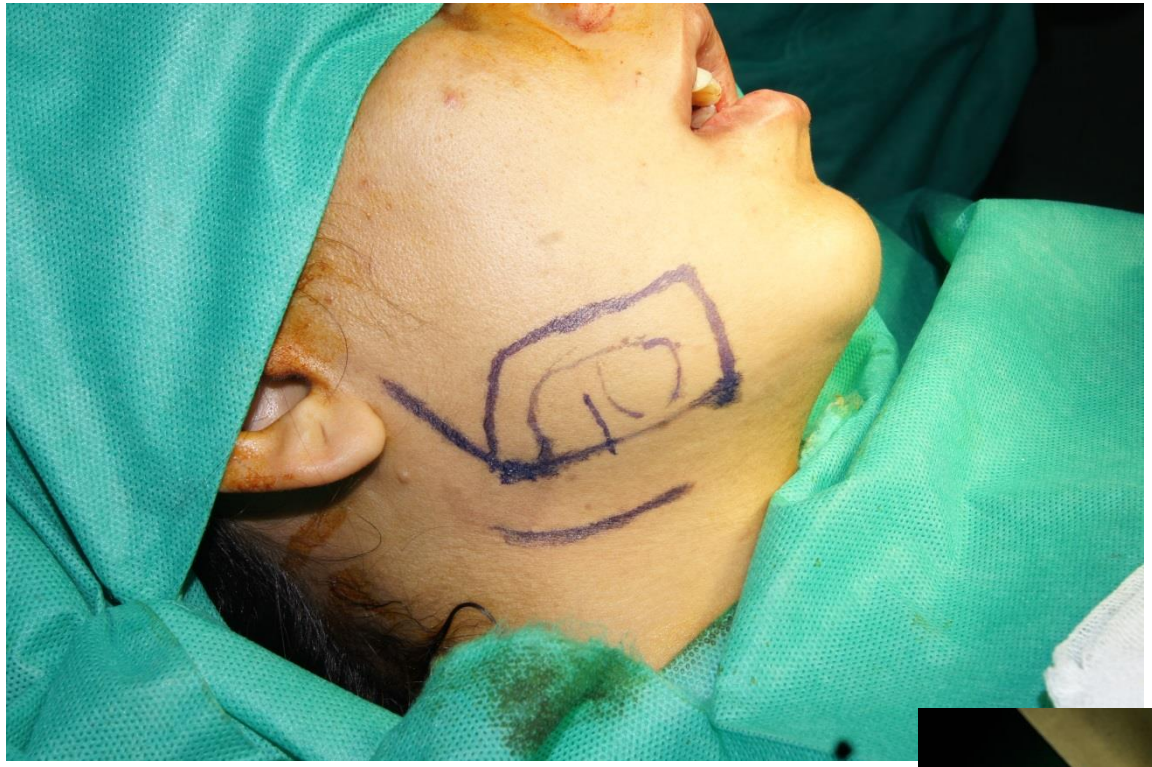


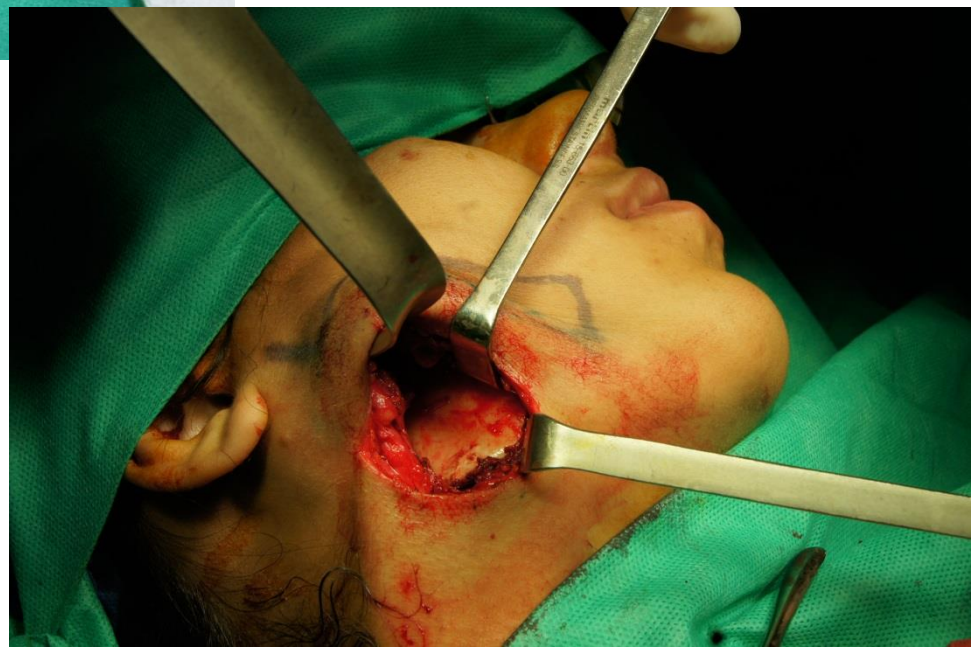


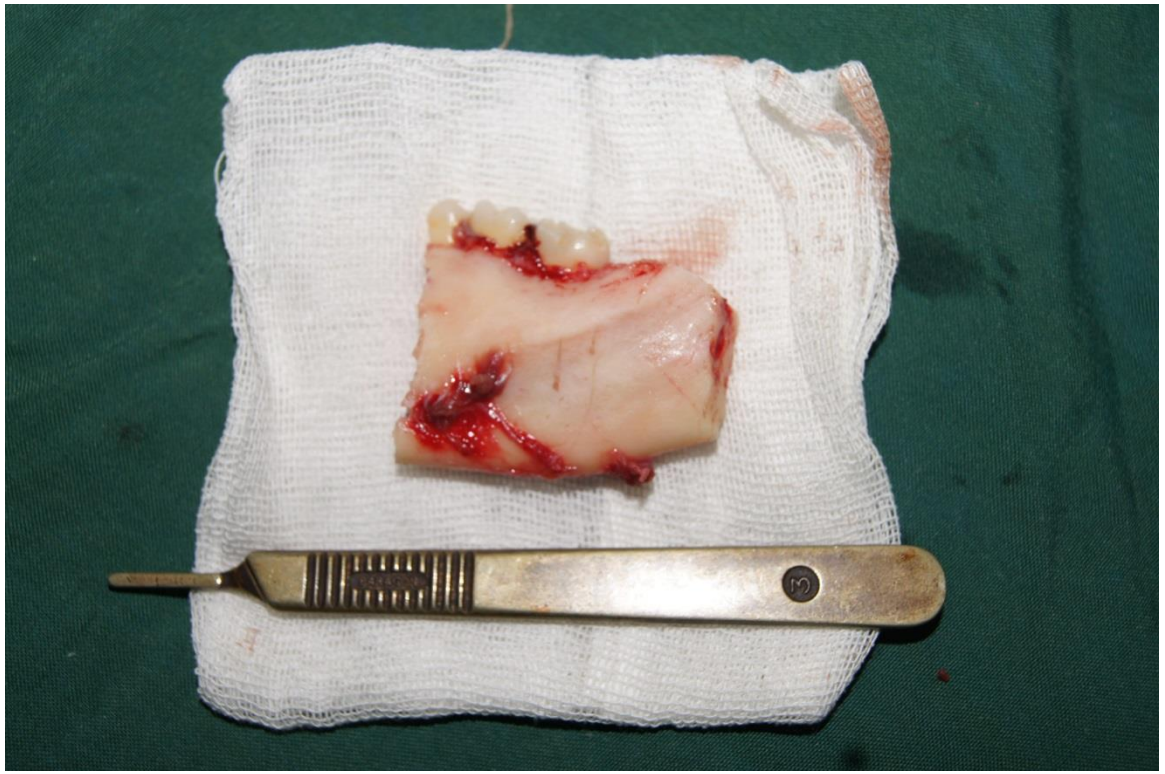


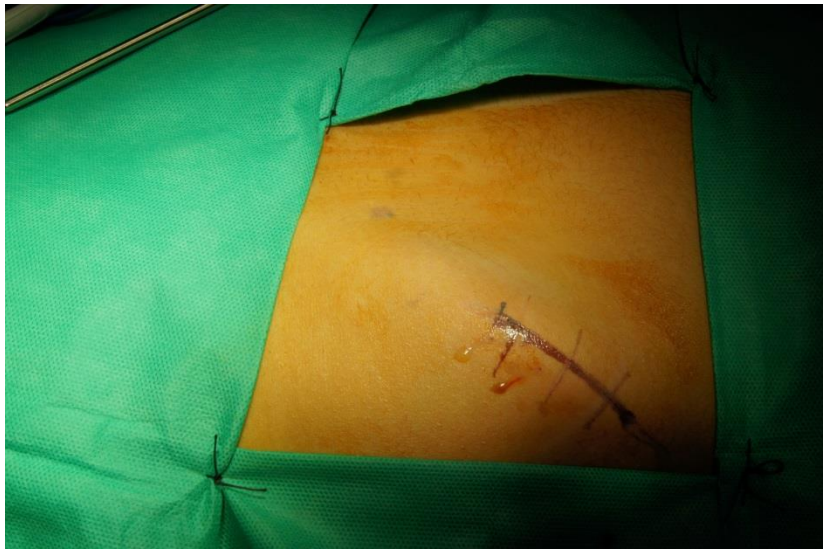


