Oral Surgery

Central Nervous System (CNS) Diseases

Patients with neurologic diseases are at high risk during dental treatment for complications such as stroke, myocardial infarction (MI), adverse bleeding, altered consciousness, and infection. The dentist must be able to detect these patients, based on history and clinical findings, refer them for medical diagnosis and management, and work closely with their physicians to develop dental management plans that will be effective and safe for these patients.

Epilepsy

Epilepsy is a term that refers to a group of disorders characterized by chronic recurrent, paroxysmal changes in the neurologic function (seizures), altered consciousness, or involuntary movements caused by abnormal and spontaneous electrical activity in the brain.

Seizures (fits) may be convulsive (i.e., accompanied by motor manifestations) or may occur with other changes in neurologic function (i.e., sensory, cognitive and emotional).

Although seizures are required for the diagnosis of epilepsy, not all seizures imply epilepsy. Seizures may occur during many medical or neurologic illnesses, including stress, sleep deprivation, fever, alcohol or drug withdrawal, and syncope.

The cause of epilepsy is idiopathic in more than half of all patients. Other causes of fits include:

- Vascular abnormalities (cerebrovascular disease).
- Developmental abnormalities (cavernous malformation).
- Intracranial neoplasms (gliomas).
- Head trauma.
- Hypoglycemia
- Drug withdrawal.
- Infection and febrile illness (meningitis, encephalitis).
- Genetic conditions such as Down syndrome.
- Syncope and diminished oxygen supply to the brain also are known to trigger seizures.

Seizures sometimes can be evoked by specific stimuli like after exposure to flickering lights, music or a loud noise. During the seizure, blood becomes hypoxic and lactic acidosis occurs.

Classification of Epilepsy

1. Partial seizures (focal, local)

   a. Simple partial seizures
   b. Complex partial seizures

2. Generalized seizures (convulsive or nonconvulsive)

   a. Absence seizures (petit mal)
   b. Tonic-clonic seizures (grand mal)

Petit mal seizures: Most often come during childhood and are characterized by minimal or no movements (except for eye blinking) and an apparently blank stare, and brief sudden loss of awareness or of conscious activity; which may only last seconds.

Grand mal (tonic-clonic) epilepsy: Usually begin in childhood, or sometimes at about puberty. There is a warning **(aura),** followed by loss of consciousness, tonic and clonic convulsions and finally a variably prolonged recovery.

The **aura** may consist of a mood change, irritability, brief hallucination or headache. After the aura warning, the patient emits a sudden "epileptic cry" (caused by spasm of the diaphragmatic muscles) and immediately loses consciousness. The tonic phase consists of generalized muscle rigidity, pupil dilation, eyes rolling upward or to the side, and loss of consciousness. Breathing may stop because of spasm of respiratory muscles. This is followed by clonic activity that consists of uncoordinated beating movements of the limbs and head, forcible jaw closing, and head rocking. Urinary incontinence is common. The seizure usually does not last longer than 90 seconds; then movement ceases, muscles relax and a gradual return to consciousness occurs, which is accompanied by headache, confusion and mental dulling. Several hoursof rest or sleep may be needed for the patient to fully regain cognitive and physical abilities.

A major fit is so dramatic that it seems to be of longer duration that is in fact the case but if it lasts more than 5 minutes (by the clock) or starts again after apparently ceasing, the patient must be regarded as beingin ***status epilepticus,*** which is particularly dangerous (the mortality can be up to 20%). Brain damage may result from cerebral hypoxia, when tonic and clonic phases alternate repeatedly without consciousness being regained and there can also be inhalation of vomit and saliva. This condition is most frequently caused by abrupt withdrawal of anticonvulsant medication or an abused substance that may be triggered by infection, neoplasm, or trauma. Status epilepticus constitutes a medical emergency.

Laboratory findings

The diagnosis of epilepsy generally is based on the history of seizures and an abnormal electroencephalogram (EEG). Other diagnostic procedures that are useful for ruling out other causes of seizures include: **CT, MRI, LUMBER PUNCTURE, SERUM CHEMISTRY PROFILES, AND TOXICOLOGY SCREENING**

Medical management

The medical management of epilepsy is usually based on long-term drug therapy. Phenytoin (Dilantin), carbamazepine (Tegretol), and valproic acid (Depakene) are considered first line treatments. These drugs act by reducing the frequency of seizures by elevating the seizure threshold of motor cortex neurons, depressing abnormal cerebral electrical discharge, and limiting the spread of excitation from abnormal foci. Phenytoin and carbamazepine are efficient at blocking sodium or calcium channels of motor neurons. Phenytoin, carbamazepine and valproic acid can cause bone marrow suppression, leucopenia, and thrombocytopenia, resulting in an increased incidence of microbial infection, delayed healing, and gingival and postoperative bleeding. Valproic acid can decrease platelet aggregation, leading to spontaneous hemorrhage and petechiae.

Adverse effects of phenytoin: Gingival hyperplasia, increased incidence of infection, delayed healing, gingival bleeding (leucopenia), osteoporosis and Stevens-Johnson syndrome. {Metronidazole can interfere with phenytoin}.

Adverse effects of carbamazepine: Xerostomia, infection, delayed healing, ataxia, gingival bleeding (leucopenia and thrombocytopenia), osteoporosis, Stevens-Johnson syndrome.

Notes:

a. Erythromycin should not be administered to patients who are taking carbamazepine because of interferencewith metabolism of carbamazepine, which could lead to toxic levels of anticonvulsant drug.
b. Aspirin and NSAIDs should not be administered to patients who are taking valproic acid because they canfurther decrease platelet aggregation, leading to haemorrhagic episodes.
c. No contraindication has been identified to the use of local anesthetics in proper amounts to these patients.

Dental management

i. Identification of patient by history

a. Type of seizure

b. Age at time of onset

c. Cause of seizures (if known)

d. Medications

e. Frequency of physician visits (name and phone number)

f. Degree of seizure control

g. Frequency of seizures

h. Date of last seizure

i. Known precipitating factors

j. History of seizure-related injuries

ii. Provision of normal care: Well-controlled seizures pose no management problems

iii. If questionable history or poorly controlled seizures, consultation with physician before dentaltreatment—may require modification of medications.

iv. Attention to adverse effects of anticonvulsants; these include:

a. Drowsiness

b. Slow mentation

c. Dizziness

d. Ataxia

e. Gastrointestinal upset

f. Allergic signs (rash, erythema multiforme)

v. Possibility of bleeding tendency in patients taking valproic acid (Depakene) or carbamazepine (Tegretol) as the result of platelet interference—Pretreatment platelet function analyzer (PFA)-100; if grossly abnormal, consultation with physician.

vi. Seizure management

- **Preventive measures include:**
  - Knowing the patient's history.
  - Scheduling the patient at a time within a few hours of taking the anticonvulsantmedication.
  - Using a mouth prop.

- Removing dentures.
- Discussing with the patient the urgency of mentioning the aura as soon as it is sensed.
- If sufficient time occurs, 0.5 to 2 mg of lorazepam can be given sublingually, or 2 to 10 mg ofdiazepam can be given intravenously.

- **If the patient has a seizure while in the dental chair;**
  - The primary task is to protect the patient and try to prevent injury.
  - No attempt should be made to move the patient to the floor.
  - The instruments and the instrument tray should be cleared from the area, and the chairshould be placed in a supported supine position.
  - The patient's airway should be maintained patent (Turn the patient to the side to avoidaspiration).
  - Passive restraint should be used only, to prevent injury that may result when thepatient hits nearby objects or falls out of the chair.

- **After the seizure**
  - Oxygen 100% (if available).
  - Maintenance of a patent airway.
  - Mouth suction should be provided (to minimize aspiration of secretions).
  - Discontinue dental treatment.
  - Examine for traumatic injuries (lacerations, fractures). In the event of avulsed or fractured teeth orfractured appliance, an attempt should be made to locate the tooth or fragments to rule out aspiration.

- **In the event that a seizure becomes prolonged or is repeated (status epilepticus):**
  - Intravenous lorazepam (4 to 8 mg, or 10 mg diazepam, is generally effective in controlling it.
  - Oxygen and respiratory support should be provided because respiratory function may becomedepressed.
  - If the seizure lasts longer than 15 minutes, the following should be provided: IV access, repeatlorazepam dosing, phenytoin administration and activation of the emergency medical system.

Stroke

Stroke is a generic term that is used to refer to a cerebrovascular accident—a serious and often fatal neurologic event caused by sudden interruption of oxygenated blood to the brain. This in turn results in focal necrosis of brain tissue and possibly death. Even if a stroke is not fatal, the survivor often is to some degree debilitated in motor function, speech, or mentation.

Etiology

Stroke is caused by the interruption of blood supply and oxygen to the brain as a result of ischemia or haemorrhage.

Ischemic stroke (most common type): Induced by thrombosis or occlusion of a cerebral blood vessel by distant emboli. Its risk increased by atherosclerosis and cardiac pathosis (myocardial infarction, atrial fibrillation).

Hemorrhagic stroke: Hypertension is the most important risk factor for intracerebral hemorrhagic stroke.

- Additional factors that increase the risk for stroke include:

1. The occurrence of transient ischemic attacks.
2. Previous stroke.
3. High dietary fat.
4. Obesity and elevated blood lipid levels.
5. Physical inactivity.
6. Uncontrolled hypertension.
7. Cardiac abnormalities.
8. Diabetes mellitus.
9. Heavy tobacco smoking.
10. Increasing age.
11. Periodontal disease.

Signs and Symptoms

Familiarity with the warning signs and symptoms and the phases of stroke can lead to appropriate action that may be lifesaving.

- Four Events Associated With Stroke Are:

A. Transient ischemic attack (TIA): Is a "mini" stroke that is caused by a temporary disturbance in blood supply to a localized area of the brain. A TIA often causes numbness of the face, arm, or leg on one side of the body, weakness, tingling, and speech disturbances that usually last less than 10 minutes.

B. Reversible ischemic neurologic deficit (RIND): Neurologic deficit recovers within 24 hours.

C. Stroke-in-evolution: In which the deficit has been present for several hours and continues to worsen during a period of observation.

D. Completed stroke: Signs of stroke include hemiplegia, temporary loss of speech or trouble in speaking or understanding speech, temporary dimness or loss of vision, particularly in one eye (may be confused with migraine), unexplained dizziness, unsteadiness, or a sudden fall.

Laboratory Findings

Patients suspected of having had a stroke usually receive a variety of laboratory and diagnostic imaging tests to rule out conditions that can produce neurologic alterations, such as diabetes mellitus, uremia, abscess, tumor, acute alcoholism, drug poisoning, and extradural hemorrhage.

Laboratory tests often include urinalysis, blood sugar level, complete blood count, erythrocyte sedimentation rate, serologic tests for syphilis, blood cholesterol and lipid levels, chest radiographs, and electrocardiogram.

Doppler blood flow, EEG, cerebral angiography, CT, and MRI, are important for determining the extent and location of arterial injury.

Medical management

- ***Prevention:*** This is accomplished by identifying risk factors in individuals (e.g., hypertension, diabetes, atherosclerosis, cigarette smoking) and attempting to reduce or eliminate as many of these as possible. Blood pressure lowering, antiplatelet therapy and statin therapy are primary stroke prevention methods.
- ***Stroke Treatment:***

1. The immediate task is to sustain life during the period immediately after the stroke. This is done by means of life support measures and transport to a hospital.
2. The second task involves emergency efforts to prevent further thrombosis or haemorrhage, and to attempt to lyses the clot in cases of thrombosis or embolism. Thrombolysis and improved neurologic outcomes have been achieved with intravenous recombinant tissue-type plasminogen activator (rt-PA).
3. After the initial period, efforts to stabilize the patient continue with anticoagulant medications such as heparin, coumarin, aspirin, and dipyridamole combined with aspirin (Aggrenox) in cases of thrombosis or embolism. Heparin is administered intravenously during acute episodes, whereas coumarin, dipyridamole, aspirin, subcutaneous low molecular weight heparin, or platelet receptor antagonists (ticlopidine) are employed for prolonged periods to reduce risk of thrombosis.
4. Corticosteroids may be used acutely after a stroke to reduce the cerebral edema that accompanies cerebral infarction. This can markedly lessen complications.
5. Surgical intervention may be indicated for removal of a superficial hematoma or management of a vascular obstruction.
6. Valium, Dilantin, and other anticonvulsants are prescribed in the management of seizures that may accompany the postoperative course of stroke.

7. If the patient survives, the final task consists of institution of preventive therapy, administration of medications that reduce the risk of another stroke (statins and antihypertensive drugs), and initiation of rehabilitation (physical and speech therapy).

Dental Management of the Patient with Stroke

1. Identify risk factors.

1. Hypertension*
2. Congestive heart failure*
3. Diabetes mellitus*
4. TIA or previous stroke*
5. Increasing age ≥75 years*
6. Elevated blood cholesterol or lipid levels
7. Coronary atherosclerosis
8. Cigarette smoking

Note: Risk of stroke increases by a factor of 1.5 for each condition above indicated by*. Thus, havingmultiple risk factors listed above greatly increases the risk of a stroke.

2. Encourage control of risk factors (referral to physician, if appropriate).

3. Obtain thorough history of stroke.

i. Note date of event, current status, medical therapy, and any residual disabilities.

ii. Provide only urgent dental care during first 6 months after a stroke, TIA, or RIND.

iii. Avoid elective care in patients who have had recent TIAs or RINDs.

iv. Determine risk for bleeding problems in patients taking anticoagulant drugs, and minimizeperioperative bleeding;

a) Aspirin + dipyridamole (Aggrenox), ticlopidine (Ticlid); obtain pretreatment PFA100.
b) Coumarin-Pretreatment INR 3.5. Higher levels require consultation with physician to reduce dose.
c) Heparin (IV)—Use palliative emergency dental care only, or 6 to 12 hours before surgery, discontinue heparin and start another anticoagulant (e.g., coumadin) withphysician's approval. Then, restart heparin after clot forms (6 h later).

d) Heparin (subcutaneous, low molecular weight)—generally, no changes required.
e) Use measures that minimize haemorrhage (atraumatic surgery, pressure, gelfoam, suturing), as needed.
f) Have available nonadrenergic haemostatic agents and devices (stents, electrocautery).

4. Schedule short, stress-free, midmorning appointments. Provide N20-02 inhalation as needed.
5. Monitor blood pressure and oxygen saturation.
6. Use minimum amount of anaesthetic containing vasoconstrictor. (A local unesthetic with 1:100,000 or 1:200,000 epinephrine may be used in judicious amounts (>= 4 mL).
7. Avoid epinephrine in gingival retraction cord.
8. Recognize signs and symptoms of a stroke, provide emergency care, and activate emergency medical support system.
9. A prior stroke may require assistance for patient transfer to the chair, effectiveoral evacuation and airway management, and rigorous oral hygiene measures delivered by a health care provider.

Oral Manifestations and Complications

A stroke-in-evolution

A patient may show all or any of the following;

- Slurred speech
- Weak muscles
- Difficulty in swallowing.

Complete stroke

- Loss of or difficulty in speech
- Unilateral paralysis of the orofacial musculature
- Loss of sensory stimuli of oral tissues may occur
- The tongue may be flaccid, with multiple folds, and may deviate on extrusion.
- Dysphagia
- Difficulty in managing liquids and solids.
- Patients with right-sided brain damage may neglect the left side. Thus, food and debris may accumulate around the teeth, beneath the tongue, or in alveolar folds.
- Severe periodontal bone loss is associated with carotid artery plaques and increased risk for stroke.

INR, International normalized ratio; IV, intravenous; PFA, platelet function analyzer; RIND, reversible ischemic neurologic deficits; TIA, transient ischemic attack.

ORAL SURGERY

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Osteomyelitis

Definition

Osteomyelitis is defined as inflammation of bone; clinically, however, the term is synonymous with an "infection of the bone."

Etiology and pathogenesis

Osteomyelitis will usually originate and spread from the medullary spaces of the jaws. Inoculation of bacteria in the marrow spaces usually causes edema of the marrow, and because this space is confined by the cortical bony walls, the hydrostatic pressure rises, similar to that of an infected dental pulp, and once this pressure is greater than that of the feeding arterial vessels, soft tissue necrosis and pain usually ensue. The failure of the microcirculation of the cancellous bones of the jaws is crucial in the development of osteomyelitis, because oxygen and nutrients required for healing are unable to reach the marrow space. In addition, the body's blood-borne immune system has inhibited transport to the marrow space, resulting in proliferation and spread of the offending organism.

Osteomyelitis is more common in the mandible than the maxilla; this is due to the fact that the blood supply to the maxilla is multifocal and robust, which is in contrast with the mandible that primarily obtains its blood supply from the inferior alveolar artery and periosteum. In contrast, the periosteal blood supply to the maxilla penetrates its cortex to perfuse the underlying porous bone much easier than that of the much thicker cortex of the mandible. Though the marrow of the maxilla and mandible are often exposed to periapical pathogens, osteomyelitis is rare. This is because host defenses usually localize the infection to a periapical abscess and limit the progression. However, in those individuals who are immunocompromised, such as those with human immunodeficiency virus infection, who have poorly controlled diabetes mellitus, or who use chronic corticosteroid regimens, as well as those who are chronic drug abusers, who suffer from chronic malnutrition, or who have an immunosuppressive disease, osteomyelitis may ensue due to the lack of intact host defense mechanisms.