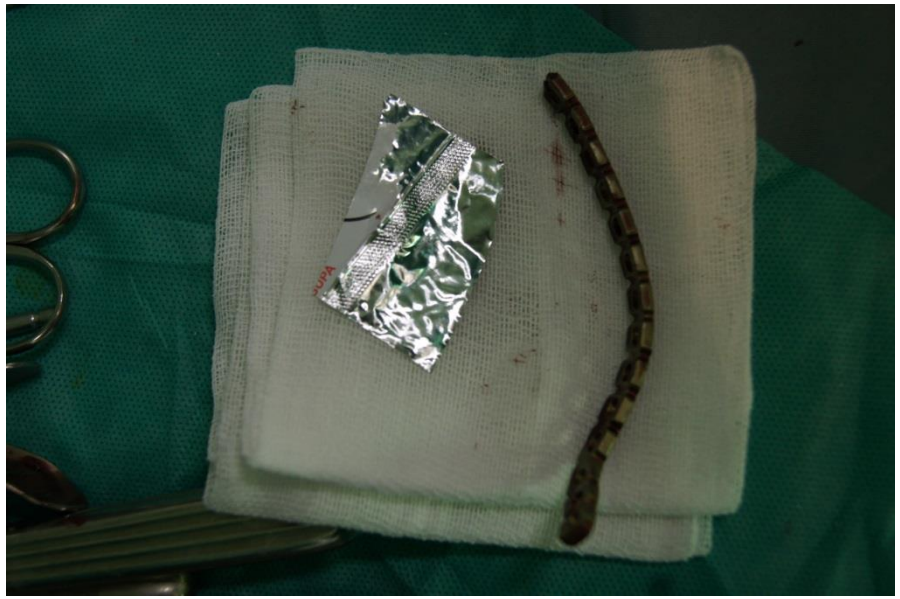


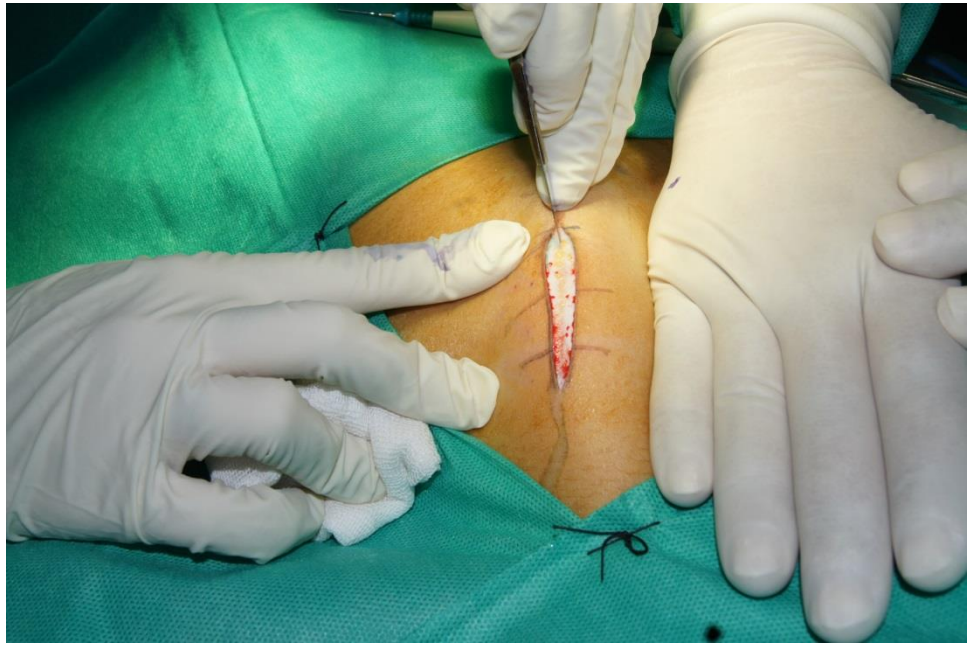


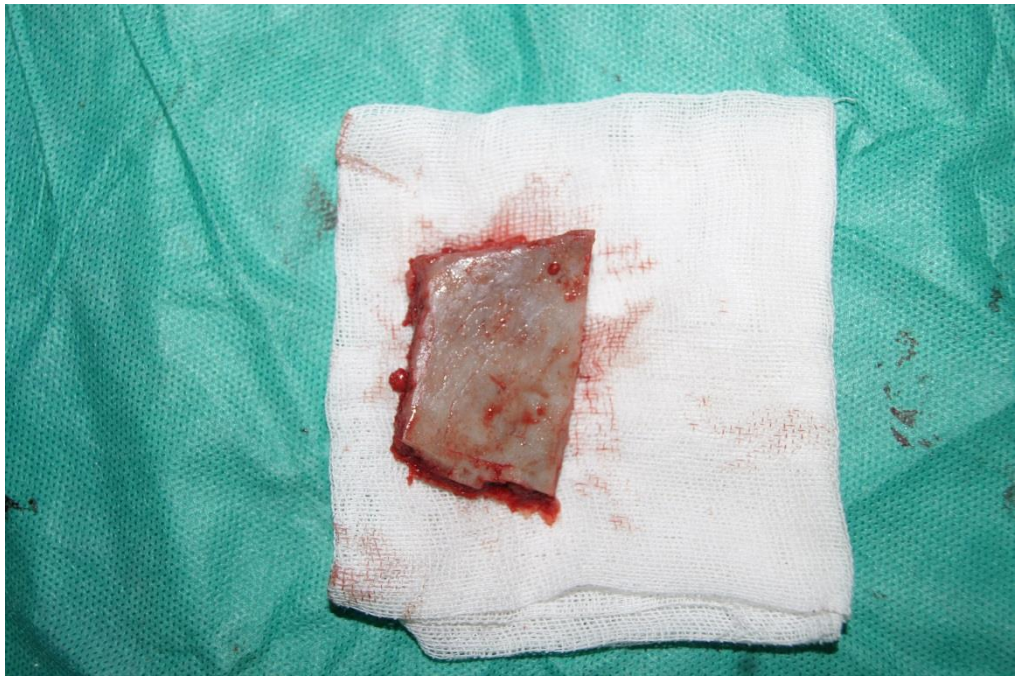


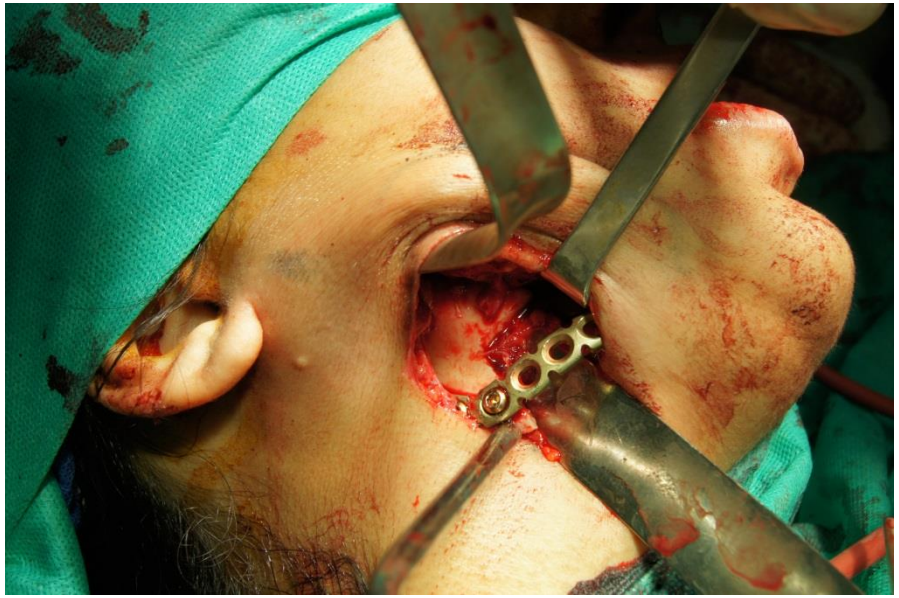


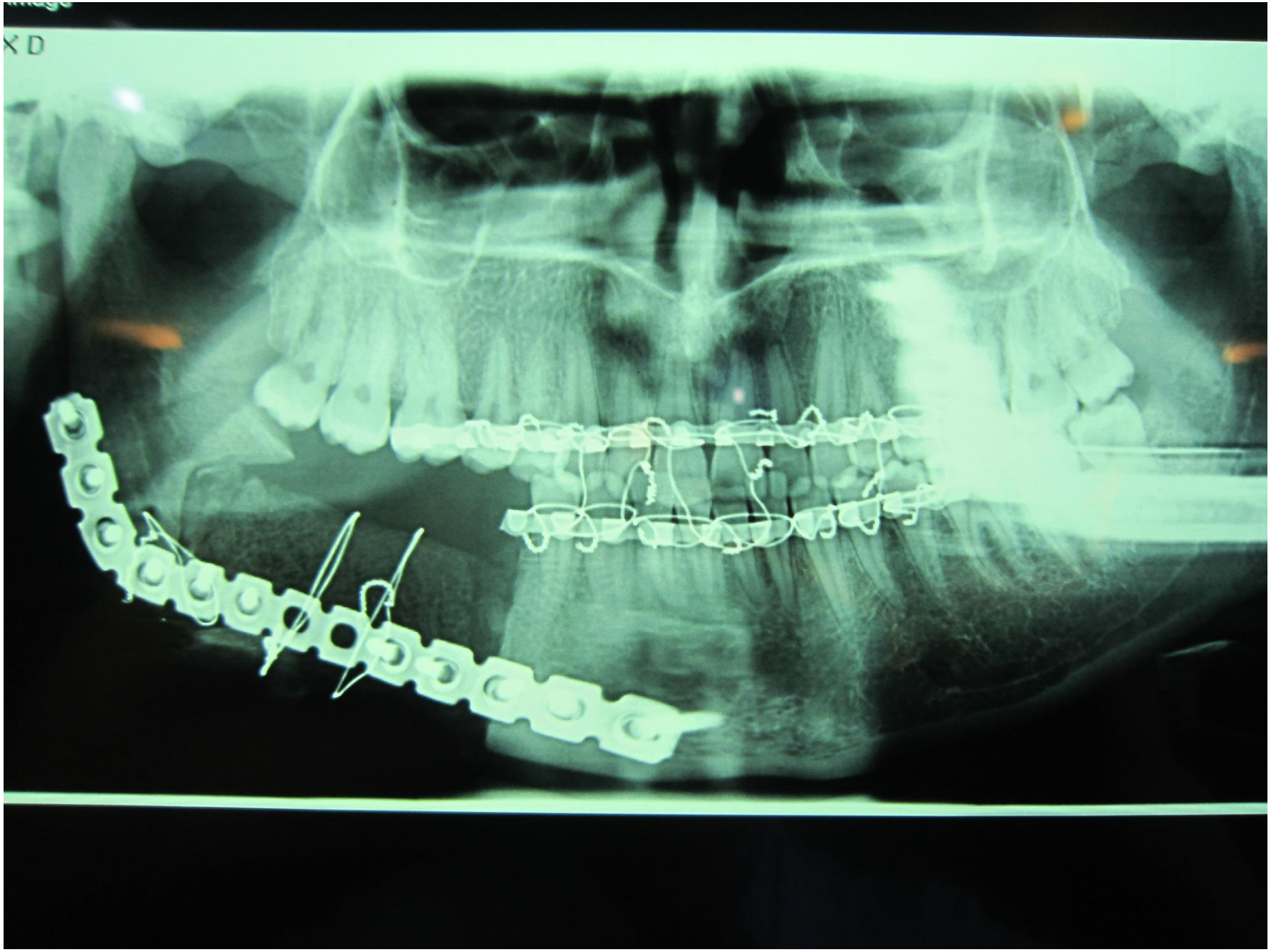








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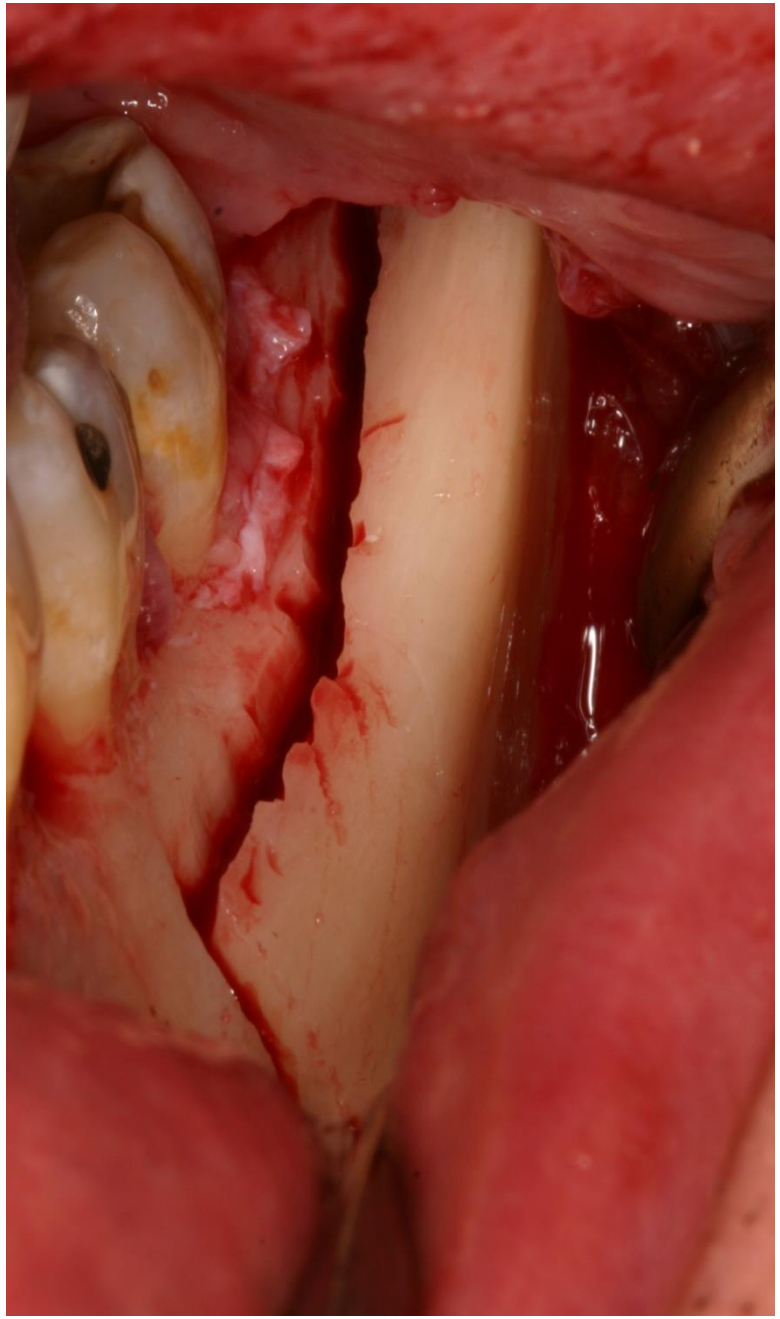