Microbiology

Traditionally Staphylococcus species were the predominant bacteria involved as in the other bones of the body, although it is known now that several other organisms may contribute to the disease process. The microbiologic profile most often present in cases of osteomyelitis of the mandible includes Streptococci spp., as well as anaerobic bacteria, such as Bacteroides or Peptostreptococcus. Less commonly seen organisms include Eikenella, Candida, Staphylococcus, Actinomyces, Bacteroides, Klebsiella, Fusobacterium, Lactobacillus, and Haemophilus spp.

Pharmacology

Ideally, specific (not empiric) antimicrobial therapy, based upon culture and sensitivity testing results, is used to prevent the development of bacterial drug resistance as well as adverse side effects. Osteomyelitis of the jaws is often polymicrobial in nature; therefore, culture and sensitivity testing often fail to identify one or more specific offending organisms. Penicillin remains the empiric antibiotic of choice for orofacial infections, followed by clindamycin and fluoroquinolones; these are used until speciation and sensitivity of cultures are performed. These antibiotics are preferred due to their efficacy and coverage for most of the usual odontogenic microbiota.

Classification

1. Acute Suppurative Osteomyelitis

Acute suppurative osteomyelitis is an infection of the medullary bone that also has associated production of purulence. This entity is often seen in osteoradionecrosis (ORN) or medication-related osteonecrosis of the jaws (MRONJ), where microorganisms colonize areas of necrotic bone. A key point here is that the organisms tend to colonize the surface of the bone initially, before they enter the medullary space, which is likely also necrotic in some instances.

Clinical findings: may include edema, restricted movement of the affected area, erythema, and pain. Most patients do not develop systemic manifestations from this process.

Radiological findings: In the acute phase, no radiographic findings are seen because there is minimal bone loss. When there are radiographic findings, destructive lesions are characterized by radiolucency in the involved areas. There is often a moth-eaten appearance to the bone radiographically, which can be confused with malignancy. Within this radiolucency there may be radiopaque areas of bone that have not yet been resorbed by the usual bone turnover mechanisms. These radiopaque areas are termed sequestra, and the surrounding radiolucent area is termed an involucrum.

Treatment: In the early phases, acute supportive osteomyelitis is managed surgically with aggressive debridement of the affected necrotic bone to expose normal bleeding bone, as well as the use of adjunctive empiric antibiotic therapy. The etiology of the infection must also be addressed, and this often is a carious tooth, a failed root canal treatment, or dental implant, or, in the case of ORN or MRONJ, the precipitating necrotic bone. Often, if the disease process has progressed sufficiently, the mandible may fracture in the area of the necrotic bone (pathologic fracture). When possible, this mandible fracture should be reduced and fixated, but due to the poor healing potential in these cases, additional reconstructive procedures may be required.

2. Chronic Suppurative Osteomyelitis

Long-standing, or chronic suppurative osteomyelitis, is treated in a manner similar to the acute form, with removal of the source of the infection. In addition, if the area of involvement has been treated previously with bone grafting or rigid fixation, all nonviable tissue and hardware should be removed. Standard management of chronic suppurative osteomyelitis should include culture and sensitivity testing of a bone biopsy, aggressive debridement of necrotic bone (may include large segments of the jaws), and high dose intravenous antibiotic treatment. High-dose empiric IV antibiotics should be initiated, with more selective antibiotic administration once speciation and sensitivity has been established. The duration of antibiotic administration (generally a minimum of 6 weeks of outpatient IV antibiotics) is longer than used for common odontogenic infections because bone penetration of the antibiotic and resolution of bony

colonization is more difficult. For more chronic, unresponsive types of osteomyelitis, coverage may require up to 6 months or more of IV antibiotic administration to prevent progression of the disease process.

3. Chronic Sclerosing Osteomyelitis

Microbiology: This rare form of osteomyelitis is an intramedullary bone infection with one of the Actinomyces species as well as Eikenella corrodens as the offending organisms. The combination of these two organisms produces a sclerosis and fibrosis of the medullary space.

Clinical presentation: The pathognomonic clinical sign is intense pain. This pain may fluctuate along with acute exacerbations of mandibular expansion and soft tissue edema. Usually a chronic dull pain is always present. In general, there is no purulence of drainage present. Symptoms may persist for up to 5 years before recognition and establishment of a diagnosis.

Radiographical features: Radiographically an increased trabecular bone density is present in the alveolar and basal bone of the mandible.

Treatment: Although antibiotic therapy, combined with or without hyperbaric oxygen therapy, may mitigate the progression of the disease, surgical resection of the diseased bone is often required.

4. Osteomyelitis with Proliferative Periostitis (Garre Osteomyelitis)

Clinical presentation: usually affects children due to their increased vascularity and regenerative capabilities.Clinically there is expansion of the mandible with pain, but no purulence, drainage, or erythema. Though termed by some as periostitis ossificans, this is not an appropriate term because the periosteum does not become ossified. It is actually the chronic infection that causes an inflammation mediated deposition of new bone lifting the periosteum from the cortex.

Radiographical features: The most notable radiographic finding is paracortical bone formation ("onion-skinning") due to repetitive irritation of the periosteum usually associated with a periapical infection of the mandibular tooth. Radiographically extracortical bone formation in the form of woven bone in layers parallel to the cortex connected by bridges of bone perpendicular to the cortex is seen.

Treatment : Removal of the infectious source is of paramount importance, and biopsy is considered when a source of infection is not identified because malignancy may have similar radiographic findings. Routine treatment includes removal of the offending infectious source and, if needed in the acute phase, a short course of antibiotic therapy (penicillin, tetracycline, or clindamycin) until the bone inflammation resolves spontaneously. Long-term antibiotic therapy is not indicated for osteomyelitis with proliferative periostitis (Garre osteomyelitis).

ORAL SURGERY

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Radiation induced osteomyelitis and osteoradionecrosis

Radiation Effects on Bone

One of the most severe and complicating sequelae of radiotherapy for patients with head and neck cancer is osteoradionecrosis. The bone within the radiation beam becomes virtually nonvital from endarteritis, which results in elimination of the fine vasculature within bone. The turnover rate of any remaining viable bone is slowed to the point of being ineffective in self-repair. The continual process of remodeling normally found in bone does not occur, and sharp areas on the alveolar ridge will not smooth themselves, even over considerable time. The bone of the mandible is denser and has a poorer blood supply than that of the maxilla. Thus, the mandible is the part of the jaw most commonly affected by nonhealing ulcerations and osteoradionecrosis.

Osteoradionecrosis

Etiology: Most mucosal breakdown and subsequent osteoradionecrosis occur in the mandible. These conditions occur most often in mandibles that have received radiation in excess of 6500 rad (65 Gy) and do not usually occur in mandibles that have received radiation doses less than 4800 rad (48 Gy).49–51

Treatment: Severe pain may be caused by osteoradionecrosis. The patient should discontinue wearing any prosthesis and try to maintain a good state of oral health. Irrigations should be instituted to remove necrotic debris. Systemic antibiotics are necessary only occasionally because osteoradionecrosis is not an infection of bone but rather a nonhealing hypoxic wound. Because of the decreased vascularity of tissues, systemic antibiotics do not readily gain access to the area to perform the function for which they are intended. However, in acute secondary infections, antibiotics may be useful to help prevent the spread of the infection. Any loose sequestra are removed, but no attempt is made initially to close the soft tissue over exposed bone. Most wounds smaller than 1 cm eventually heal, although this may take weeks to months.

Surgical intervention: For nonhealing wounds or extensive areas of osteoradionecrosis, surgical intervention may be indicated. In this instance, resection of exposed bone and a margin of unexposed bone and primary soft tissue closure can be attempted. This treatment is successful in many cases. Greatly improved results have recently been obtained with the use of HBO therapy in conjunction with surgical intervention. Reconstructive efforts with bone grafts used for continuity defects can also be undertaken successfully in many patients who have undergone irradiation. Free microvascular grafting techniques are becoming more popular for restoring continuity defects in patients who have received radiotherapy. These bone grafts have their own blood supply from a reconnection of blood vessels and are therefore less dependent on the local tissues for incorporation and healing.

Prevention

Before radiotherapy

1- All teeth with a questionable or poor prognosis should be extracted before radiotherapy
2- Patient preparation prior to radiotherapy is similar to patient preparation prior to orthodontic procedures. If an individual cannot or will not care for his or her mouth before the application of the braces, it will be impossible for him or her to do so when faced with future obstacles**.**
3- If the radiotherapist feels that therapy must be instituted urgently, sufficient time may not be available to perform the necessary extractions**,** the dentist may elect to maintain the dentition; but he or she must work closely with the patient throughout the course of radiotherapy and thereafter in an attempt to maintain the patient's oral health as optimally as possible.
4- The more the salivary glands and bone are involved in the field of radiation, the more severe are the resultant xerostomia and vascular compromise of the jaws.
5- Xerostomia of itself may not result in severe problems if the dentition can be maintained because bone is still healthy. The combination of xerostomia and irradiated bone usually causes the problem.
6- The higher the radiation dose, the more severe the damage to normal tissue. Squamous cell carcinomas of the oral cavity make up approximately 90% of malignant tumors for which radiation therapy is used. Unfortunately, this cancer requires a large dose of radiation (>6000 rad [60 Gy]) to affect a result. Other malignancies such as lymphoma require much less radiation for a response, and the oral cavity is therefore less affected.
7- Every tooth to be maintained must be carefully inspected for pathologic conditions and restored to the best state of health obtainable.

During radiotherapy

8- During radiation treatment, the patient should rinse the mouth at least 10 times a day with saline rinses. The patient should be placed on chlorhexidine mouth rinses twice a day to help minimize bacterial and fungal levels within the mouth.
9- The patient's ability to open the mouth should be carefully monitored throughout the course of radiation treatment. Radiation causes progressive fibrosis within the muscles of mastication
10- The combination of mucositis and xerostomia makes oral intake extremely uncomfortable. However, malnutrition causes further difficulties by delaying the healing of oral tissues and giving the patient an overall feeling of illness. In severe cases it may be necessary to feed the patient via nasogastric tube to maintain a reasonable nutritional status.

After radiotherapy

11- the dentist should see the patient every 3 to 4 months. A prophylaxis is performed during these post irradiation visits, and topical fluoride is applied.
12- The patient should be instructed in the use of the costume trays for daily self-administration of topical fluoride applications. The use of a 1% fluoride rinse for 5 minutes each day has been found to decrease the incidence of radiation caries.
13- All patients should also be monitored for the possible onset of trismus. It is easier to prevent trismus than to treat it. The patient should perform mouth-opening exercises with any decrease in the maximum interincisal dimension.

Medication-Related Osteonecrosis of the Jaws

Recently a new oral complication of cancer and osteoporosis treatment that looks similar to osteoradionecrosis, with exposure of devitalized areas of the bone of the jaws, has been identified. However, the complication is seen in patients who have not had any radiation treatment, and the methods used to treat osteoradionecrosis do not seem to be effective for the treatment of these lesions. This oral lesion is called medication-related or, more appropriately, medication-induced, osteonecrosis of the jaws (MRONJ), because what patients with these lesions have in common is that they are taking an antiresorptive medication, usually as an adjunct to chemotherapy for malignant disease.

MRONJ is a condition of chronically exposed necrotic bone; it is usually painful and often primarily or secondarily infected. Bone exposure might occur spontaneously or more commonly following an invasive dental procedure. Patients complain of halitosis and have difficulty eating and speaking. Clinically, the lesions appear as oral mucosal ulcerations that expose the underlying bone and frequently are extremely painful. The lesions are persistent and do not respond to conventional treatment modalities such as debridement, antibiotic therapy, or HBO therapy.

Three main classes of medications that can cause MRONJ, as described below.

1- Bisphosphonates

Bisphosphonates are a class of agents used to treat osteoporosis and malignant bone metastases. Bisphosphonates inhibit bone resorption and thus bone renewal by suppressing the recruitment and activity of osteoclasts, thereby shortening their life span. Millions of postmenopausal women are taking bisphosphonates to stabilize bone loss caused by osteoporosis, thus reducing their risk of pathologic fractures. Besides osteoporosis, bisphosphonates are used to manage Paget disease of bone and hypercalcemia of malignancy. Bisphosphonates are given to patients with cancer to help control bone loss resulting from metastatic skeletal lesions.

Pathophysiology: The mechanism of action of bisphosphonates is that they bind to bone mineral, where they are concentrated and accumulate over time. Bisphosphonates are potent inhibitors of osteoclastic activity, which is why they are usually prescribed.Depending on the duration of the treatment and the specific bisphosphonate prescribed, the drug may remain in the body for years. Physiologic bone deposition and remodeling are severely compromised in patients receiving bisphosphonate therapy. Bisphosphonates also have antiangiogenic properties and may be directly tumoricidal, which makes them an important agent in cancer therapy.

Many bisphosphonate medications are available, some given intravenously (pamidronate, zoledronic acid, clodronate) and some orally (alendronate, etidronate, risedronate, tiludronate, ibandronate). The choice varies with the type of medical condition being treated and the potency of the drug required. For example, orally administered bisphosphonates often are used in patients with osteoporosis, whereas the injectable bisphosphonates are used in patients with cancer who have primary lesions of bone or skeletal metastasis.

2- RANK Ligand Inhibitors

RANK ligand inhibitors (denosumab; Prolia) are antiresorptive agents that exist as a fully humanized antibody against RANK ligand (RANK-L) and inhibits osteoclast function and associated bone resorption. In contrast to bisphosphonates, RANK-L inhibitors do not bind to bone and their effects on bone remodeling are mostly diminished within 6 months of treatment cessation.

3- Antiangiogenic Medications

Angiogenesis inhibitors interfere with the formation of new blood vessels by binding to various signaling molecules disrupting the angiogenesis signaling cascade. These novel medications have demonstrated efficacy in the treatment of various tumors.

Mechanism of Action of Antiresorptive Medications

Of all three medications that can induce MRONJ, bisphosphonates are clearly the most commonly prescribed; therefore, most cases will be caused by this medication. Bisphosphonates, and other antiresorptive such as denosumab, inhibit osteoclast differentiation and function and increase apoptosis, all leading to decreased bone resorption and remodeling. Bisphosphonates bind to bone and are incorporated in the osseous matrix. During bone remodeling, the drug is taken up by osteoclasts and internalized in the cell cytoplasm, where it inhibits osteoclastic function and induces apoptotic cell death. Bisphosphonates also inhibit osteoblast mediated osteoclastic resorption and have antiangiogenic properties. As a result, bone turnover becomes profoundly suppressed, and over time bone shows little physiologic remodeling.

Bone becomes brittle and unable to repair physiologic microfractures that occur in the human skeleton with daily activity. The need for repair and remodeling is greatly increased with infection in the maxilla or the mandible and when an extraction is performed. Therefore, MRONJ results from a complex interplay of bone metabolism, local trauma, increased demand for bone repair, infection, and hypo-vascularity. Patients receiving bisphosphonates intravenously clearly are more susceptible to MRONJ than are those receiving the drug orally. Thus, it is not common to see MRONJ in patients taking bisphosphonates orally for prevention or treatment of osteoporosis; however, beginning in 2006, cases began to be reported in the literature and now number several hundred. Other metabolic factors that may play a role in the development of MRONJ include diabetes mellitus and the concomitant use of steroids, anticancer chemotherapeutic agents, and smoking.

Clinical Signs and Symptoms of MRONJ

Apparently, MRONJ affects the jaws exclusively. The most common clinical presentation associated with MRONJ is an ulcer with exposed bone in a patient who has had a dental extraction. An ulcer caused by an ill-fitting prosthetic device has also been implicated in the initiation of this pathologic process. However, spontaneous bone exposures that cannot be associated with any injury or infection occur in many cases. Similar to osteoradionecrosis, no radiographic manifestations can be seen in the early stages of oral MRONJ. Patients may be asymptomatic but may have severe pain after the necrotic bone is exposed to the oral environment and becomes infected secondarily. The osteonecrosis often is progressive and may lead to extensive areas of bony exposure and dehiscence. In cancer patients taking intravenous forms of bisphosphonates, the median time from starting therapy to developing necrosis of bone in the jaws was reported to be 25 months, although many cases do occur earlier. However, anyone taking intravenous bisphosphonates for over 12 months is at serious risk.

In addition, older adults (>65 years) also may have increased risk. The most common dental comorbidity in these patients reportedly is clinically and radiographically apparent periodontitis. Other local factors associated with MRONJ are infected teeth, dental abscesses, previous endodontic treatments, and tori. In patients in whom MRONJ develops spontaneously, the most common initial complaint is the sudden presence of intraoral discomfort and roughness of the exposed bone, which may progress to traumatize the oral soft tissues surrounding the area of necrotic bone. Often, a purulent discharge and local swelling may

occur in adjacent soft tissue, with trismus and regional lymphadenopathy. One must differentiate MRONJ from simple cases of transient mucosal ulcerations (in patients who have not been taking bisphosphonates) associated with ill-fitting prosthetic appliances, traumatic dental extractions, or spontaneously occurring denudation of bone in areas where the overlying mucosa is thin and prone to abrasion (e.g., mylohyoid ridge and tori). These areas heal spontaneously once the irritation has been removed, but lesions of MRONJ will not.