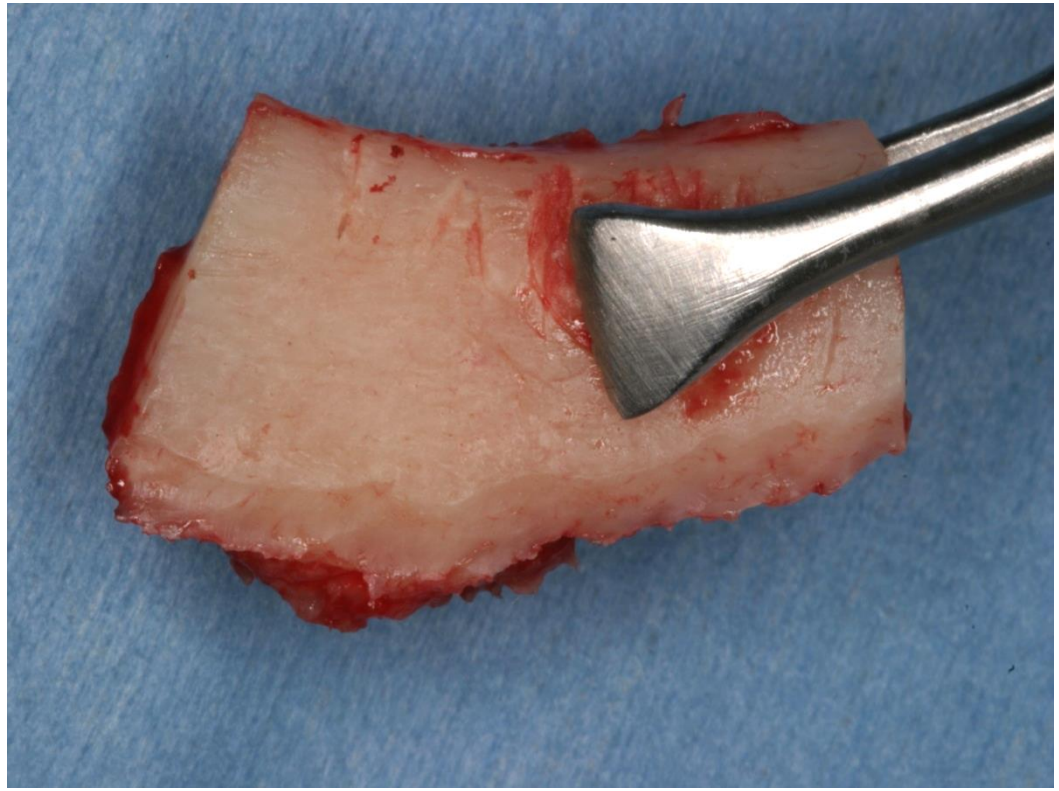

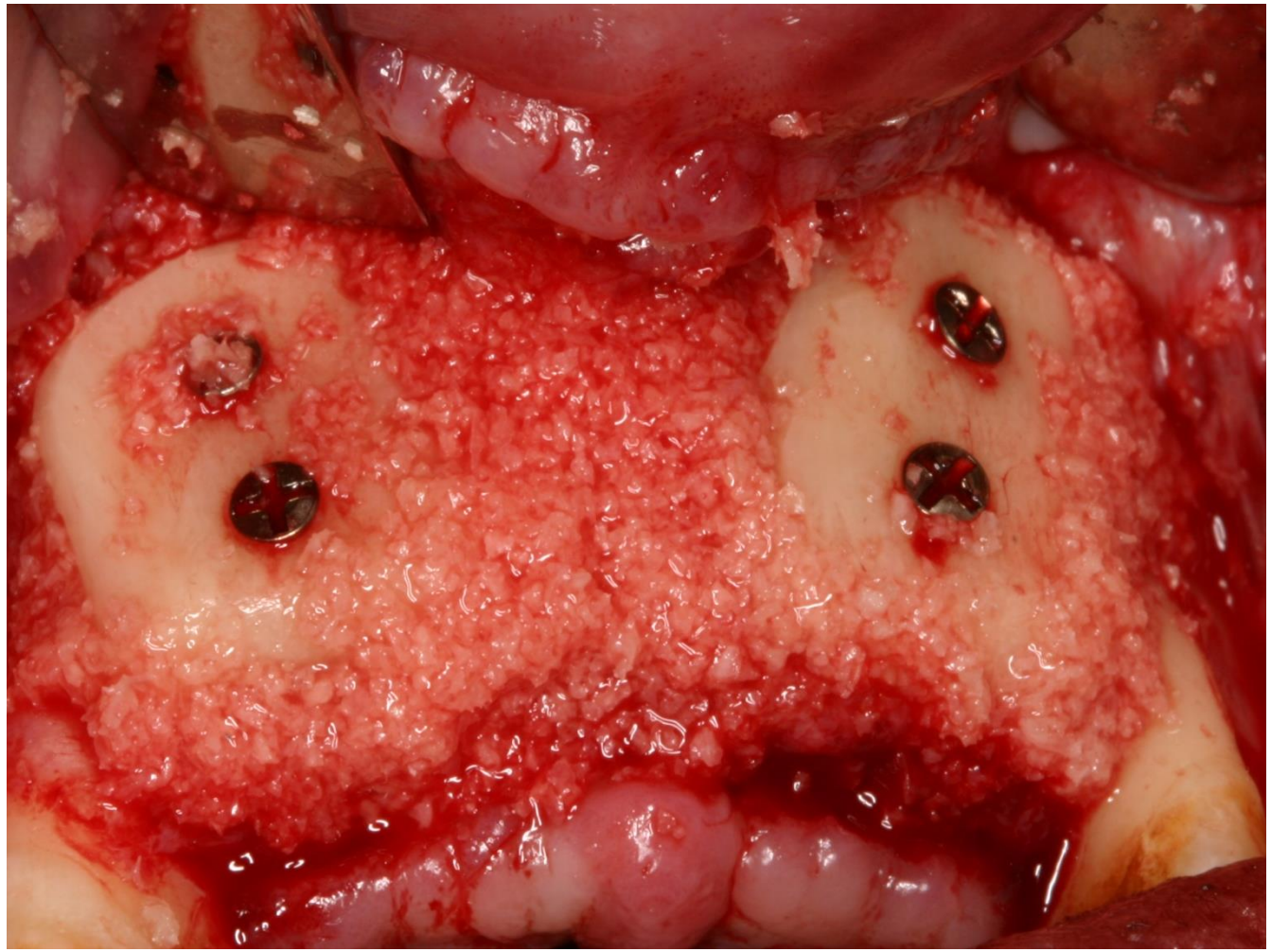

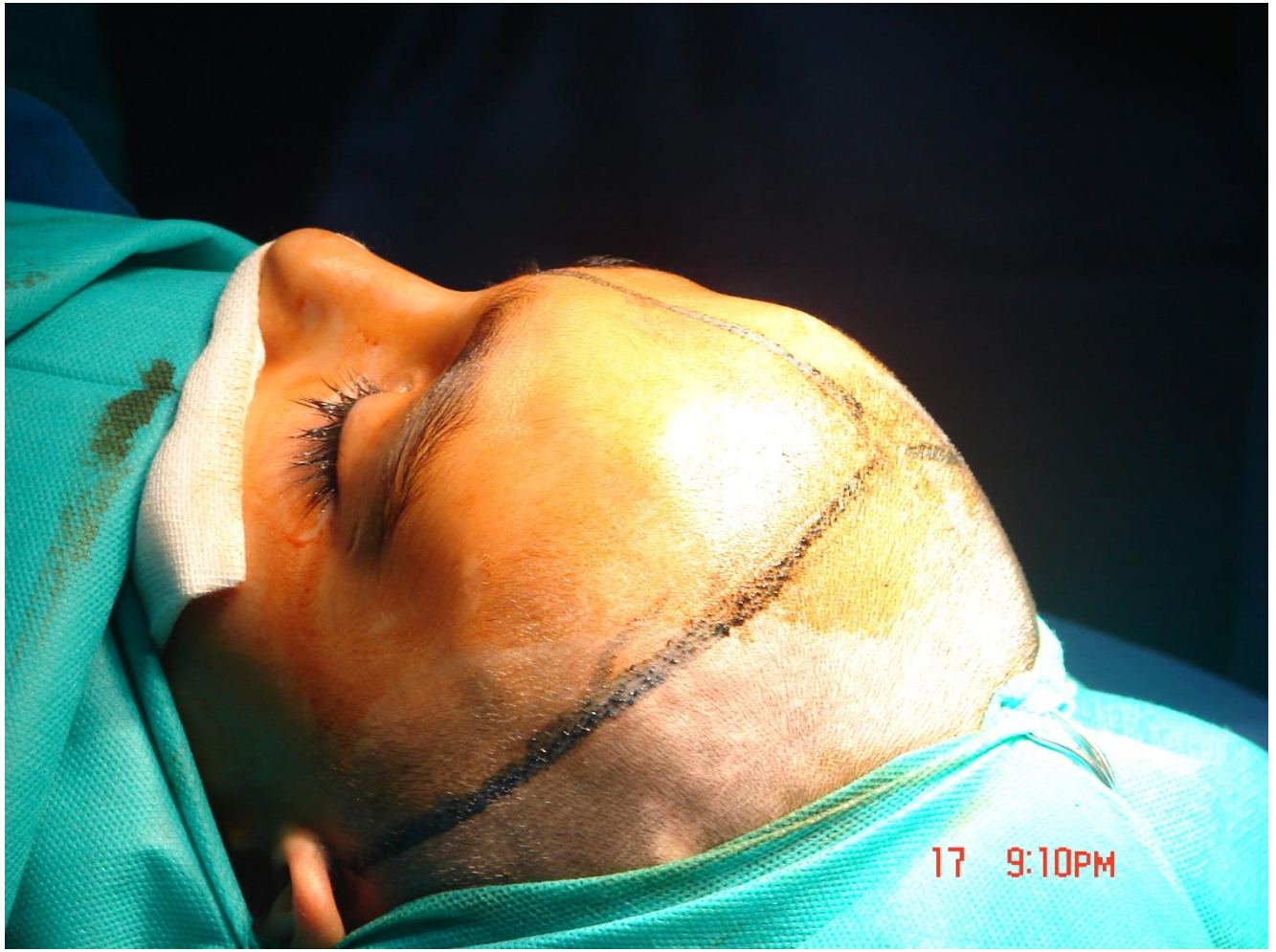
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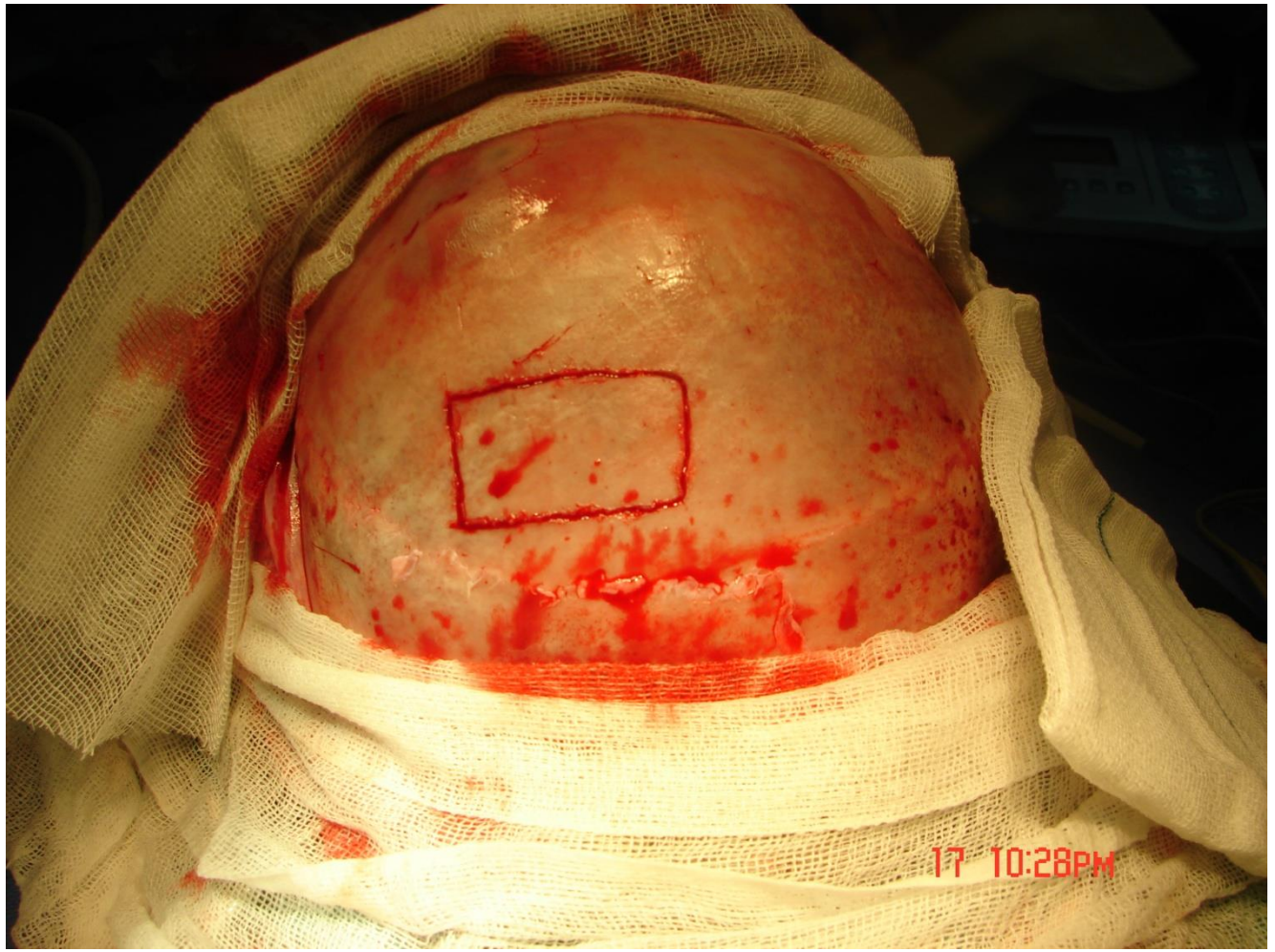
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NONSTEROIDAL ANTIINFLAMMATORY DRUGS

NSAIDs are effective analgesic agents with antiinflammatory and antipyretic activity.