Dental Care for Patients About to Start Taking an Antiresorptive Medication

Most reports of MRONJ occur after the patient has been taking antiresorptive medications for 6 months or more, so it may be possible to provide dental care early in the treatment without unduly risking the development of MRONJ from dental treatment. Although a small percentage of patients receiving these medications have MRONJ spontaneously, the majority of affected patients experience this complication following routine dentoalveolar surgery (i.e., extraction, dental implant placement, or apical surgery). Therefore,

1- teeth with a poor prognosis should be removed before antiresorptive medication administration or as early as possible after institution of treatment.
2- If possible, institution of antiresorptive therapy should be delayed for approximately 4 to 6 weeks after invasive procedures, such as dental extractions, to give the bone a chance to recover.
3- Dental prophylaxis, caries control, and conservative restorative dentistry are critical to maintaining functionally sound teeth. This level of care must be continued indefinitely.
4- Patients with full or partial dentures should be examined for areas of mucosal trauma, especially along the lingual flange region.
5- It is critical that patients be educated as to the importance of dental hygiene and regular dental evaluations and specifically instructed to report any pain, swelling, or exposed bone that would predict or characterize MRONJ.

Dental Care for Patients Taking Antiresorptive Medications

The treatment of patients receiving oral or intravenous antiresorptives is principally preventive.

1- The dentist should contact the patient's physician to find out why the patient is taking the bisphosphonate, the type of bisphosphonate the patient is taking, and the expected duration of treatment. It is recommended that dentists follow existing guidelines for a dental consultation for the prevention of oral complications of cancer therapy (chemotherapy, radiation therapy).
2- Restorative dentistry should be performed to eliminate caries and defective restorations. Crowns and more extensive fixed prosthodontic work may not be appropriate for some patients. Prosthodontic appliances should be evaluated for fit, stability, and occlusion, and necessary adjustments should be made.
3- Extraction of teeth should be avoided, when possible. The goal of therapy should be to attain a state of good oral health to prevent the need for invasive dental procedures in the future.
4- Prophylaxis should be performed and oral hygiene instructions given. The patient should also be given information about MRONJ and be made aware of the early signs of development of this condition.
5- Once the active dental treatment is over, frequent periodic follow-up visits should be scheduled to reinforce the importance of oral hygiene maintenance and to conduct a new oral examination.

Role of Orally Administered Alendronate

It is unclear whether patients taking alendronate and having MRONJ had other systemic or local comorbid factors. Because of the vast numbers of patients taking alendronate (Fosamax) for osteoporosis (approximately 22 million), a frequently asked question is whether such individuals can safely have invasive procedures such as dental extraction and dental implantation.

The risk of developing MRONJ after dental extraction, dental implantation, and periodontal and other surgical procedures for patients taking oral bisphosphonates such as alendronate is unknown.

The duration of the physiologic effect of these drugs is variable. Evidence shows that severe suppression of bone remodeling may occur during long-term alendronate therapy and that bone resorption and formation markers may remain suppressed for the time during which the patient is taking the medication

At this time, it appears that the incidence of MRONJ manifesting in patients taking alendronate orally for osteoporosis is 1:1000 to 1:25,000. However, the longer a patient takes this medication, the higher the risk for MRONJ.

One thing that can be done when one is contemplating an invasive procedure in a patient taking an oral bisphosphonate is withdrawing the medication for a time (drug holiday). This possibility can be discussed with the patient's physician because it may be possible to use alternative medication. Studies have shown that 6 to 12 months after cessation of an oral bisphosphonate, the development of MRONJ after invasive procedures is reduced.

Dental Care for Patients With MRONJ

For patients with established MRONJ lesions, the goal is to get the patient comfortable, because it is likely the patient will have to live with the exposed bone.

Treatment should be directed at

- eliminating or controlling pain and preventing progression of the exposed bone.
- If the exposed bone has sharp edges that are irritating adjacent soft tissue, the sharp edges of bone may be eliminated with a rotating diamond burr. This is particularly important when the lingual aspect of the posterior mandibular arch is involved. However, superficial debridement should be performed only as a last resort.
- Attempts to cover the exposed bone with flaps may cause more bone exposure and worsening of symptoms, with a risk of pathologic fracture.
- Several treatment modalities for MRONJ are reported in the literature and include minor debridement under local anesthesia, major surgical sequestrectomies, marginal and segmental mandibular resections, partial and complete maxillectomies, and HBO therapy. Unfortunately, none of these therapeutic modalities have proved routinely successful.

Despite the appearance of vascularized bone at the surgical margins, healing may not occur because the entire bone is affected, making it impossible to debride to normal bone. Many cases have a very poor outcome in spite of therapy, progressing to extensive dehiscence and exposure of bone.

- Patients should be closely monitored so as to reevaluate the affected areas and ensure that they have not become suppurative. If the area around the exposed bone exhibits painful erythema,

suppuration, and/or sinus tracts, the patient should be treated with antibiotics until the areas heal.

- Use of chlorhexidine mouth rinse three or four times a day is also recommended to reduce bacterial load and colonization.
- The dentist should discuss the patient's care with the patient's oncologist. Because of the extremely long half-life of bisphosphonates (years), it is not reasonable to discontinue the medication in an attempt to facilitate healing of the MRONJ. Further, patients taking bisphosphonates for metastatic cancer need their medication. However, if no cancer-related indication exists for continued bisphosphonate therapy or the original indication has resolved, it might be reasonable to discontinue the medication, although the drug will be present in the patient's bones for a long time. Discontinuation of oral bisphosphonate therapy in patients with MRONJ has been associated with a gradual improvement in clinical disease. Discontinuation of oral bisphosphonates for 6 to 12 months may result in either spontaneous sequestration or resolution following debridement surgery.
- Routine restorative care may be provided to patients with MRONJ. Local anesthetic may be used as necessary. Scaling and prophylaxis should be done as atraumatically as possible, with gentle soft tissue management. If the tooth is nonrestorable because of caries, root canal treatment and amputation of the crown may be a better option than removing the tooth unless it is very loose.
- Dental extractions should be avoided if possible; if necessary, they should be performed as atraumatically as possible. Patients should be monitored closely for the first several weeks thereafter and then monthly until the sockets are completely closed and healed. If any indication for antibiotic use exists, penicillin V, amoxicillin, or clindamycin may help reduce the incidence of local infection.
- Any existing prosthetic appliances should be reevaluated to ensure that they fit well. Relining the denture with a soft liner to promote a better fit and to minimize soft tissue trauma and pressure points is recommended.
- Odontogenic infections should be treated aggressively with systemic antibiotics. Although penicillin is the first-choice antibiotic in dentistry, amoxicillin, clindamycin, or both provide better bone penetration and a wider spectrum of coverage.
- If debridement, resection, or a combination of both seems necessary, these patients would best be managed by referral to an oral-maxillofacial surgeon

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Hypertension

Hypertension is an abnormal elevation in arterial pressure that can be fatal if sustained and untreated. The blood pressure is the product of cardiac output and peripheral resistance and is dependent on the heart and vasculature, autonomic nervous system, endocrine system and kidneys. In adults, a sustained systolic blood pressure of 140 mmHg or greater and/or a sustained diastolic blood pressure of 90 mmHg or greater is defined as hypertension. The **systolic pressure** is the force that the blood exerts on the artery walls as the heart contracts to pump the blood to the peripheral organs, while the **diastolic pressure** is residual pressure exerted on the arteries as the heart relaxes. The difference between diastolic and systolic pressures is called **pulse pressure**. **Mean arterial pressure** is roughly defined as the sum of the diastolic pressure plus one-third the pulse pressure.

Etiology

Primary/Essential/Idiopathic Hypertension

Primary/essential hypertension is the most common type of hypertension and accounts for about 90% of all cases presenting with high blood pressure with no readily identifiable cause for their disease. This type of hypertension has and a genetic link and is often associated with cardiovascular risk factors, smoking, obesity, lipid problems, and diabetes.

Secondary Hypertension

Secondary hypertension is always due to an underlying cause such as intrinsic renal diseases, renovascular disease, Pheochromocytoma, Cushing's syndrome, thyroid or parathyroid disease, heavy alcohol consumption, chronic corticosteroid therapy, chronic NSAIDS therapy, or long-term oral contraceptive use can lead to secondary hypertension. The secondary hypertension patient experiences symptoms quite early on compared to the primary/essential hypertension patient and the symptoms are more severe. Many patients with secondary hypertension may be cured after treatment of the underlying cause.

Classification of Hypertension Adults and Recommendations for Follow-Up

A recent classification redefines "**normal**" blood pressure as <120/80 mmHg and introduces a new category of "**prehypertension**" (120-139/80-89 mmHg).

TABLE 1 Classification of Blood Pressure (BP) in Adults and Recommendations for Follow-Up

BP Classification	Systolic BP (mm Hg)		Diastolic BP (mm Hg)	Recommended Follow-Up
Normal	<120	and	<80	Recheck in 2 years.
Prehypertension	120–139	or	80–89	Recheck in 1 year.
Stage 1 hypertension	140–159	or	90–99	Confirm within 2 months.
Stage 2 hypertension	≥160	or	≥100	Evaluate or refer to source of care within 1 month. For those with higher BP (e.g., >180/110 mm Hg), evaluate and treat immediately or within 1 week, depending on the clinical situation and complications.

Adapted from the National Heart, Lung, and Blood Institute

Some authors define hypertension as **Mild** when the systolic blood pressure is140-159 mmHg and the diastolic is 90-99 mmHg, **Moderate** when the systolic is 160-179 mmHg and diastolic is 100-109 mmHg and **Severe** when the systolic pressure is ≥180 mmHg and the diastolic is ≥110 mmHg.

Lifestyle risk factors modifying hypertension

Lifestyle can play an important role in the severity and progression of hypertension; obesity, excessive alcohol intake, excessive dietary sodium, and physical inactivity are significant contributing factors.

Clinical features

One third of the hypertensive patients are asymptomatic or only have trivial complications like epistaxis. **Symptoms** include; headaches, visual disturbances, tinnitus, dizziness. **Signs** include; hypertension on testing, retinal changes, left ventricular hypertrophy, proteinuria and hematuria.

Blood pressure is measured with the use of a **sphygmomanometer.** The diagnosis is based on an average of two or more properly measured, seated blood pressure readings on each of two or more office visits.

FIG 1 (**A**) Standard blood pressure cuff (sphygmomanometer) and stethoscope, (**B**) and (**C**) automated blood pressure devices.

The manual technique for recording blood pressure includes the following steps:

1. Alcohol and smoking should be avoided for 30 min before measurement.
2. Allow the patient to sit comfortably seated without the legs crossed for as long as possible (at least 5 minute).
3. Palpate right brachial pulse Before placement of the cuff.
4. Place sphygmomanometer cuff on right upper arm with about 3 cm of skin visible at the antecubital fossa: The standard cuff typically has a mark or arrow that designates the midpoint of the bladder, which is centered above the previously palpated brachial artery.
5. The stethoscope is placed over the previously palpated brachial artery at the bend of the elbow in the antecubital fossa (not touching the cuff)
6. Inflate the cuff slowly to about 200–250 mmHg, or until the pulse is no longer palpable.
7. Deflate cuff slowly while listening with stethoscope over the brachial artery over skin on inside of arm below cuff.
8. Record the systolic pressure as the pressure when the first tapping sounds appear **(Korotkoff sounds).**
9. Deflate cuff further until the tapping sounds become muffled (diastolic pressure) and then disappear.
10. Record blood pressure as systolic/diastolic pressures.

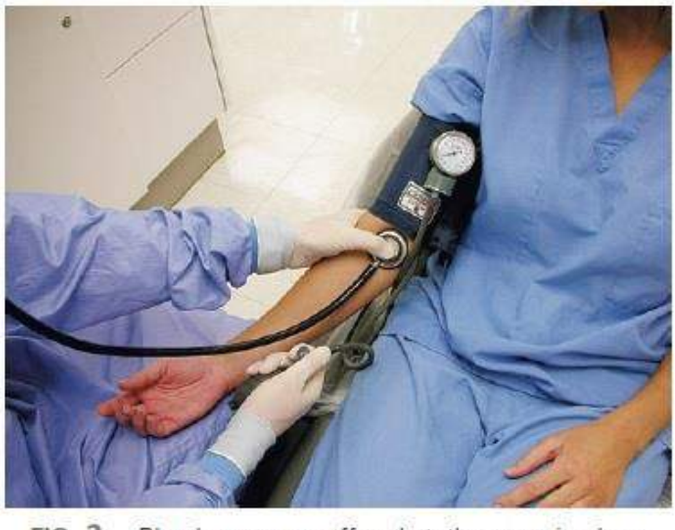


FIG 2 Blood pressure cuff and stethoscope in place.

Management

Hypertension is diagnosed by standardized serial blood pressure measurements. Investigations to identify a 'secondary' cause and assess end-organ damage (also called target organ damage) include: chest radiography (cardiomegaly is suggestive of hypertensive heart disease); ECG (may indicate ischemic heart diseases); serum urea and electrolytes (deranged in hypertensive renal disease and endocrine causes of secondary hypertension); urine testing (blood and protein suggests renal disease).

- Relaxation, weight loss, high-fiber diet, reduction in salt intake, restricting alcohol consumption, restricting caffeine intake, stopping smoking and taking more exercise and avoidance of acute emotions.
- Antihypertensive therapy; the minimum dose should be used with minimum side effects.

Note: About 20% of patients with untreated stage 1 hypertension have what is called **white coat hypertension**, which is defined as consistently elevated BP only in the presence of a health care worker but not elsewhere. In these patients, accurate BP readings may require self-measurement at home or 24-hour ambulatory monitoring. Persons with BP elevation in this setting are at lower risk for hypertensive complications than are those with sustained hypertension.

Antihypertensive agents

- **Diuretics**; these include **Thiazide diuretics** like Chlorothiazide, **Loop diuretics** like Furosemide, **Potassium-sparing diuretics** like Amiloride, **Aldosterone receptor blocker** like Spironolactone, or a **combination** like Aldactazide.
- **Angiotensin-converting enzyme** (ACE) **inhibitors**; like Captopril, Enalapril, ramipril).
- **Angiotensin receptor blockers** (ARBs); like Candesartan and Losartan.
- **Beta-adrenergic blockers**; these are either **Cardioselective** like Atenolol, or **Nonselective** like propranolol.
- **Calcium-channel blockers** (CCBs); like Amlodipine, Nifedipine, Verapamil.
- **Alpha1-adrenergic blockers**; like Doxazosin, Prazosin.
- **Combined alpha and beta blockers**; like Carvedilol.
- **Central alpha2-agonists and centrally acting drugs (Sympatholytics);** like (Clonidine, methyldopa).
- **Vasodilators**; like Hydralazine, and Minoxidil.

Malignant (accelerated) hypertension

Accelerated hypertension (systolic >200 mmHg, diastolic >130 mmHg) typically affects young adults, especially those of African or Afro-Caribbean heritage and, like essential hypertension, often causes no symptoms until complications develop. It may present with headaches, visual impairment, nausea, vomiting, fits (seizures) or acute cardiac failure. The chief complication is severe ischemic damage to the kidneys and renal failure, which can be fatal within 1 year of diagnosis. Other causes of death are cardiac failure or cerebrovascular accidents. Life-threatening accelerated hypertension requires urgent hospital admission with the aim to reduce the blood pressure slowly with oral antihypertensives. Rarely, intravenous antihypertensives (sodium nitroprusside) are used but a sudden drop in blood pressure may result in a stroke (cerebral infarction), thus it should be avoided. Vigorous treatment, if started before renal damage is too far advanced, can greatly improve the life expectancy. About 50% of such patients can now expect to live for at least 5 years.

Dental management

- The first task is to identify patients with hypertension, both diagnosed and undiagnosed. A medical history, including the diagnosis of hypertension, treatment, identification of antihypertensive drugs, compliance of the patient, the presence of symptoms and signs associated with hypertension, and the level of stability of the disease, should be obtained.

- Blood pressure measurements should be routinely performed for all new patients and at recall appointments, also for patients who are not compliant with treatment, who are poorly controlled, or who have comorbid conditions such as heart failure, previous MI, or stroke.

- The main concerns when one is providing dental treatment for a patient with hypertension:
  1. During the course of treatment, the patient might experience an acute elevation in blood pressure that could lead to a serious outcome such as stroke or MI. This acute elevation in blood pressure could result from the release of endogenous catecholamines in response to stress and anxiety, from injection of exogenous catecholamines in the form of vasoconstrictors in the local anesthetic, or from absorption of a vasoconstrictor from the gingival retraction cord.
  2. Potential drug interactions between the patient's antihypertensive medications and the drugs prescribed and oral adverse effects that might be caused by antihypertensive medications.

Hypertension is regarded as a minor clinical predictor of increased perioperative cardiovascular risk.