They reduce pain, fever, and inflammation.

The primary mechanism of action is the inhibition of cyclooxygenase (COX) enzymes responsible for the conversion of arachidonic acid into prostaglandins.

Studies have determined that two different isoforms of COX exist, referred to as COX-1 and COX-2.

NONSTEROIDAL ANTIINFLAMMATORY DRUGS

COX-1-mediated prostaglandins maintain homeostasis in the GI tract, kidney, heart, brain, and vasculature.

Prostaglandins protect the GI mucosal integrity by the stimulation and production of mucus and bicarbonate.

In the kidney, prostaglandins regulate blood flow, renin release, and renal tubular salt and water resorption.

In the circulatory system, prostaglandins regulate vascular homeostasis and platelet function.

NONSTEROIDAL ANTIINFLAMMATORY DRUGS

Endogenous **prostaglandins mediated by COX-2** release the inflammatory mediators (including histamine, bradykinin, leukotrienes, and substance P) during tissue trauma.

These mediators result in increased vasodilation and permeability of the peripheral vasculature, edema, erythema, hyperalgesia, loss of function, and pain.

NONSTEROIDAL ANTIINFLAMMATORY DRUGS

NSAIDs possess many advantages, including:

analgesic effects

antiinflammatory effects

antipyretic effects

do not result in sedation

do not result in respiratory depression

do not interfere with bowel and bladder function

they are relatively safe

they have a very low addiction rate

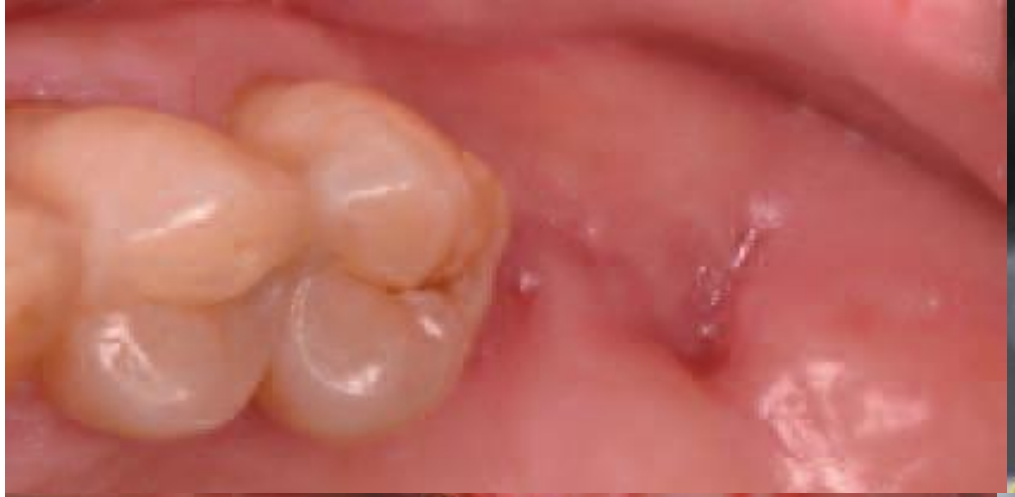
NONSTEROIDAL ANTIINFLAMMATORY DRUGS

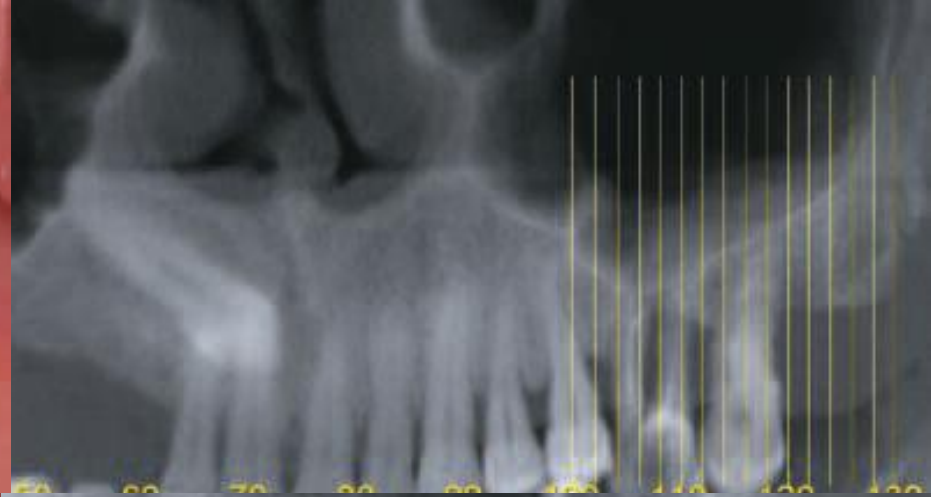
Side effects are infrequent:

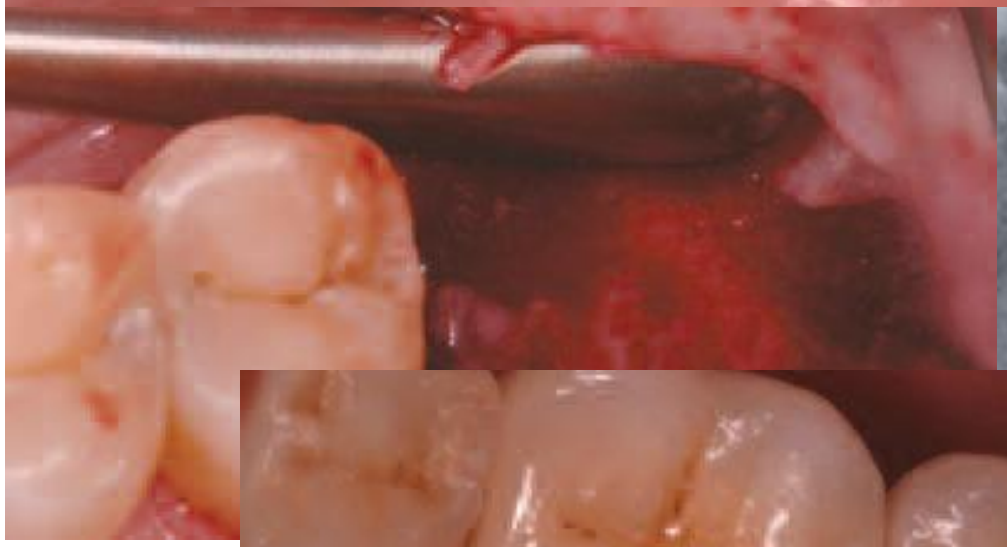
NSAIDs used as analgesic for acute postsurgical pain should be limited to 5 days

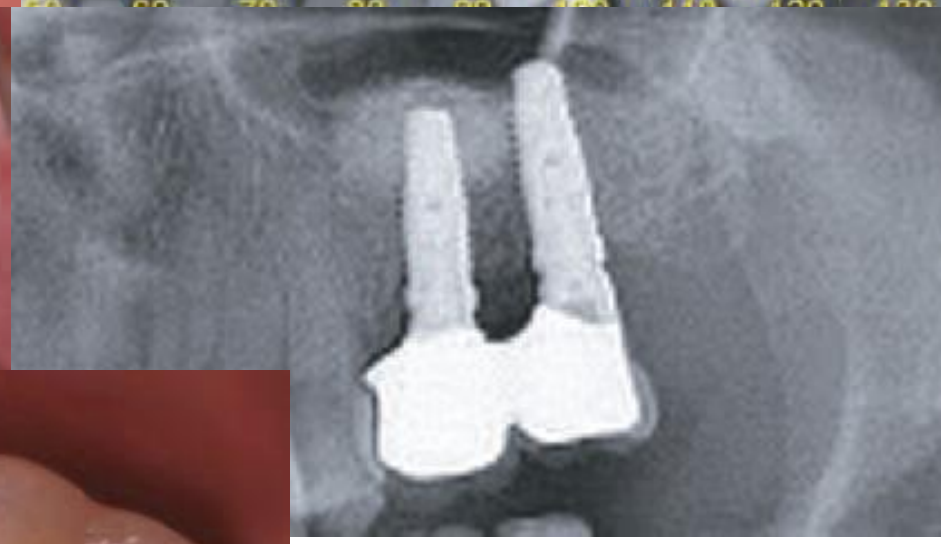
To minimize negative GI side effects, NSAIDs should be taken after meals or with food