- Based on blood pressure measurements, the dental management is as follows:

Blood pressure	**Dental management**
≤120/80	Any treatment can be provided.
≧120/80 but < 140/90	Any treatment can be provided but encourage the patient to seek medical consultation.
≥ 140/90 but < 160/100	Any treatment can be provided but encourage the patient to seek medical consultation.
≥ 160/100 but < 180/110	Any treatment; consider intraoperative monitoring of blood pressure for upper level stage 2, treatment should be terminated if blood pressure rises above 179/109. The patient should be referred to physician promptly.
≥ 180/110	Any elective treatment is deferred and the patient is referred to physician. Only emergency treatment is provided; the patient should be managed in consultation with the physician, and measures such as intraoperative blood pressure monitoring, electrocardiogram monitoring, establishment of an intravenous line, and sedation may be used. The decision must always be made as to whether the benefit of proposed treatment outweighs the potential risks.

- **Once it has been determined that the hypertensive patient can be safely treated, the following should be considered:**

- ✓ **Stress/anxiety** reduction.
- ✓ Establishment of **good rapport**.
- ✓ Short, morning appointments.
- ✓ Consider premedication with **sedative/anxiolytic** like Diazepam 2-5 mg the night before surgery and/or 1 hour before surgery
- ✓ Consider the use of **nitrous oxide/oxygen** (conscious sedation), ensure adequate oxygenation at all times, especially at the termination of administration. Hypoxia is to be avoided because of the resultant elevation in blood pressure that may occur.
- ✓ **Slow position changes** to prevent orthostatic hypotension.
- ✓ Consider periodic **intraoperative blood pressure monitoring** for patients with upper level stage 2 hypertension; terminate appointment if blood pressure rises above 179/109.
- ✓ Obtain **excellent local anesthesia**; adrenalin in modest amounts is acceptable. One or two cartridges of 2% lidocaine with 1:100.000 adrenalin are of little clinical significance in most patients with hypertension. Use of more than this amount may well be tolerated but with increasing risk for adverse hemodynamic changes. Intravascular injections should be avoided through the use of **aspirating syringes**.
- ✓ **Avoid the use of adrenalin-impregnated gingival retraction cord** because these cords contain highly concentrated adrenalin, which can be quickly absorbed through the gingival sulcular tissues, resulting in tachycardia and elevated blood pressure.
- ✓ **Noradrenalin and levonordefrin should be avoided** in patients with hypertension because of their comparative excessive alpha1 stimulation.
- ✓ The use of **adrenalin is generally not advised in patients with uncontrolled or severe hypertension**, and indeed. However, if urgent treatment becomes necessary, a decision must be made about the use of adrenalin, which will be dictated by the situation.
- ✓ The other concern is for the adverse **interactions between vasoconstrictors and the nonselective beta-blocking agents** (such as propranolol) or peripheral adrenergic antagonists (such as Reserpine and Guanethidine). Available reports and clinical experience suggest that adrenalin in small doses of one to two cartridges containing 1:100.000 adrenalin can be used safely in most patients.

Oral Manifestations

<u>No oral complications have been associated with hypertension itself.</u>

- Patients with **malignant hypertension** have been reported to occasionally develop facial palsy.
- Patients with **severe hypertension** have been reported to bleed excessively after surgical procedures or trauma.
- Patients who take **antihypertensive drugs**, especially diuretics, may report dry mouth.
- **Lichenoid reactions** have been reported with thiazides, methyldopa, propranolol, and labetalol.
- **ACE inhibitors** may cause neutropenia, resulting in delayed healing or gingival bleeding, non-allergic angioedema and burning mouth.
- All **calcium channel blockers** may cause gingival hyperplasia

Ischemic Heart diseases

Ischemic Heart Disease (IHD) also called **Coronary Artery Disease (CAD**) is the most common and important cardiac disease and it is the most common cause of death. IHD is cause by Atheroma (Atherosclerosis, also called Arteriosclerosis), it is characterized by the accumulation of cholesterol and lipids in the intima of arterial walls, and can lead to thromboses, which sometimes break off and move within the vessels to lodge in and occlude small vessels (embolism). Atheroma can thus lead to IHD with angina, myocardial infarction, cerebrovascular disease and stroke. It also affects other arteries and can cause, for example, ischemic pain in the calves whilst walking – intermittent claudication – seen especially in young smokers. Atheroma results from a combination of genetic and lifestyle factors**.**

- **Irreversible (fixed) risk factors** include increasing age, male gender and family history of atheroma.
- **Potentially reversible risk factors** for atheroma include:
  - Cigarette smoking. Persons who smoke 20 or more cigarettes daily have a two or threefold increase in coronary artery diseases.
  - Blood lipids.
  - Hypertension.
  - Diabetes mellitus. There is a two-eightfold higher rate of cardiovascular events.
  - Obesity and lack of exercise.

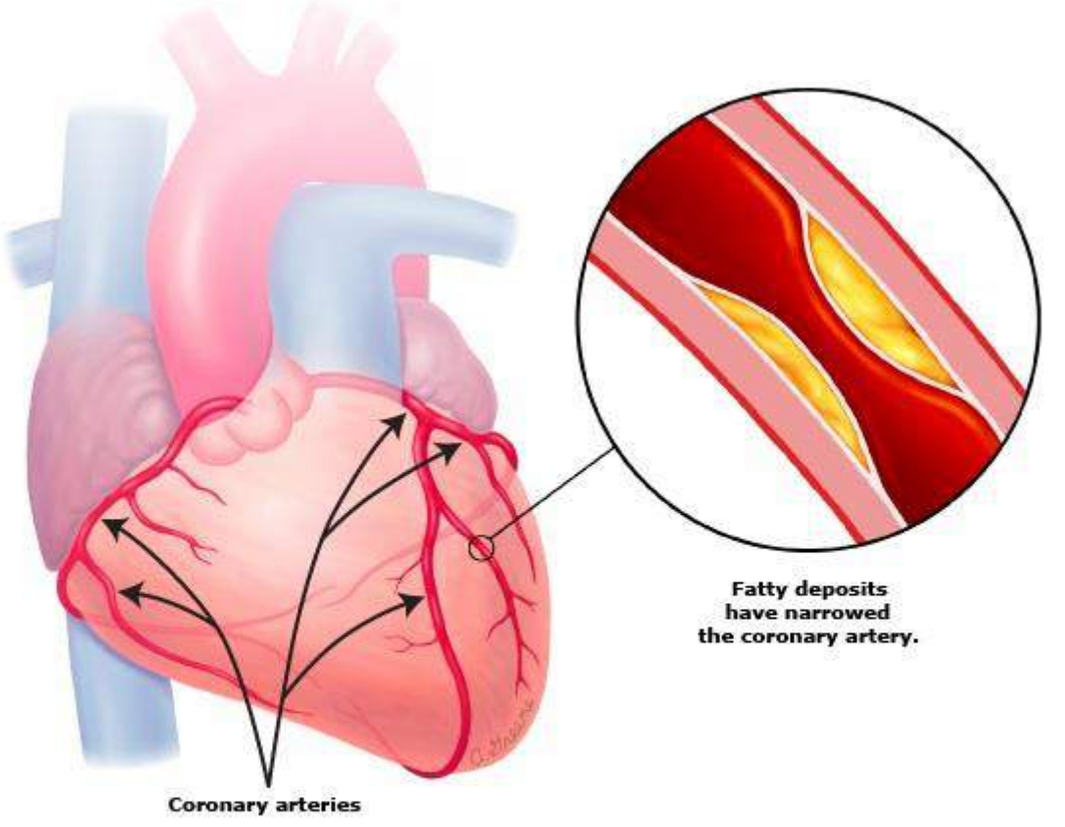


Fig. 3 Atherosclerosis

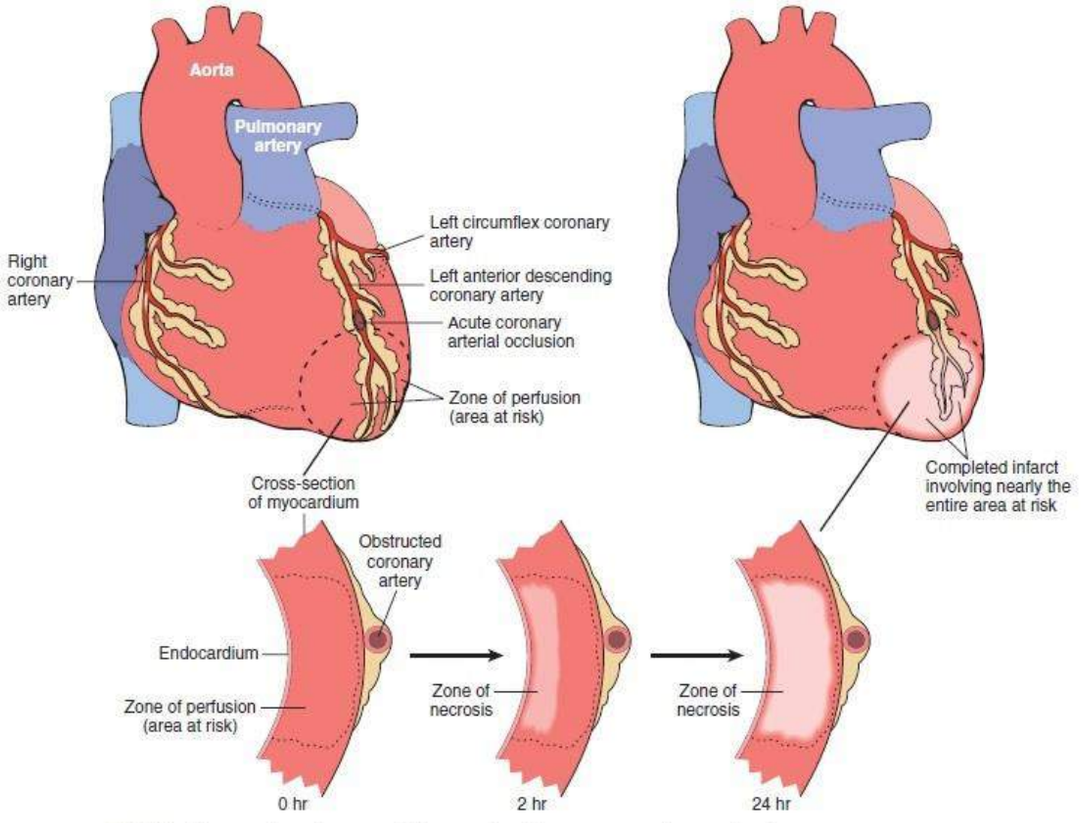


FIG 4 Progression of myocardial necrosis after coronary artery occlusion.

Angina pectoris

Episodes of chest pain caused by myocardial ischemia, it affects 1% of the adult population and its prevalence increases with age. The usual underlying causes are atherosclerotic plaques that rupture. Arterial spasm alone may, rarely, be responsible. The mortality rate in angina is about 4% per year, the prognosis depending on the degree of coronary artery narrowing. **The most common precipitating causes of angina pain** are physical exertion (particularly in cold weather); emotion (especially anger or anxiety); and stress caused by fear or pain, leading to adrenal release of catecholamines (epinephrine and norepinephrine) and consequent tachycardia, vasoconstriction and raised blood pressure. Consequently, an increased cardiac workload is accompanied by a paradoxical drop in blood flow and myocardial ischemia occurs resulting in angina.

Clinical features of angina

- Chest pain described as a pressure sensation, fullness or squeezing in the mid-portion of the thorax, the pain lasts for less than 10-15 minutes about 2-5 minutes in most cases, relieved by rest or glyceryl trinitrate.
- Radiation of chest pain into the jaw/teeth, shoulder, arm, and/or back.
- Occasionally associated dyspnea or shortness of breath, epigastric discomfort or sweating.

Types of angina

- **Stable angina:** induced by effort, stress or sometimes eating, it is relieved by rest or Nitroglycerin.
- **Unstable angina**: (crescendo) angina of increasing frequency or severity, occurs on minimal exertion or at rest, the pain is not readily relieved by Nitroglycerin, there is increased risk of MI.
- **Decubitus angina**: precipitated by lying down.
- **Variant or Prinzmetal's angina**: caused by coronary artery spasm.

The term acute coronary syndromes (ACS) are a term used in unstable angina and evolving MI which share a common underlying pathology; plaque rupture, thrombosis and inflammation.

Tests

Resting ECG; ST depression, flat or inverted T wave.

- ✓ Exercise ECG.
- ✓ Thallium-201 scan; highlights ischemic myocardium.
- ✓ ambulatory (Holter) electrocardiography and exercise echocardiography,
- ✓ Coronary angiography.

Management

- ❖ Identify and correct risk factors; stop smoking, encourage exercise and weight loss, control hypertension and diabetes.
- ❖ Antiplatelet drugs like spirin 75-325 mg/24 h and\or clopidogrel.
- ❖ β blockers; atenolol 50-100 mg/24h, unless contraindicated (asthma, COPD, bradycardia, coronary artery spasm).
- ❖ Nitrates; for symptoms give glyceryl trinitrate (GTN) spray or sublingual tablets 0.3 mg to reduce the peripheral vascular resistance and reduce oxygen demands. Also long acting nitrates isosorbide mononitrate 20-40 mg twice daily or slow-release nitrate.
- ❖ Calcium antagonists; amlodipine, diltiazem, especially when β-blockers are contraindicated.
- ❖ Potassium channel activator; nicorandil.
- ❖ Percutaneous transluminal coronary angioplasty (PTCA); aims to open up the coronary blood flow by inserting a balloon- tipped catheter through the groin up into the area of arterial blockage.
- ❖ Coronary artery bypass grafts (CABG) are vascular grafts made to bridge the obstructions in the coronary blood vessels.

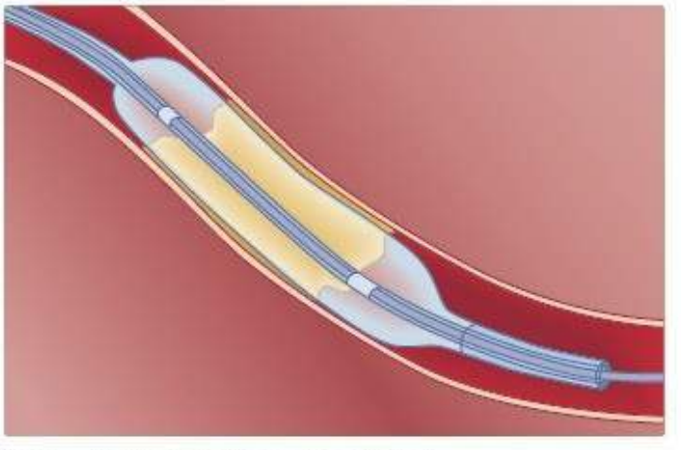
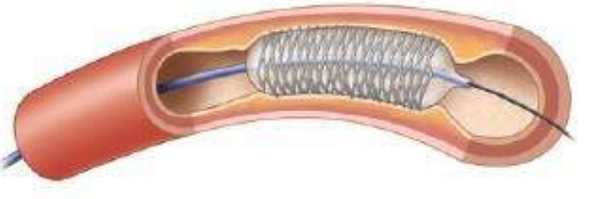


FIG .5 Balloon angioplasty catheter.



Expandable metallic stent. The stent is left in place after deflation and withdrawal of the balloon catheter.

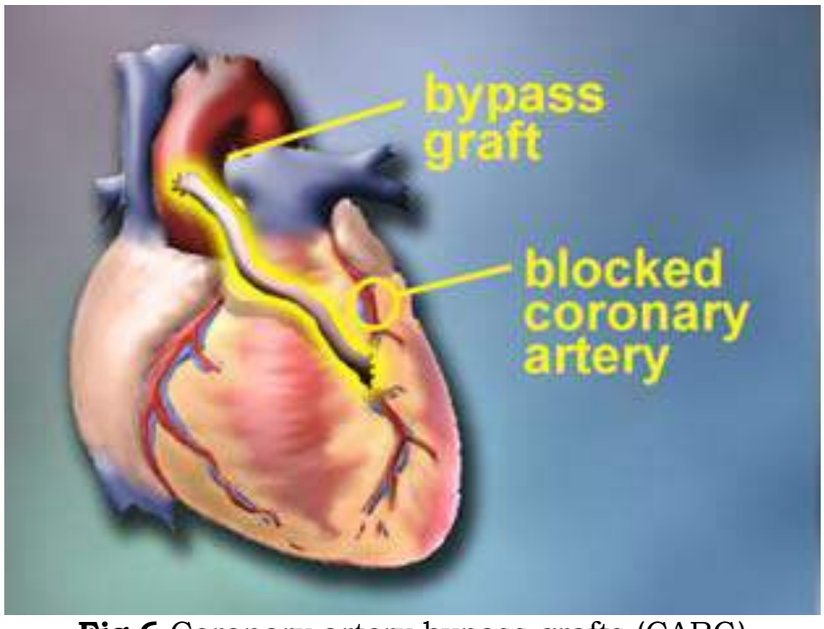


Fig.6 Coronary artery bypass grafts (CABG)

Myocardial Infarction (MI)

Myocardial infarction results from the complete occlusion (blockage) of one or more coronary arteries. It arises when atherosclerotic plaques rupture causing platelet activation, adhesion and aggregation with subsequent thrombus formation within the coronary circulation. Angina may progress to MI but fewer than 50% of patients with MI have any preceding symptoms. **Complications** of MI include weakened heart muscle, resulting in acute congestive heart failure, post-infarction angina, infarct extension, cardiogenic shock, pericarditis, and arrhythmias. Causes of death in patients who have had an acute MI include ventricular fibrillation, cardiac standstill, congestive heart failure, embolism, and rupture of the heart wall or septum.

Clinical features of myocardial infarction

- Chest pain described as a pressure sensation, fullness or squeezing in the mid-portion of the thorax. 10-20% of individuals have silent (painless) MI.
- Radiation of chest pain into the jaw/teeth, shoulder, arm, and/or back.
- Associated dyspnea or shortness of breath.
- Associated epigastric discomfort with or without nausea and vomiting.
- Associated diaphoresis or sweating.
- Syncope or near-syncope without other cause.
- Impairment of cognitive function without other cause.

50% of the patients die within the first hour of MI and a further 10-20% within the next few days.

Diagnosis of MI; is by clinical features, ECG (large Q wave, ST elevation and T inversion), change in the serum levels of cardiac enzymes which include; Troponin T (TT), Creatine Kinase MB (CK-MB), Aspartate transaminase (AST) and Lactic dehydrogenase (LDH).

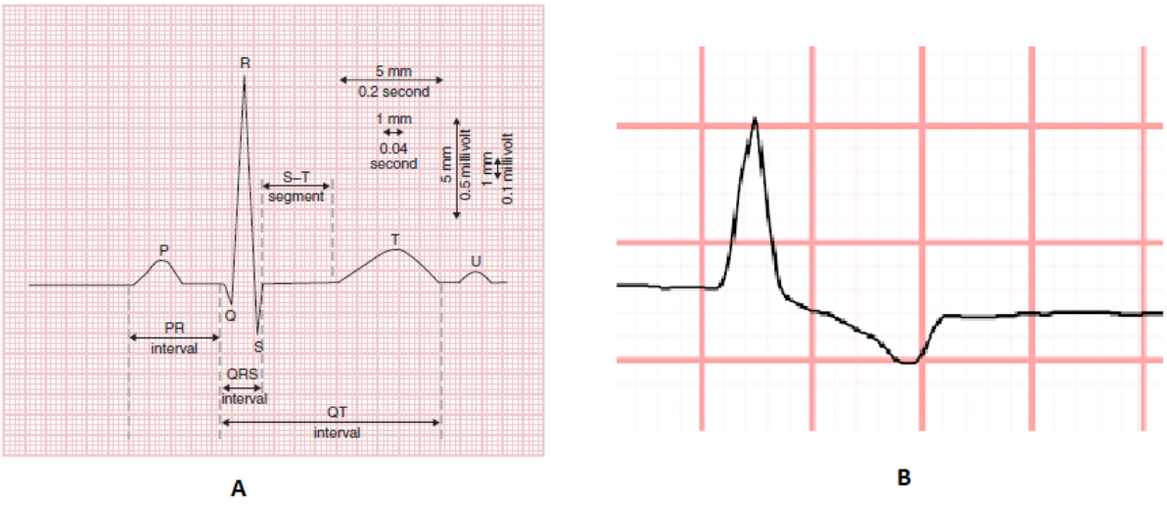


Fig.7 A. Normal ECG, **B.** ECG in Pt with angina or MI

Management

- Alert **emergency services** (if in community) or cardiac arrest team (if in hospital).
- **Aspirin** (300 mg); to be chewed. **Clopidogrel.**
- **Rest** and reassure.
- **Pain relief:** opioid analgesia (diamorphine) is usually necessary.
- **Oxygen** administration.
- Early **thrombolytic therapy** (Streptokinase, Urokinase, Alteplase or Reteplase), the greatest benefit is realized when patients receive thrombolytic drugs within the first 3 hours after infarction; however, modest benefit is possible even up to 12 hours after the event. The early use of thrombolytic drugs may decrease the extent of necrosis and myocardial damage and dramatically improve outcome and prognosis.
- **Primary percutaneous intervention** to dissolve the coronary thrombus provided the patient is not at risk of a life-threatening hemorrhage.
- **Insulin** infusion to prevent stress hyperglycemia.
- General **pharmacologic measures** for patients with acute MI include the use of nitrates, beta blockers, calcium channel blockers, ACE inhibitors, and lipid-lowering agents. Antiplatelet drugs are significant in decreasing morbidity and mortality.
- **Sedatives and anxiolytic** medications also may be used.
- Prompt **treatment of complications**, particularly cardiac arrhythmias. During the first several weeks after an infarction, the conduction system of the heart may be unstable, and patients are prone to serious arrhythmias and re-infarction.

Dental management

- Identification of patients with a history of ischemic heart diseases, and if the patient is not under medical control we have to refer him to physician for an evaluation and control.
- The potential problem related to the dental procedure is that, the stress and anxiety during the dental procedure, and excessive amount of vasoconstrictor in local anesthesia may precipitate the attack of angina pectoris, MI, arrhythmia, or sudden death.

- In general, recent MI and unstable angina are classified as clinical predictors of **major risk** for perioperative complications like serious arrhythmias and re-infarction. Stable (mild) angina and past history of MI are identified as clinical predictors of **intermediate risk** for perioperative complications.

Patients with stable angina or past history of MI (more than 6 months) [Intermediate risk]

- Short Morning appointment with comfortable chair position is recommended.
- Pretreatment vital signs should be recorded including pulse rate and blood pressure as a base line record.
- Nitroglycerin sublingual tablets should be readily available and in some cases prophylactic preoperative nitroglycerin is advisable especially when the patient has angina more than once a week.
- Stress-reduction measures which include:
  - ✓ Good communication
  - ✓ Oral sedation (e.g., Diazepam 2-5 mg on the night before and 1 hour before the appointment)
  - ✓ Intraoperative Nitrous oxide and oxygen but hypoxia should be avoided.
  - ✓ Excellent local anesthesia
- Limited use of vasoconstrictor (maximum 0.036 mg adrenalin, 0.20 mg levonordefrine), not more than two cartridges containing 1: 100.000 adrenalin or 1:20.000 levonordefrine, this is also applicable if patient is taking a nonselective beta-blocker. Avoid intravascular injections through the use of aspirating syringe.
- Avoidance of adrenalin-impregnated retraction cord
- Antibiotic prophylaxis not recommended for history of coronary artery bypass graft (CABG).
- Avoidance of anticholinergics (e.g., scopolamine, and atropine)
- Adequate postoperative pain control.
- For patients with coronary artery stents, elective dental care should be deferred for 6 months; emergency dental care should be in a hospital setting. It may be prudent to provide antibiotic coverage if emergency dental treatment is required during the first six weeks postoperatively. Patients may require long-term anticoagulant medication, but most patients are on aspirin or clopidogrel rather than warfarin.
- If a patient experiences chest pain in the dental surgery, dental treatment must be stopped, the patient should be given nitroglycerine sublingually and oxygen, and be kept sitting upright. Vital signs should be monitored. The pain should be relieved in 2–3 min; the patient should then rest and be accompanied home. If chest pain persists after three doses of nitroglycerin given every 5 min, that lasts more than 15–20 min, or that is associated with nausea, vomiting, syncope or hypertension is highly suggestive of MI, oxygen should be continued, and 300 mg of aspirin should be chewed.

Patients with unstable angina or recent MI (less than 6 months) [Major risk]

- Avoid elective care, only emergency treatment should be provided, consult with physician and limit treatment to pain relief, treatment of acute infection, or control of bleeding.
- Prophylactic nitroglycerin sublingually is advisable.
- Placement of intravenous line.
- Sedation and oxygen.
- Frequent monitoring of blood pressure and vital signs, using a pulse oximeter and continuous electrocardiographic monitoring
- Cautious use of adrenalin in local anesthetic, local anesthesia without adrenalin can be used or not more than two cartridges containing 1: 100.000 adrenalin or 1:20.000 levonordefrine using aspirating syringes.
- In general, conscious sedation and general anesthesia should be deferred for at least 3 months in patients with recent-onset angina, unstable angina or recent development of bundle branch block and, in any case, it should be given in hospital.
- Adrenalin-impregnated retraction cords are avoided.
- If the patient experiences pain during treatment, the management is the same as above.

The use of antiplatelet and anticoagulation drugs

Patients who take **aspirin or another platelet aggregation antagonist** such as clopidogrel can expect some increase in bleeding. This effect generally is not clinically significant, and bleeding may be controlled through local measures. Discontinuation of these agents before dental treatment generally is unnecessary.

Patients who take **warfarin** for anticoagulation must have a current international normalized ratio (INR) determined before any invasive procedure and they are managed after consultation with physician.

The Local hemostatic measures that are used to control bleeding include the use of hemostatic agents in the sockets (gelfoam, surgicel), suturing and gauze pressure packs.

د.ياسر رياض الخناق

Heart failure

Heart failure (HF), also known as congestive heart failure (CHF), defined by (ACC\AHA) as a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood which leads to blood output insufficient to meet the body requirement.
Prevalence is 1-3% of the general population and 10% among the elderly population. The prognosis is poor, 25-50% of the patients die within 5 years.

Causes

Heart failure is a symptom complex that is caused by many diseases such as; ischemic heart diseases (the most common cause), congenital heart diseases, hypertension, pulmonary hypertension, pulmonary embolism, myocarditis, infective endocarditis, cardiomyopathies, valvular heart diseases, endocrine diseases, chronic anemia and arrhythmias.

Classification

The American Heart Association and the American College of Cardiology (AHA/ACC) classify heart failure into 4 stages:(reflecting the fact that HF is a *progressive disease and whose outcome can be modified* by early identification and treatment).

- **Stage A:** patients with risk factors that predispose to HF but with no left ventricular hypertrophy or dysfunction (structural heart disease).
- **Stage B:** patients with risk factors that predispose to HF with left ventricular hypertrophy or dysfunction but with no symptoms.
- **Stage C:** patients with past or present symptoms of HF with structural heart disease.
- **Stage D:** patients with refractory HF who require specialized care.

Based on *severity of symptoms and the amount of effort needed to elicit symptoms,* another classification was developed by the New York Heart Association (NYHA); it is complementary to the previous system.

- ✓ **Class I:** No limitation of physical activity, no signs or symptoms with ordinary activity.
- ✓ **Class II:** Slight limitation of the physical activity but the patients remains comfortable at rest.
- ✓ **Class III:** Marked limitation of activity but the patients are comfortable at rest.
- ✓ **Class IV:** Symptoms are present at rest and physical activity exacerbates the symptoms.

The term **compensated** HF is used when neurohumoral responses eliminate the symptoms while the symptomatic HF is termed as **decompensated** HF.

Signs and Symptoms

- Dyspnea (perceived shortness of breath).
- Fatigue and weakness (especially muscular).
- Orthopnea (dyspnea in recumbent position)
- Paroxysmal nocturnal dyspnea (dyspnea that awakens patient from sleep)
- Acute pulmonary edema (cough or progressive dyspnea)
- Exercise intolerance (inability to climb a flight of stairs)
- Dependent peripheral edema (swelling of feet and ankles after standing or walking)
- Report of weight gain or increased abdominal girth (fluid accumulation; ascites)
- Right upper quadrant pain (liver congestion)
- Anorexia, nausea, vomiting, constipation (bowel edema)
- Cheyne-Stokes respiration (hyperventilation alternating with apnea during sleep)
- Heart murmur**.**
- Increased venous pressure.
- Enlargement of cardiac silhouette on chest radiograph
- Pulsus alternans; a regular rhythm with alternating strong and weak ventricular contractions.
- Distended neck veins.
- Cyanosis
- Clubbing of fingers

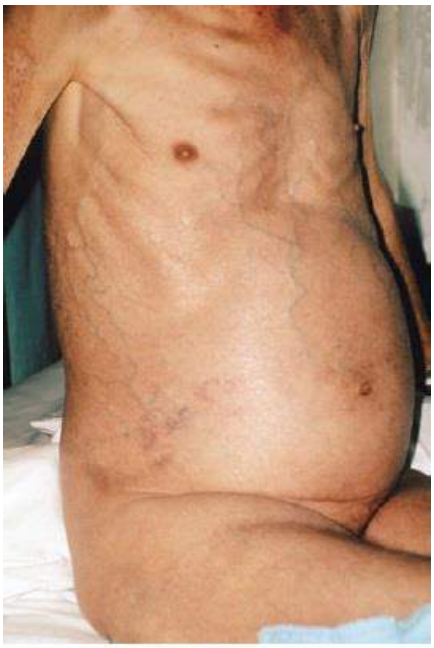


Fig 1 Ascites.

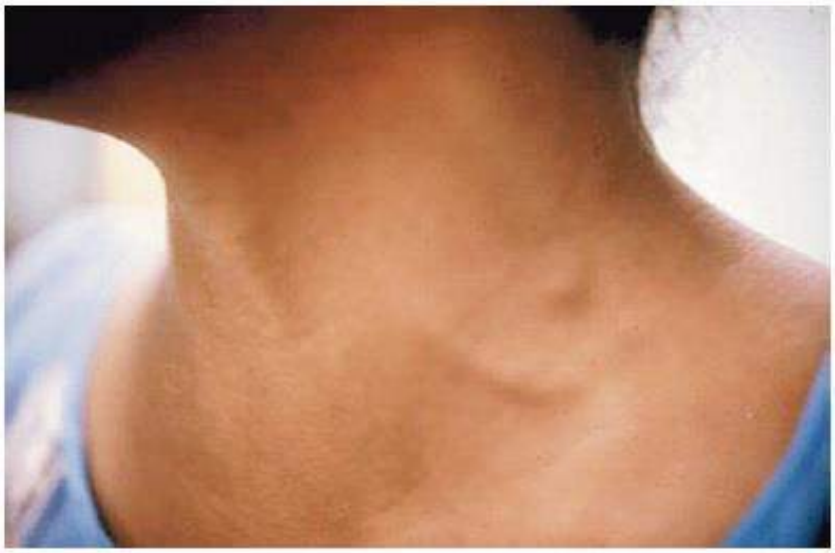


Fig.2 Distended jugular vein in a patient with heart failure.

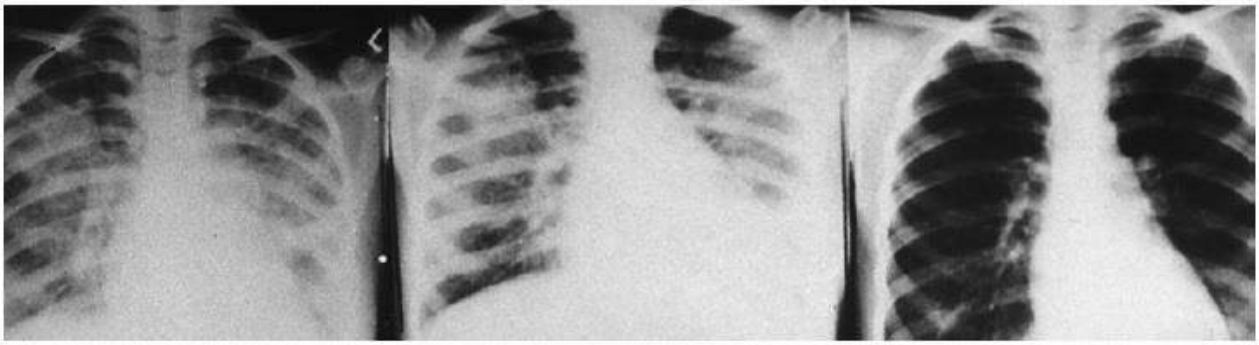


Fig. 3 Serial chest radiographs demonstrating the resolution of pulmonary edema (*left* to *right*).

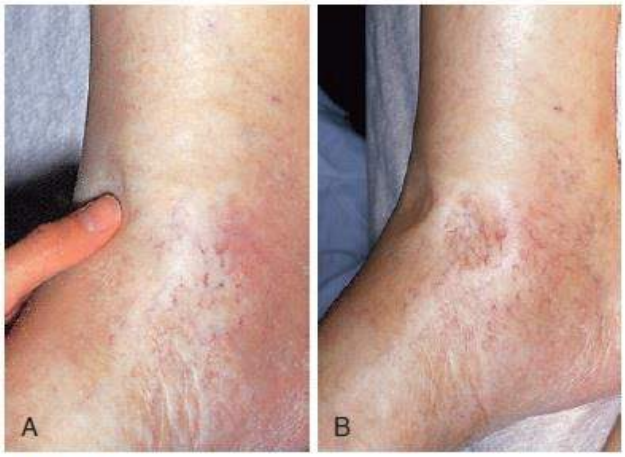


Fig. 4 **A** and **B**, Pitting edema in a patient with heart failure. A depression ("pit") remains in the edematous tissue for some minutes after firm fingertip pressure is applied.

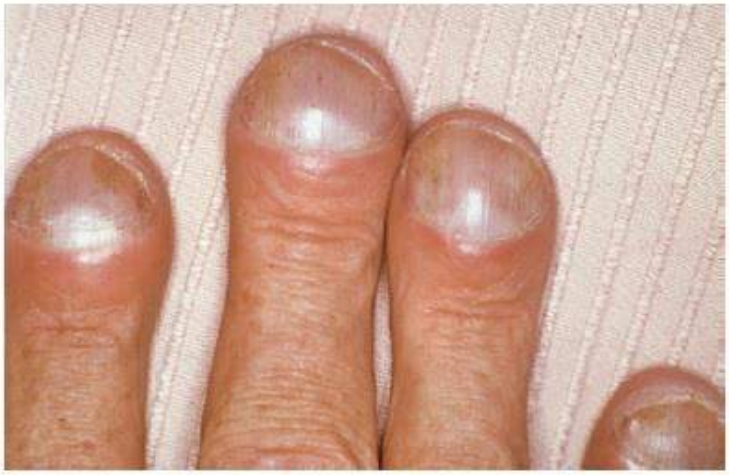


Fig 5 Clubbing of the fingers in a patient with congestive heart failure.