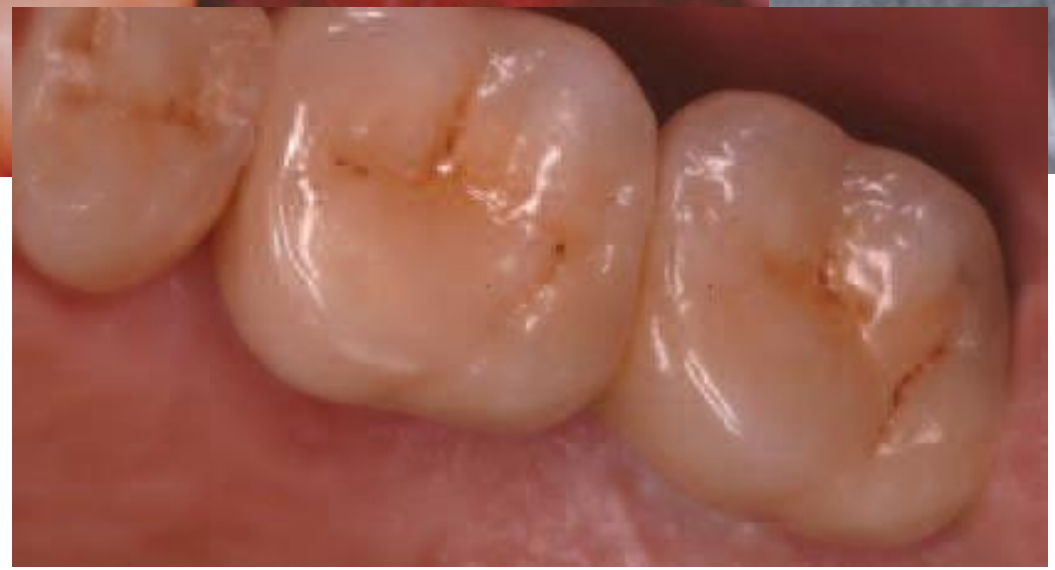
Patients predisposed to GI disease should take NSAIDs with caution, especially if they have a history of alcohol abuse or peptic ulcer disease











Preoperative administration of nsaids

A state of hyperalgesia exists as a result when the local anesthetic wears off.

Patient discomfort subsequently increases during the lag time between NSAID administration and when therapeutic plasma concentrations of the analgesic are attained.

The administration of an NSAID preoperatively appears to maximize the usefulness of this drug class.

The efficacy of various NSAIDs is increased by administering the compound 30 minutes before the beginning of surgery.

Preoperative administration of nsaids

The agent of choice should be that with the fewest side effects.

Aspirin has demonstrated increased ecchymosis.

Diflunisal has demonstrated dry sockets.

Ibuprofen is suggested as the NSAID of choice for pretreatment.

Analgesic potency of this strategy does not appear to strengthen with doses of ibuprofen in excess of 400 mg in comparison with the increased undesirable side effects.

Preoperative administration of nsaids

BOX 6-2 **Recommended Preoperative NSAID Protocol for Postoperative Pain Control**

- Ibuprofen (400 mg) 30 min before the initiation of treatment
- Benefits:
  1. Delayed onset of postoperative pain
  2. Decreased severity of postoperative pain
- Precautions:
  1. DO NOT use in patients for whom NSAIDs are contraindicated (e.g., NSAID allergy or sensitivity, GI ulcerations, renal disease).
  2. Doses of ibuprofen in excess of 400 mg are associated with a greater incidence of unwanted side effects and have not been demonstrated to increase analgesic efficacy.

Disadvantages and contraindications of nsaids

The most common adverse side effects of NSAIDs are GI, including gastritis, ulceration, and bleeding.

The potential for which increases with prolonged usage or high daily doses and prior history of such complications.

Attention has been paid to the potential for NSAIDs to increase the possibility of postoperative bleeding.

With the exception of aspirin, the inhibition of platelet function is reversible with NSAIDs.

DISADVANTAGES AND CONTRAINDICATIONS OF NSAIDS

The production of the powerful platelet aggregating agent, **thromboxane A2**, is indirectly decreased by NSAID inhibition of COX, increasing the likelihood for **GI bleeding**.

Great caution should be exercised when using NSAIDs during pregnancy or with the elderly population taking anticoagulants or corticosteroids.

Additional GI side effects are dyspepsia, peptic ulcer, dysphagia, and abdominal pain.

Disadvantages and contraindications of nsaids

In the **genitourinary system**, NSAIDs adversely affect kidney function in patients with chronic renal disease.

They **decrease** renal blood flow and glomerular filtration rate.

NSAIDs are therefore **contraindicated** in patients with severe renal disease and may cause nephrotoxicity when taken chronically or in combination with other NSAIDs.

Disadvantages and contraindications of nsaids

Other non-GI side effects include:

Severe allergic reactions or anaphylaxis secondary to prostaglandin synthesis inhibition

Tachycardia

Edema

Dizziness

Headache

Increased liver enzymes

Disadvantages and contraindications of nsaids

NSAIDs may **diminish the antihypertensive effect** of three classes of agents, including the ACE inhibitors, ß-blockers, and diuretics, by inhibiting prostaglandin synthesis.

Because NSAID use for this effect is at least 7 to 8 days, their use **should be limited to 4 days** in patients taking antihypertensives.

Disadvantages and contraindications of nsaids

BOX 6-3 NSAID Contraindications

- History of allergy or sensitivity to aspirin or NSAIDs
- History of gastric ulcers
- Bleeding disorders (does not apply to acetaminophen)
- Renal disease
- Hepatic disease
- Pregnant or lactating females
- Asthma

REVIEW OF NONOPIOID ANALGESICS

Aspirin is perhaps the **most common** NSAID.

It has proven to be an effective analgesic, antiinflammatory, and antipyretic.

It has a low therapeutic ceiling; can cause gastric ulcerations, perforations, and bleeding; and inactivates platelets for their lifespan following consumption of the drug.

It can lead to the development of **Reye's syndrome** in children with viral infections.

REVIEW OF NONOPIOID ANALGESICS

Acetaminophen is similarly common to aspirin and similarly safe.

It provides effective relief for mild pain, can be combined with opioids for more serious pain, and has minimal short-term side effects.

It does not impact platelet function.

It does not possess the antiinflammatory properties.

It can cause severe hepatic damage (making it dangerous for alcohol abusers) and has a low therapeutic ceiling.

REVIEW OF NONOPIOID ANALGESICS

Ketorolac is an analgesic used commonly in conjunction with facial trauma, orthognathic surgery, and maxillofacial surgery.

It is injectable and has short-term side effects, such as renal ischemia, GI perforation, and bleeding, that can be lessened by limiting prescriptions to less than 5 days.

It has proven at least as effective as parenteral opioids in treating moderate to severe pain.

REVIEW OF NONOPIOID ANALGESICS

Celecoxib is currently the only **COX-2 inhibitor** up for commercial sale.

The drug only inhibits COX- 2-mediated prostaglandins, which cause inflammation, limiting the negative effects on the gastroprotective qualities of the COX-1 isoform.