Diagnosis

Heart failure is diagnosed clinically and by chest radiography (cardiomegaly), echocardiography, ECG and biochemistry. Echocardiography determines the **stroke volume** (SV; the amount of blood that exits the ventricles with each heartbeat), the **end-diastolic volume** (EDV; the amount of blood at the end of diastole), and the SV in proportion to the EDV (the **ejection fraction**; EF). Normally, the EF should lie between 50 and 70% but, in cardiac failure, it is < 40%.

Management

The management depends on the stage (NYHA) of the disease but the general lines of treatment are:

- General measures; rest, control of stress, salt restriction and controlling hypertension, anemia or any underlying causes.
- Angiotensin-converting enzyme inhibitors ACE-I; like enalapril and lisinopril.
- Angiotensin II receptor blockers; like losartan and valsartan.
- Vasodilators like isosorbide dinitrate plus hydralazine.
- Diuretics like furosemide and spironolactone.
- Digoxin may be helpful when failure is associated with atrial fibrillation.
- Supplemental oxygen may be required.
- Heart transplantation.

Dental management

The risk of treating a patient with symptomatic heart failure is that symptoms could abruptly worsen and result in acute failure, a fatal arrhythmia, stroke, or myocardial infarction.

- **Identification** of patients with a history of heart failure, those with undiagnosed heart failure, or those prone to developing heart failure is the first step in risk assessment, this is accomplished by obtaining a thorough medical history, including a pertinent review of systems, and measuring and evaluating vital signs (i.e., pulse rate and rhythm, blood pressure, respiratory rate).
- For patients with symptoms of **untreated or uncontrolled heart failure**, defer elective dental care and refer to physician.
- For **patients diagnosed and treated for heart failure**:
- ✓ Confirm status with patient or physician
- ✓ Identify underlying cardiovascular disease (i.e., coronary artery disease, hypertension, cardiomyopathy, valvular disease), and manage appropriately.
- ✓ New York Heart Association (NYHA) class I patients (asymptomatic), routine care can be provided.
- ✓ NYHA class II (and some class III patients), obtain consultation with physician for medical clearance and provide routine care.
- ✓ NYHA (some class III and class IV) patients obtain consultation with physician; consider treatment in a special care or hospital setting.
- **Drug considerations**:
- ✓ For patients taking digitalis, avoid adrenalin; if considered essential, use cautiously (maximum 0.036 mg adrenalin or 0.20 mg levonordefrin), which is no more than 2 cartridges containing 1:100.000 adrenalin or 1: 20.000 levonordefrine with care to avoid intravascular injection; avoid gag reflex; avoid erythromycin and clarithromycin, which may increase the absorption of digitalis and lead to toxicity.
- ✓ For patients with NYHA class III and IV congestive heart failure, avoid use of vasoconstrictors; if use is considered essential, discuss with physician.
- ✓ Avoid adrenalin-impregnated retraction cord.
- Schedule **short, stress-free** appointments.
- Use **semisupine or upright** chair position.
- Watch for **orthostatic hypotension**, make position or chair changes slowly, and assist patient into and out of chair.
- **Avoid the use of nonsteroidal antiinflammatory drugs** (NSAIDs) because they can exacerbate symptoms of heart failure.
- Watch for **signs of digitalis toxicity** (tachycardia, hypersalivation, visual disturbances) which if it occurs the patient must be referred to physician promptly.
- **Nitrous oxide/oxygen sedation** may be used with a minimum of 30% oxygen.
- The dentist should be **aware** that even these HF patients with NYHA **class I should not be considered "mild"** because they indeed could be decompensated during dental treatment.

Oral manifestations

No oral manifestations are related to heart failure but some drugs can cause:

- **Dry mouth** in patients taking diuretics or vasodilators.
- **Angioedema** of lip, face, or tongue, taste changes, burning mouth in patients taking ACE inhibitors.
- **Lichenoid reactions** in patients taking ACE inhibitors and Beta blockers.
- **Increased gag reflex and hypersalivation** in patients taking Digitalis.
- **Lupus like lesions** and lymphadenopathy in patients taking vasodilators.

Cardiac Arrhythmias

Arrhythmia is simply defined as disturbance of heartbeat including disturbance rhythm, rate or conduction pattern of the heart, in which there is abnormal electrical activity in the heart. 15-17% of the population may have arrhythmias and the prevalence increases with age. It has been shown that potentially fatal arrhythmias can be precipitated by strong emotion such as anxiety or anger, as well as by various drugs, both of which can be precipitated by dental treatment.

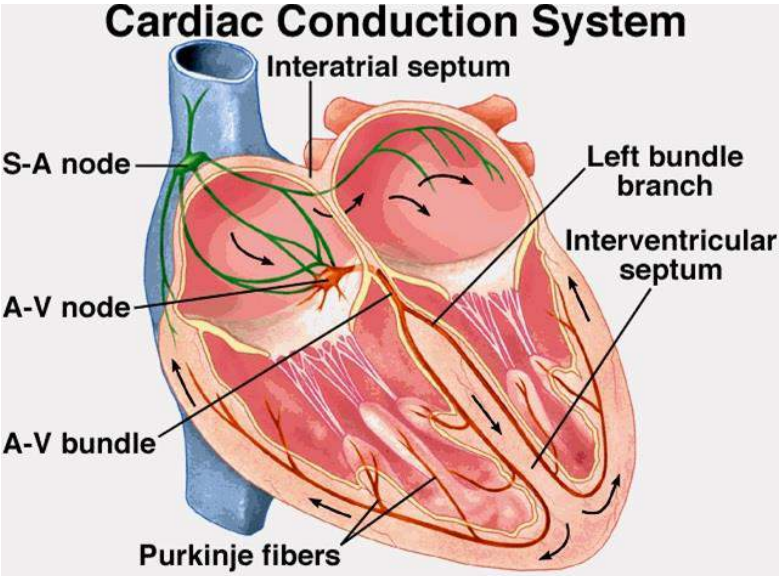


Fig. 6 Cardiac Conducting system

Causes; are broadly classified as:

- **Cardiac**; as in MI, mitral valve diseases, cardiomyopathy, pericarditis, or aberrant conduction pathways.
- **Non-cardiac**; caffeine, smoking, alcohol, fever, respiratory, autonomic, endocrine diseases, hypoxia or electrolyte disturbances. Surgery is sometimes implicated.

Classification

They are classified according to:

- ✓ **Rate** into: tachycardia and bradycardia.
- ✓ **Mechanism** into: automaticity, re-entry and fibrillation.
- ✓ **Site of origin** into: supraventricular and ventricular arrhythmias.

Clinical features

Signs include; slow (less than 60 beat/min) or fast (more than 100 beat/min heart rate, irregular rhythm.
Symptoms include; palpitation, fatigue, dizziness, syncope, angina pectoris, dyspnea and those related to congestive heart failure (e.g., Shortness of breath, Orthopnea, Peripheral edema).
The primary tool for diagnosis of arrhythmia is electrocardiogram (ECG).

Medical management

- **Physical maneuvers**. In supraventricular arrhythmias, pressure on the neck may increase parasympathetic stimulation to the heart inhibiting electrical conduction through the AV nodes.
- **Antiarrhythmic drugs**; these are divided into 4 classes: *class I* are sodium channel blockers, *class II* drugs are beta blockers, *class III* drugs act on potassium channels and prolong the duration of action potential, while *class IV* drugs are calcium channel blockers.
- **Oral Anticoagulant (OAC) Therapy:** Patients who have AF are at increased risk for stroke and thromboembolism. To reduce this risk, the American Heart Association (AHA) recommends OAC therapy.
- **Defibrillation or cardioversion**:
- **Pacemakers**. Which is a subcutaneously implanted generator in the left infraclavicular area, it produces an electrical impulse that is transmitted by a lead inserted into the heart via subclavian vein to an electrode in contact with endocardial or myocardial tissue.
- **Implanted cardioverter-defibrillator** (ICD) which is similar to pacemaker. Both are subject to electromagnetic interferences (EMI). ICDs are capable not only of delivering a shock but also of providing antitachycardia pacing (ATP) and ventricular bradycardia pacing.
- **Radiofrequency catheter ablation**. In which a catheter is introduced through the vein to the area which is the source of arrhythmia, radiofrequency energy is then delivered that cause irreversible tissue destruction.
- **Surgery.**
- **Anticoagulants.**

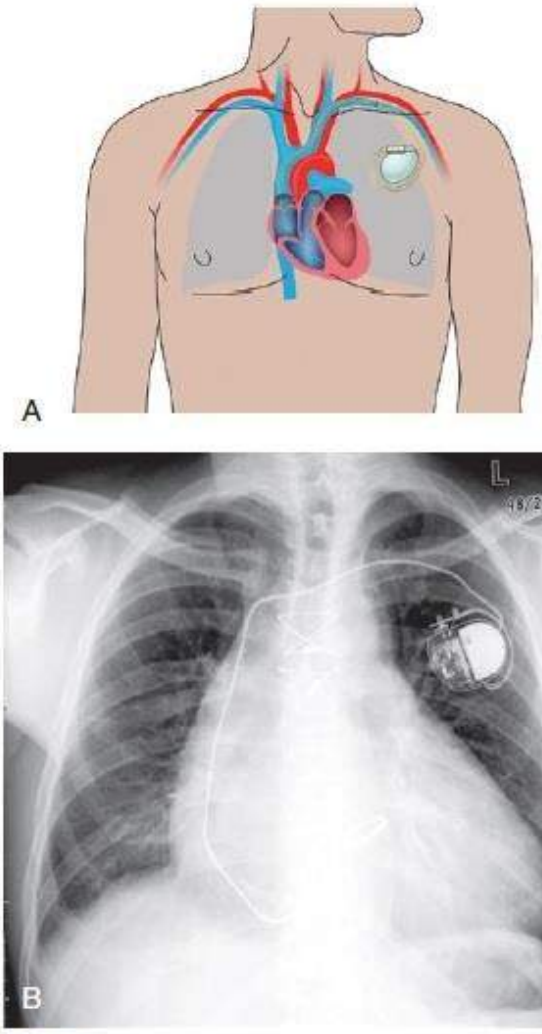


Fig. 7 **A,** The site of implantation of a permanent pacemaker (note: can be inserted in the left or right intraclavicular chest wall). **B,** A chest radiograph showing a pacemaker in a patient. *LV,* Left ventricular.

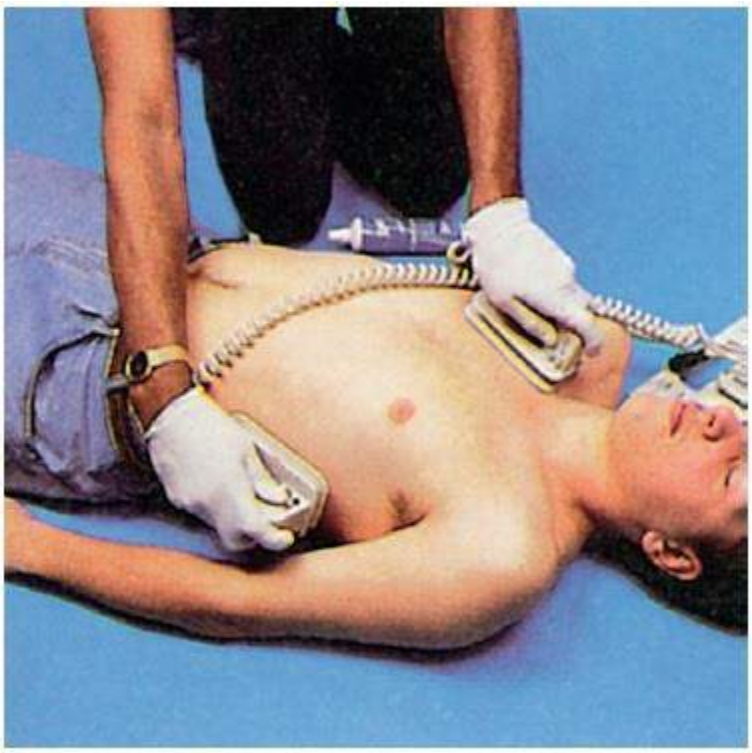


Fig. 8 Cardioversion/defibrillation paddles in place on a patient.

Dental management

Stress associated with dental treatment or excessive amounts of injected adrenalin may lead to life threatening cardiac arrhythmias in susceptible dental patients. The keys to successful dental management of patients prone to developing a cardiac arrhythmia and those with an existing arrhythmia are **identification and prevention.**

- Patients with cardiac arrhythmias may be **identified by** the following:
  - ✓ Medical history to identify: type of arrhythmia, treatment, presence of pacemaker or defibrillator and stability. The dentist may need to consult with physician to obtain or verify this information
  - ✓ Risk for arrhythmia is increased in the presence of other cardiovascular or pulmonary disease
  - ✓ Patient does not report an arrhythmia, but may be taking one or more of the antiarrhythmic drugs
  - ✓ The presence of symptoms that could be caused by arrhythmias.
  - ✓ Vital signs are suggestive of arrhythmia (rapid pulse rate, slow pulse rate, irregular pulse)

Refer patient to physician if signs or symptoms are present that are suggestive of a cardiac arrhythmia or other cardiovascular disease

- Cardiac arrhythmias that may be associated with **major perioperative risk** during dental treatment include:
  - ❖ High-grade atrioventricular (AV) block.
  - ❖ Symptomatic ventricular arrhythmias in the presence of underlying heart disease.
  - ❖ Supraventricular arrhythmias with uncontrolled ventricular rate.

Elective dental treatment is avoided in such cases, only urgent care is provided and preferably in hospital, the following should be considered:

- ✓ Consult with physician.
- ✓ Provide limited care only for pain control, treatment of acute infection, or control of bleeding.
- ✓ Intravenous line.
- ✓ Sedation
- ✓ Electrocardiogram (ECG) monitoring
- ✓ Pulse oximeter
- ✓ Blood pressure monitoring
- ✓ Avoid or limit adrenalin.

Other types of cardiac arrhythmias are associated with **intermediate or minor perioperative risk** during dental treatment in such cases elective dental treatment is allowed.

- Stress and anxiety reduction
  - ✓ Establish good rapport.
  - ✓ Schedule short, morning appointments.

- ✓ Ensure comfortable chair position.
- ✓ Provide preoperative sedation (short-acting benzodiazepine night before and/or 1 hour before appointment).
- ✓ Administer intraoperative sedation (nitrous oxide/oxygen).
- ✓ Obtain pretreatment vital signs.
- ✓ Ensure profound local anesthesia.
- ✓ Provide adequate postoperative analgesia.

➢ The use vasoconstrictors
- ✓ The use of vasoconstrictors in local anesthetics poses potential problems for patients with arrhythmias because of the possibility of precipitating cardiac tachycardia or another arrhythmia. A local anesthetic without vasoconstrictor may be used as needed.
- ✓ If a vasoconstrictor is deemed necessary, patients in the low- to intermediate-risk category and those taking nonselective beta-blockers can safely be given up to 0.036 mg of epinephrine (two cartridges containing 1: 100,000 epinephrine); intravascular injections should be avoided. Greater quantities of vasoconstrictor may well be tolerated, but increasing quantities are associated with increased risk for adverse cardiovascular effects.
- ✓ Vasoconstrictors should be avoided in patients taking digoxin because of the potential for inducing arrhythmias.
- ✓ For patients at major risk for arrhythmias, the use of vasoconstrictors should be avoided, but if their use is considered essential, it should be discussed with the physician.
- ✓ Avoid the use of adrenalin in retraction cord.

➢ Patients who are taking Warfarin
- ✓ Should have current international normalized ratio (INR) (within 24 hours of surgical procedure).
- ✓ If INR is within the therapeutic range (INR, 2.0-3.5), dental treatment, including minor oral surgery, can be performed without stopping or altering the drug.
- ✓ Local measures include gelatin sponge or oxidized cellulose in sockets, suturing, gauze pressure packs, preoperative stents, and tranexamic acid or aminocaproic acid mouth rinse and/or to soak gauze.

➢ Patients with pacemakers
- ✓ Antibiotic prophylaxis to prevent bacterial endocarditis is not recommended
- ✓ *Avoid the use of* **electrosurgery and ultrasonic scalers.**

➢ Patients taking Digoxin
- ✓ Watch for signs or symptoms of toxicity (e.g., hypersalivation)
- ✓ Avoid adrenalin or levonordefrine

Oral and Maxillofacial Surgery/Fourth Year

د.ياسر رياض الخناق

Infective Endocarditis

Infective endocarditis (IE) is defined as a microbial infection of the endothelial surface of the heart or heart valves that most often occurs in proximity to congenital or acquired cardiac defects. Its intracardiac effects include severe valvular insufficiency, which may lead to intractable congestive heart failure and myocardial abscesses, therefore, emphasis has long been directed toward its prevention.

Although bacteria most often cause these diseases, fungi and other microorganisms may also cause infection; thus, the term infective endocarditis (IE) is used to reflect this multimicrobial origin.

Classification

IE is classified based on:

- The causative microorganism (e.g., streptococcal endocarditis, staphylococcal endocarditis, candidal endocarditis)
- The type of valve that is infected (e.g., native valve endocarditis [NVE], prosthetic valve endocarditis [PVE]).
- The source of infection; whether community acquired or hospital acquired, or whether the patient is an intravenous (IV) drug user or not.

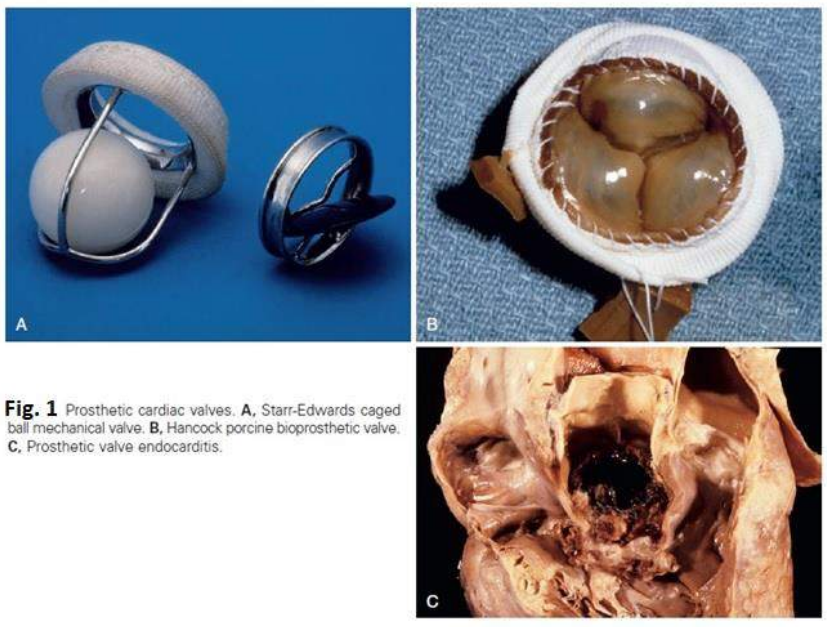


Fig. 1 Prosthetic cardiac valves. **A,** Starr-Edwards caged ball mechanical valve. **B,** Hancock porcine bioprosthetic valve. **C,** Prosthetic valve endocarditis.

Etiology

- ✓ Streptococci are the most common cause of IE 30%-65%, of which streptococci viridans (alpha-hemolytic streptococci), which are normal constituents of the oral flora and gastrointestinal tract, remain the most common cause of community acquired NVE.
- ✓ Staphylococci are the cause of at least 30%-40% of cases of IE; mostly coagulase-positive Staphylococcus aureus which is the most common pathogen in IE associated with IV drug abuse, it is also the most common pathogen in nonvalvular cardiovascular device infections.
- ✓ In some recent studies, S. aureus has emerged as the most common cause of IE and rates of viridans streptococci have decreased.
- ✓ Other microbial agents that less commonly cause IE such as the HACEK group (Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella), Pseudomonas aeruginosa, Corynebacterium, Bacteroides fragilis, and fungi.

Predisposing conditions attributed to IE include:

- ➢ Mitral valve prolapse 25%-30%.
- ➢ Aortic valve disease 12%-30%.
- ➢ Congenital heart disease 10%-20%.
- ➢ Prosthetic valve 10%-30%.
- ➢ Intravenous drug abuse 5%-20%.
- ➢ No identifiable condition 25%-47%.

Pathophysiology

IE is thought to be the result of a series of complex interactions of several factors involving endothelium, bacteria, and the host immune response. The sequences of events include:

١. Injury or damage to an endothelial surface, most often of a cardiac valve leaflet.
٢. Fibrin and platelets then adhere to the roughened endothelial surface and form small clusters or masses called nonbacterial thrombotic endocarditis (NBTE), these masses are sterile and do not contain microorganisms.
٣. With the occurrence of a transient bacteremia, bacteria can be seeded into and adhere to the mass.
٤. Additional platelets and fibrin are then deposited onto the surface of the mass, which serves to protect the bacteria that undergo rapid multiplication within the protection of the vegetative mass.
٥. The **clinical outcome results from:**
   - Local destructive effects of intracardiac (valvular) lesions.
   - Embolization of vegetative fragments to distant sites, resulting in infarction or infection.
   - Hematogenous seeding of remote sites during continuous bacteremia

- Antibody response to the infecting organism with subsequent tissue injury caused by deposition of preformed immune complexes or antibody/complement interaction with antigens deposited in tissues.

Signs and symptoms

The clinical presentation may be varied; the interval between the presumed initiating bacteremia and the onset of symptoms of IE is estimated to be less than 2 weeks in more than 80% of patients.

- ✓ Fever (most common).
- ✓ Heart murmur.
- ✓ Petechiae of the palpebral conjunctiva, the buccal and palatal mucosa, and extremities.
- ✓ Osler's nodes (small, tender, subcutaneous nodules that develop in the pulp of the digits). **Named after Sir William Osler (1849-1919).** They are caused by immune-complex deposition.
- ✓ Janeway lesions (small, erythematous or hemorrhagic, macular nontender lesions on the palms and soles). **Named after Edward Janeway (1841-1911).** They are caused by septic emboli which deposit bacteria, forming micro-abscesses of the dermis with marked necrosis and inflammatory infiltrate not involving the epidermis.
- ✓ Splinter hemorrhages in the nail beds
- ✓ Roth spots (oval retinal hemorrhages with pale centers). Caused by immune complex mediated vasculitis. **Named after Moritz Roth, a Swiss Pathologist (1839-1914).**
- ✓ Splenomegaly
- ✓ Clubbing of the digits.
- ✓ Positive blood cultures in most cases. Although up to 30% of cases of IE are initially found to be "culture negative," especially in patients who have taken antibiotics prior to the diagnosis of IE.

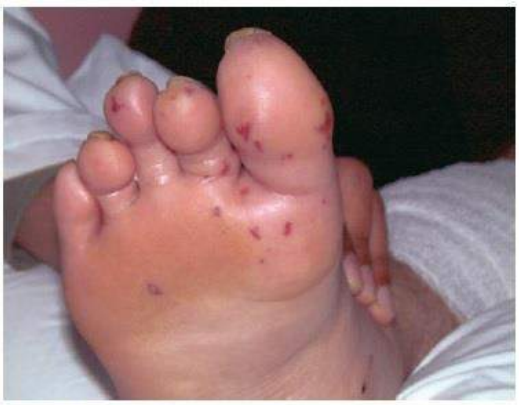


Fig. 2 Petechiae in infective endocarditis.

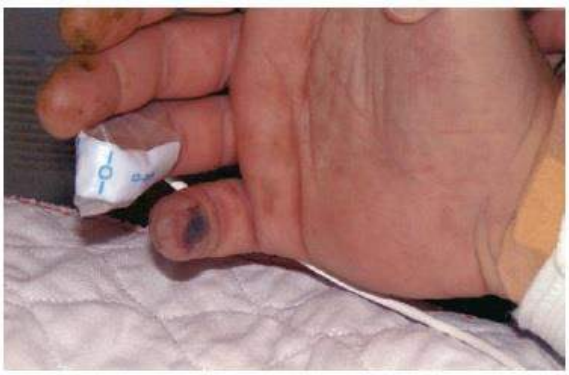


Fig. 3 Osler node in infective endocarditis. (From Fowler VG

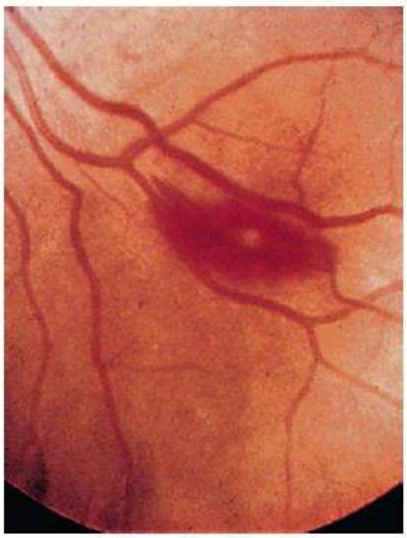


Fig. 4 A Roth spot in the retina in infective endocarditis

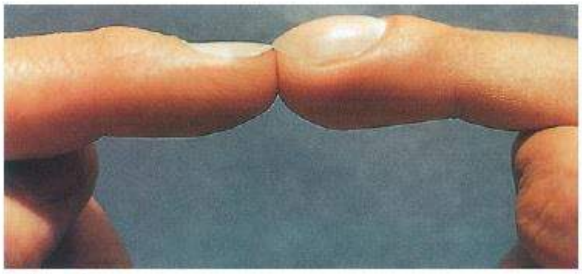


Fig. 5 Nail clubbing may appear within a few weeks of development of infective endocarditis.

Diagnosis

Duke criteria were developed to facilitate diagnosis of IE. These criteria are categorized as major and minor.

- ❖ **Major criteria:**
- ➢ Positive blood cultures.
- ➢ Evidence of endocardial involvement (e.g., positive echocardiography, presence of new valvular regurgitation.
- ❖ **Minor criteria:**
- ➢ Predisposing heart condition or IV drug use.
- ➢ Fever.
- ➢ Vascular phenomena, including embolic events.
- ➢ Immunologic phenomena.
- ➢ Microbiologic evidence other than positive blood culture.

Definitive diagnosis of IE requires the presence of two major criteria, one major and three minor criteria, or five minor criteria.

Complications

- Heart failure as a result of severe valvular dysfunction.
- Embolization of vegetation fragments leads to stroke, MI, pulmonary embolism. Emboli also may involve other systemic organs, including the liver, spleen, kidney, and abdominal mesenteric vessels.
- Renal dysfunction is also common and may be due to immune complex glomerulonephritis or infarction.

Medical Management

Generally, it consists of antibiotics and surgery. The most widely used antibiotics include penicillin, ceftriaxone, gentamicin and vancomycin while most staphylococcus aureus organisms that produce betalactamase respond to nafcillin and oxacillin and for strains resistant to oxacillin, vancomycin is combined with rifampin and gentamicin.

Surgical intervention may be necessary to facilitate a cure for IE or to repair damage caused by the infection.

Dental management

- The dentist should identify from history taking those patients with cardiac conditions that increase risk for IE and should remain alert and refer the patient with signs or symptoms of IE to physician. This would apply whether or not the patient has received prophylactic antibiotics for dental procedures.

- The basic assumption is that IE is most often due to a bacteremia that results from an invasive dental procedure, and that through the administration of antibiotics prior to those procedures, IE could be prevented. But studies have shown that bacteremia can also result from many normal daily activities such as tooth brushing, flossing, using toothpicks, using oral water irrigation devices, and chewing emphasizing the need to maintain good oral hygiene and eradicating dental/oral disease for decreasing the frequency of bacteremia produced by normal daily activities.

- Cardiac Conditions Associated with the Highest Risk of Adverse Outcome from Endocarditis for which Prophylaxis with Dental procedures is recommended:
  ١. Prosthetic cardiac valve
  ٢. Previous infective endocarditis
  ٣. Congenital heart disease (CHD)
  - ✓ Unrepaired cyanotic CHD, including those with palliative shunts and conduits.
  - ✓ Completely repaired CHD with prosthetic material or device by surgery or catheter intervention during the first 6 months after the procedure. Prophylaxis is reasonable because endothelialization of prosthetic material occurs within 6 months after the procedure.

- ✓ Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device, which inhibits endothelialization
- ٤. Cardiac transplantation recipients who develop cardiac valvulopathy.

- IE antibiotic prophylaxis is recommended only for patients listed above who undergo any dental procedure that involves the manipulation of gingival tissues or the periapical region of a tooth and for those procedures that perforate the oral mucosa.

- The following procedures and events **do not** need prophylaxis: routine anesthetic injections through noninfected tissue, restorative dentistry, taking dental radiographs, placement of removable prosthodontic or orthodontic appliances, adjustment of orthodontic appliances, placement of orthodontic brackets, shedding of deciduous teeth, suture removal, fluoride treatment and bleeding from trauma to the lips or oral mucosa.

- Antibiotic prophylaxis regimens

Situation	**Agent**	**Regimen: Single dose 30-60 Minutes before Procedure**	
		Adult	**Child**
Oral	Amoxicillin	2 g	50 mg/kg
Unable to take oral medication	Ampicillin	2g IM or IV	50 mg/kg IM or IV
	Cefazolin or Ceftriaxone	1 g IM or IV	50 mg/kg IM or IV
Allergic to Penicillins or Ampicillin Oral	Cephalexin	2 g	50 mg/kg
	Clindamycin	600 mg	20 mg/kg
	Azithromycin or Clarithromycin	500 mg	15 mg/kg
Allergic to Penicillins or Ampicillin and cannot take oral medications	Cefazolin or Ceftriaxone	1 g IM or IV	50 mg/kg IM or IV
	Clindamycin	600 mg IM or IV	20 mg/kg IM or IV

Cephalosporins should not be used in an individual with a history of anaphylaxis, angioedema, or urticaria with penicillins or ampicillin.

- Preoperative use of 0.2% Chlohexidine mouth washes is advisable.

- In patients who are already taking penicillin or amoxicillin for eradication of an infection or for long-term secondary prevention of rheumatic fever are likely to have streptococcus viridans that are relatively resistant to penicillin or amoxicillin. Therefore:
  - ✓ Clindamycin, azithromycin, or clarithromycin should be selected for prophylaxis if treatment is immediately necessary. Cephalosporins should be avoided because of cross resistance.
  - ✓ An alternative approach is to wait for at least 10 days after completion of antibiotic therapy before administering prophylactic antibiotics. In this case, the usual regimen can be used.

- In case of prolonged dental procedures (longer than 6 hours) it is advisable to administer an additional prophylactic dose (same dose).

- Prior to cardiac valve surgery or replacement or repair of congenital heart disease, it is recommended that preoperative dental evaluation be performed and necessary dental treatment provided whenever possible in an effort to decrease the incidence of late PVE caused by viridans group streptococci.

Rheumatic fever and rheumatic heart disease

Rheumatic fever is an autoimmune inflammatory process that develops after pharyngeal infection with group A beta-hemolytic streptococci (streptococcus pyogenes). It predominantly affects children between 5-15 years. Rheumatic fever may occasionally be followed by chronic rheumatic carditis with permanent cardiac valvular damage that appears to be immunologically mediated tissue damage, which may lead to fibrosis and distortion of the cardiac valves (chronic rheumatic heart disease).

Clinical manifestations

The clinical manifestations of acute rheumatic fever are so variable that the diagnosis is made only if at least two of the major criteria are fulfilled

Diagnostic criteria	
Major	**Minor**
Carditis	Pyrexia
Polyarthritis	Arthralgia
Chorea	Previous rheumatic fever
Erythema marginatum	Raised ESR and C-reactive protein
Subcutaneous nodules	Characteristic ECG changes

Commented [WU1]: **Chorea** is a movement disorder that causes involuntary, irregular, unpredictable muscle movements. The disorder can make you look like you're dancing (the word **chorea** comes from the Greek word for “dance”) or look restless or fidgety. **Chorea** is a movement problem that occurs in many different diseases and conditions.

- A sore throat may be followed after about 3 weeks by an acute febrile illness with multiple joints pain (migratory arthralgia) which heals without permanent damage in about 3 weeks.
- Cerebral involvement causing spasmodic involuntary movements (Sydenham chorea, St. Vitus dance).
- A characteristic rash (erythema marginatum).
- Lung involvement
- Subcutaneous nodules (usually around the elbows).
- The most serious cardiac complication is subendocardial inflammation, particularly along the lines of closure of the mitral and aortic valve cusps, resulting in the formation of fibrinous vegetations and later scarring, fibrotic stiffening and distortion of the heart valves, often causing mitral valve and/or aortic valve stenosis. This is essentially a mechanical, hemodynamic disorder, in which the defective valves may become infected at any time, leading to infective endocarditis. Cardiac failure can develop, often after many years.

Medical management

- ✓ Prompt antimicrobial treatment of streptococcal sore throat (within 24 hours of onset) prevents the development of rheumatic fever in most cases.
- ✓ After an attack of rheumatic carditis, there is a risk of recurrence and continuous antibiotic prophylaxis becomes necessary to lessen the risk of permanent cardiac damage. The drug of choice is usually oral phenoxymethyl penicillin until the age of 20. For those allergic to penicillin, sulfadimidine should be given.

Dental management

- Acute rheumatic fever patients are exceedingly unlikely to be seen during an attack but emergency dental treatment may be necessary.
- Patients with history of rheumatic fever but without cardiac involvement are treated as a normal person.
- Most patients with chronic rheumatic heart diseases are anticoagulated and they should be managed after determining their prothrombin time and INR and the treatment can be done under local anesthesia with vasoconstrictor in consultation with the physician. Conscious sedation with nitrous oxide may be given if cardiac function is good and with the approval of the physician.
- Indications for prophylactic antibiotics are only for the high risk patients mentioned in the dental management of IE.