COX-2 inhibitors can **disturb the hemodynamic balance** between the body's organs and adversely affects the cardiovascular system.

Trials have shown little difference in postoperative pain relief between COX-2 inhibitors and ibuprofen.

REVIEW OF NONOPIOID ANALGESICS

TABLE 6-3 Nonopioid Analgesics

Generic	Pain Level	Dose (mg)	Interval (hr)	Maximum Dose/ 24 hr (mg)	Additional Comments
SALICYLIC ACID DERIVATIVES					
Aspirin	Mild	650-1000	4-6	4000	Increased risk of bleeding with excessive alcohol intake (≥3 drinks/day), avoid use with viral infections in children or teenagers, syndrome of asthma, rhinitis, and nasal polyps
Diflunisal	Mild to moderate	500	12	1500	1000-mg loading dose
ρ-AMINOPHENOL DERIVATIVES					
Acetaminophen	Mild	650-1000	4-6	4000	Increased risk of hepatotoxicity with excessive alcohol intake (≥3 drinks/day)
PROPIONIC ACID DERIVATIVES					
Ibuprofen	Mild to moderate	400	4-6	3200	
Ketoprofen	Mild to moderate	25-50	6-8	300	
Naproxen sodium	Mild to moderate	275	6-8	1375	Start 550 mg then 275 mg every 6-8 hr
HETEROARYL ACETIC ACIDS					
Diclofenac	Mild to moderate	50	8	150	Loading dose up to 100 mg, total of 200 mg
Ketorolac	Moderate to severe	60 IM, 30 IV	Single dose	120	
		30 IM/IV	6	120	Transition from IV/IM to PO 20 mg, then every 4-6 hr; total duration of use ≤5 days
		20 then 10 PO	4-6	40	
COXIBS					
Celecoxib	Mild to moderate	200	12	400	400-mg loading dose, then 200 mg if needed, selective COX-2 inhibitor

REVIEW OF NONOPIOID ANALGESICS

Corticosteroids act earlier in the cascade by suppressing arachidonic acid production, **inhibiting** prostaglandins and leukotrienes.

They also have **central antinociceptive properties** at the spinal cord level.

This leads to decreased pain and earlier ambulation, ability to consume food orally, and ultimately a shorter hospital stay.

For procedures expected to cause significant inflammation, methylprednisolone and other steroids have been prescribed.

A **disadvantage of concurrent use** of corticosteroids with NSAIDs is the significant increase in the risk of life-threatening GI bleeding.

Perioperative pain control considerations

Each oral and maxillofacial surgeon must anticipate a **pain management strategy** for individual patients.

Attenuation of the nociceptive impulses to the surgical insult while minimizing drug side effects, including nausea, diminished cognitive and motor function, and reduced patient anxiety, should be **the goal of this strategy**.

Postoperative pain management begins preoperatively.

Perioperative pain control considerations

BOX 6-4 Conclusions of Published Studies

- Half of all patients given conventional therapy for their pain do not get adequate relief. These patients continue to feel moderate to severe pain.
- Prescribing pain medication only "as needed" can result in prolonged delays because patients may delay asking for help.
- Aggressive prevention of pain is better than treatment because once established pain is more difficult to suppress.
- Patients have a right to treatment that includes prevention of pain and adequate pain relief. The surgeon needs to develop pain control plans before surgery and inform the patient what to expect in terms of pain after the surgery.
- Fears of postsurgical addiction to properly prescribed and administered opioids are generally groundless.

Perioperative pain control considerations

BOX 6-5 Key Patient Education Steps

- Describe the expected type of pain and its probable duration to decrease the uncertainty and fears of the unknown.
- Individualize the information for the patient.
- Discuss goals of pain management and how these goals help the patient's comfort, hasten recovery, and reduce complications.
- Reinforce the concept that pain prevention is important to good pain management. The patient should try to anticipate their pain medication requirements.
- Many drug and nondrug treatment options can be helpful in preventing and managing pain.
- Inform the patients of when and how to contact the surgeon about his or her pain.
- Parents of minor patients and the surgeon will decide as a team which treatments are best to manage their pain.
- Discuss treatment plan and choices including the schedule of medications that are appropriate to the patient.

Assessment and management of acute pain

In assessing and formulating a pain management plan, the clinician must consider the multiple factors that influence analgesic requirements.

BOX 6-6 Factors That Influence Analgesics Requirements

- Age of the patient. Very old or very young patients require smaller doses.
- Sex.
- Preoperative analgesic use.
- Past history of poor pain management.
- Coexisting medical conditions, such as substance abuse, anxiety disorder, affective disorder, hepatic or renal impairments.
- Cultural factors and personality.
- Preoperative patient education. Appropriate preoperative education can improve expectations, compliance, and ability to effectively interact with pain management techniques.
- Individual variations in response and pain threshold.
- Attitude of the clinical staff.

Assessment and management of acute pain

	Type of Pain		
	Somatic Pain	**Visceral**	**Neuropathic Pain**
Location	Localized	Generalized	Radiating or specific
Patient Description	Pin prick, or stabbing, or sharp	Ache, or pressure, or sharp	Burning, or prickling, or tingling, or electric shock-like, or lancinating
Mechanism of Pain	A-delta fiber activity; located in the periphery*	C Fiber activity; involved deeper innervation*	Dermatomal ‡ (peripheral), or non-dermatomal (central)
Clinical Examples	• Superficial laceration • Superficial burns • Intramuscular injections, venous access • Osseous surgery • Stomatitis • Extensive abrasion	• Periosteum, joints, muscles • Muscle spasm pain† • Osseous surgery	• Trigeminal • Avulsion neuralgia • Post-traumatic neuralgia • Peripheral neuropathy (diabetes, human immunodeficiency virus [HIV]) • Herpetic neuralgia
Most Responsive Treatments	• Acetaminophen • Cold packs • Corticosteroids • Local anesthetic either topically or by infiltration • Non-steroidal anti-inflammatory drugs (NSAIDs) • Tactile stimulation	• Corticosteroids • NSAIDs • Opioid via any route • Antispasmodics	• Anticonvulsants • Corticosteroids • Neural blockade • NSAIDs • Opioids via any route • Tricyclic antidepressants

* Most post-operative patients experience A-delta and C Fiber pain and respond best to narcotic of any route and NSAIDs.

† Muscle spasms may be less responsive to opioids. Respond best to antispasmodics, NSAIDs, benzodiazepines, baclofen.

‡ Segmental distribution follows a dermatome chart. This traces the pathway of sensation to its nerve root.

Pre-emptive analgesia therapy

By **preventing the sensitization of the CNS**, which would normally amplify subsequent nociceptive input, one may reduce the severity of postoperative pain.

Giving an NSAID 30 minutes before a surgical visit improves the patient's postoperative comfort with delayed onset and decreased severity of postoperative pain.

During the postoperative period, regular interval dosing results in steady plasma concentration of the analgesic.

Pre-emptive analgesia therapy

Administration of intraoperative ketorolac has demonstrated a decrease in the amount and incidence of postoperative pain therapy.

The long-acting local anesthetics bupivacaine and etidocaine are preemptive in that they prevent neurotransmission of nociceptive stimuli for up to 8 to 12 hours.

Corticosteroids have been demonstrated to reduce opioid requirements in both orthognathic and third molar surgery. Steroids are recommended as a perioperative adjunct medication.

Patient-controlled analgesia

PCA is a method of inpatient, self-administered analgesia according to the surgeon's orders to control his or her pain.

A programmable infusion pump that delivers opioids at a continuous infusion rate (milligrams per hour) along with a patient-controlled demand bolus is administered via an IV.

A lock-out interval, when the pump will not allow more boluses to be administered, is programmed into the pump.

The primary advantage of the PCA is patient convenience and preemptive pain control because the patient controls when a dose of analgesia is given.

Thanks for your attention