Congenital heart diseases

Congenital heart diseases (CHD) are the most common type of cardiac diseases present in children. They can broadly be classified as Cyanotic and Acyanotic (non-cyanotic).

❖ Cyanotic CHDs

The cyanosis results from shunting of deoxygenated blood from the right ventricle into the left side of the heart and the systemic circulation (right to left shunt) leading to chronic hypoxemia, they include:

- ✓ Eisenmenger syndrome **(Named after Victor Eisenmenger who described this condition in 1897)**.
- ✓ Fallot's tetralogy **(Named after the French physician Etienne-Louis-Arthur Fallot 1850-1911)**.
- ✓ Pulmonary atresia.
- ✓ Pulmonary valve stenosis.
- ✓ Total anomalous venous drainage.
- ✓ Transposition of great vessels.
- ✓ Tricuspid atrasia.

Patients may crouch to improve venous return, but eventually polycythemia with hemorrhagic and thrombotic tendencies develop, finger and toe clubbing develops but after 3 months of age. If untreated, 40% of patients with cyanotic CHD die within 5 years.

❖ Acyanotic CHDs

They are further divided into those with no shunt like; Aortic stenosis, bicuspid aortic valve, coarctation of the aorta, dextrocardia and mitral valve prolapse. The other division of the Acyanotic CHD is those diseases with left to right shunt and these include; Atrial septal defects (ASD), Ventricular septal defects (VSD) and patent ductus arteriosus (PDA).

Some CHD start as Acyanotic diseases and become cyanotic with time. Most of these cardiac defects are well tolerated in utero, and it is only after birth that their anatomic and hemodynamic abnormalities become evident.

CHD is most commonly diagnosed through echocardiography, and confirmed by cardiac magnetic resonance imaging (MRI). Early correction of the congenital defect, often by transvenous catheter techniques, is the treatment of choice. More complex defects may require an operation. Medical treatment may be needed for the management of pulmonary edema, heart failure, polycythemia, infection or emotional disturbances.

Modern surgical and medical care helps children survive into adult life and patients are then often called adult or 'grown-up' CHD. Nevertheless, complications observed in adults who were previously thought to have had successful repair of CHD include arrhythmias, valve disorders and cardiac failure, and residual defects can still predispose to complications such as infective endocarditis.

Dental management

- The most important aspect for dentists to consider is how well the patient's heart condition is compensated. Consultation with the physician is recommended.
- Patients with heart disease should take their medications as usual on the day of the dental procedure, and should bring all their medications to the dental office for review at the time of the first appointment.
- Patients with stable heart disease receiving atraumatic treatment under local anesthesia can receive treatment.
- Late morning or early afternoon appointments are advisable.
- Stress-reduction and good analgesia should be provided.
- Limited use of vasoconstrictor with aspirating syringes.
- Retraction cords containing adrenalin should be avoided.
- Conscious sedation preferably with nitrous oxide can be given with the approval of the physician. General anesthesia should only be provided by expert anesthetists in hospital.
- Bleeding tendencies due to platelet dysfunction or coagulation defects should be evaluated and managed accordingly.
- There may be susceptibility to infective endocarditis, so prophylactic antibiotics should be used in the following cases:

✓ Unrepaired cyanotic CHD, including those with palliative shunts and conduits

✓ Completely repaired CHD with prosthetic material or device by surgery or catheter intervention during the first 6 months after the procedure. Prophylaxis is reasonable because endothelialization of prosthetic material occurs within 6 months after the procedure.

✓ Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device, which inhibits endothelialization.

- Prior to cardiac valve surgery or replacement or repair of congenital heart disease, it is recommended that preoperative dental evaluation be performed and necessary dental treatment provided whenever possible in an effort to decrease the incidence of late PVE caused by viridans group streptococci.

Oral manifestations

- Delayed eruption of both dentitions
- Enamel hypoplasia; the teeth often have a bluish-white 'skimmed milk' appearance and there is gross vasodilatation in the pulps
- Greater caries and periodontal disease activity, probably because of poor oral hygiene and lack of dental attention
- After cardiac surgery, transient small white non-ulcerated mucosal lesions of unknown etiology may appear.

ORAL SURGERY

LECTURE 4 **ا.م.د.احمد فاضل ابراهيم القيسي**

Pregnancy

A pregnant patient poses a unique set of management considerations for the dentist. Dental care must be rendered to the mother without adversely affecting the developing fetus, and although routine dental care of pregnant patients is generally safe, the delivery of dental care involves some potentially harmful elements, including the use of ionizing radiation and drug administration. Thus, prudent practitioners must balance the beneficial aspects of dentistry while minimizing or avoiding exposure of the patient (and the developing fetus) to potentially harmful procedures.

Medical Consideration:

1- **physiologic finding;**
   a- during first trimester
      i- Fatigue that may have a psychological impact.
      ii- syncope and postural hypotension
   b- During the second trimester; relatively few symptoms.
   c- During the third trimester,
   - increasing fatigue and discomfort and mild depression
   - cardiovascular changes
      i- Blood volume increases by 40% to 50%
      ii- High-flow, low-resistance circulation; tachycardia; and heart murmurs, and it may unmask glomerulopathies, peripartum cardiomyopathy, arterial aneurysms, or arteriovenous malformations.
      iii- cardiac output increases by 30% to 50%
      iv- blood pressure falls (usually to ≤100/70 mm Hg) during the second trimester, and a modest increase is noted in the last month of pregnancy.
      v- A benign systolic ejection murmur is common, disappears after delivery.
   d- late pregnancy; supine hypotensive syndrome, an abrupt fall in blood pressure, bradycardia, sweating, nausea, weakness, and air hunger when the patient is in a supine position caused by impaired venous return to the heart that results from compression of the inferior vena cava by the uterus. This leads to decreased blood pressure, reduced cardiac output, and impairment or loss of consciousness. The remedy for the problem is to roll the patient over onto her left side, which lifts the uterus off the vena cava.

2- **Blood changes**
   a- Anemia, blood volume increases more rapidly than RBC mass (insufficient iron)
   b- decreased hematocrit value.

c- hypercoagulable state; increase in many of the coagulation factors, combined with venous stasis; risk of thromboembolism
d- WBC and immunologic changes occur
i- WBC count increases progressively
ii- the immune system shifts from helper T-cell 1 (TH1) dominance to TH2 dominance. This leads to immune suppression. Clinically, the decrease in cellular immunity leads to increased susceptibility to intracellular pathogens such as cytomegalovirus virus, herpes simplex virus, varicella, and malaria.

3- Changes in respiratory function
i- elevation of the diaphragm, which decreases the volume of the lungs in the resting state
ii- ventilatory changes; increased rate of respiration (tachypnea) and dyspnea that is worsened by the supine position (sleep is impaired, especially in 3rd trimester)

4- increased appetite and often a craving for unusual foods (unbalanced diet)
a- affect the mother's dentition
b- weight gain.

5- Taste alterations and an increased gag response

6- Nausea and vomiting, or "morning sickness (between 4 and 8 weeks)

Pattern of fetal development

- Normal pregnancy lasts 40 weeks.
- first trimester, organs and systems are formed (organogenesis); susceptible to malformation
- After the first trimester, growth and maturation; malformation diminished, with the exception of the fetal dentition; susceptible to malformation from toxins or radiation and to tooth discoloration caused by administration of tetracycline.

Common complications

1- infection,
2- inflammatory response,
3- glucose abnormalities
4- hypertension; endorgan damage or preeclampsia, a clinical condition of pregnancy that manifests as hypertension, proteinuria, edema, and blurred vision.
Preeclampsia (hypertension with proteinuria) progresses to eclampsia, a life-threatening condition, if seizures or coma develop.

5- Insulin resistance is a contributing factor to the development of gestational diabetes mellitus (GDM)
a- increases the risks for infection
b- large birth weight babies.

6- spontaneous abortion (miscarriage); natural termination of pregnancy before the 20th week of gestation; causes

i- morphologic or chromosomal abnormalities that prevent successful implantation. It is most unlikely that any dental procedure would be implicated in spontaneous abortion, provided fetal hypoxia and exposure of the fetus to teratogens are avoided.
ii- Febrile illness and sepsis; prompt treatment of odontogenic infection and periodontitis is advised

*Pharmacologic challenge of fetus to be avoided; immature liver and enzyme systems

7- postpartum period
   a- lack of sleep
   b- postpartum depression
   c- risks for autoimmune disease; rheumatoid arthritis, multiple sclerosis, and thyroiditis

DENTAL MANAGEMENT

1- assess the general health of the patient through a thorough medical history.
   a- current physician
   b- medications (obstetrician consulted)
   c- use of tobacco, alcohol, or illicit drugs;
   d- GDM;
   e- miscarriage;
   f- hypertension
   g- morning sickness
2- Establishing a good patient–dentist relationship
3- measuring vital signs for identifying undiagnosed abnormalities (Systolic pressure at or above 140 mm Hg and diastolic pressure at or above 90 mm Hg are signs of hypertension- referred)
4- if blood pressure increases 30 mm or more systolic or an increase of 15 mm Hg in diastolic blood pressure compared with prepregnancy values; can be a sign of preeclampsia

5- Preventive Program; establish optimum level of oral hygiene, throughput pregnancy, plaque control program helps to
- reduces exaggerated inflammatory response of gingival tissues to local irritants that commonly accompany the hormonal changes of pregnancy.
- reduce the risk that infant will develop caries
   A- Coronal scaling and polishing or root curettage may be performed whenever necessary
   B- Acceptable oral hygiene techniques should be taught
   C- Diet counseling, with emphasis on limiting the intake of refined carbohydrates
   D- Chlorhexidine 0.12% mouth rinse may be used safely during pregnancy, if needed

*there was a lack of evidence to support a recommendation for the use of prenatal fluoride.

Dental Treatment Timing

First trimester; Other than as part of a good plaque control program, elective dental care is best avoided (vulnerability of fetuses).

Second trimester is the safest period

- provide routine dental care
- controlling active disease
- eliminating potential problems that could occur later
- Extensive reconstruction or significant surgical procedures are best postponed until after delivery.

Early part of the third trimester;

- provide routine dental care.

After the middle of the third trimester;

- elective dental care is best postponed (increasing feeling of discomfort).
- Prolonged time in the dental chair avoided to prevent supine hypotension.
- If supine hypotension develops, rolling the patient onto her left side
- short appointments
- semireclined position
- frequent changes of position

Dental Radiographs

1- The safety of dental radiography has been well established, provided these features are used
   a- fast exposure techniques (e.g., high-speed film or digital imaging),
   b- filtration
   c- collimation
   d- lead aprons
   e- thyroid collars
2- Ionizing radiation should be avoided, if possible, during pregnancy, especially during the first trimester because developing fetuses are particularly susceptible to radiation damage.
3- The teratogenicity of ionizing radiation is dose dependent.
4- maternal thyroid exposure to diagnostic radiation in excess of 0.4 mGy has been associated with a slight decrease in birth weight
5- Teratogenicity is also dependent on the gestational age of the fetus at the time of exposure.

- During the organogenesis fetuses are extremely sensitive to ionizing radiation, particularly CNS between the 8th and 15th weeks of pregnancy.
- From the 16th to the 25th week, there is a reduction in the radiosensitivity of the CNS
- After the 25th week, the CNS becomes relatively radioresistant, and major fetal malformations and functional anomalies are highly improbable.

6- with use of a lead apron, rectangular collimation, and E-speed film or faster techniques, one or two intraoral films of minute significance in terms of radiation effects on a developing fetus (fetal dose of two periapical dental films (when a lead apron is used) is 700 times less than 1 day of average exposure to natural background radiation in the United States.30,31

7- Radiographs should be used only when necessary
8- Bitewing, panoramic, periapical film recommended for minimizing dose.
9- measures to further reduce the radiation dose
   a- rectangular collimation
   b- E-speed or F-speed film or faster techniques (digital imaging reduces radiographic exposure by at least 50% compared with E-speed exposures)
   c- lead shielding (abdominal and thyroid collar)
   d- high kilovoltage (kV) or constant beams
10- Production of congenital defects is negligible from fetal exposures of 50 mSv.

Oral Complications and Manifestations

1) pregnancy gingivitis;
   a- results from an exaggerated inflammatory response to local irritants and less than meticulous oral hygiene during periods of increased secretion of estrogen and progesterone and altered fibrinolysis.
   b- begins at the marginal and interdental gingiva,
   c- usually in the second month of pregnancy.
   d- Progression leads to fiery red and edematous interproximal papillae that are tender to palpation.
2) In 1% of these ladies; hyperplastic response (seen in pregnancy gingivitis) may exacerbate in a localized area, resulting in a pyogenic granuloma or "pregnancy tumor"
   A- common location is the labial aspect of the interdental papilla.
   B- generally asymptomatic
   C- tooth brushing may traumatize the lesion and cause bleeding.
- Gingival changes become apparent around the second month and continue until after parturition, then these changes regress to normal, provided if good oral hygiene is maintained
- Surgical or laser excision is occasionally required if symptoms, bleeding, or interference with mastication dictates.

3) Pregnancy does not cause periodontal disease but may modify and worsen what is already present.
4) Caries incidence increases; attributed to

- the presence of cariogenic bacteria
- diet containing fermentable carbohydrates
- poor oral hygiene.

Control of the carious process through fluoride and chlorhexidine is important because maternal saliva is the primary vehicle for transfer of cariogenic streptococci to the infant.

5) Many women are convinced that pregnancy causes tooth loss (i.e., "a tooth for every pregnancy") or that calcium is withdrawn from the maternal dentition to supply fetal requirements (i.e., "soft teeth").

- Calcium is present in the teeth in a stable crystalline form and hence is not available to the systemic circulation to supply a calcium demand.
- Calcium is readily mobilized from bone to supply these demands.
- Calcium supplementation needed for general nutritional requirements of the mother and infant.

6) Tooth mobility, localized or generalized,
   - Uncommon
   - sign of gingival disease, disturbance of the attachment apparatus, and mineral changes in the lamina dura.
   - vitamin deficiencies may contribute to it and other congenital problems (e.g., folate deficiency: spina bifida),
   - the dentist should educate the patient about the benefits of the use of multivitamins.
   - Reversal of tooth mobility may result from
     1- . Daily removal of local irritants
     2- adequate levels of vitamin C
     3- delivery of the new born should
7) Hypersensitive gag reflex and/or morning sickness, may contribute to regurgitation and lead to halitosis and enamel erosion. Advise the patient to rinse after regurgitation with a solution that neutralizes the acid (e.g., baking soda, water).

Drug Administration

Drugs prescribed during pregnancy should not be

1) Toxic
2) Teratogenic
3) respiratory depressant, causing maternal hypoxia, resulting in fetal hypoxia, injury, or death.

General rules

I- drug administration should be avoided during pregnancy, especially during the first trimester.

II- most of the commonly used drugs in dental practice can be given during pregnancy with relative safety

PREGNANCY LABELING CATEGORIES FOR DRUGS POTENTIAL RISK OF FETAL INJURY

A drug shown by controlled studies in humans have no risk to the fetus, and the possibility of fetal harm appears remote.

B drugs shown by animal studies have not indicated fetal risk, and human studies have not been conducted

C drugs shown by animal studies have a risk, but controlled human studies have not been conducted, or studies are not available in humans or animals.

D drugs shown to have human fetal risk, but in certain situations, the drug may be used despite its risk.

X drugs shown to inflict fetal abnormalities and fetal risk exists based on human experience

- Drugs in categories A or B are preferable for prescribing during pregnancy.
- Drugs used in dentistry fall into category C (safety uncertain, e.g., narcotic analgesic for a pregnant patient who is in severe pain.)
- Local Anesthetics (lidocaine, prilocaine) with epinephrine generally safe.
- Articaine, bupivacaine, and mepivacaine used with caution.
- local anesthetic and the vasoconstrictor cross the placenta, subtoxic threshold doses have not been shown to cause fetal abnormalities.
- topical anesthetics, (e.g., benzocaine, dyclonine, and tetracaine) used with caution.
- topical lidocaine is safe

Analgesics

- The analgesic of choice is acetaminophen.
- Aspirin and nonsteroidal antiinflammatory drugs convey risks for constriction of the ductus arteriosus, as well as for postpartum hemorrhage and delayed labor (risk increases during the third trimester). Therefore, it is best to avoid these analgesics (especially in the third trimester)
- Same risk is shown with potent antiinflammatory drugs (e.g., glucocorticoids and indomethacin).
- opioids, including codeine, Demerol, and propoxyphene, are associated with multiple congenital defects
- hydrocodone and oxycodone safety are unclear (best avoided)

Antibiotics

- Penicillins (including amoxicillin), erythromycin (except in estolate form), cephalosporins, metronidazole, and clindamycin, are safe
- tetracycline, including doxycycline, is contraindicated during pregnancy. Tetracyclines bind to hydroxyapatite, causing
  a- brown discoloration of teeth

b- hypoplastic enamel
c- inhibition of bone growth, and other skeletal abnormalities

- Clarithromycin should be avoided

Antibiotics and Oral Contraceptives

- Select antibiotics (e.g., rifampin, an antituberculosis drug) reduces plasma levels of circulating oral contraceptives.
- American Dental Association Council on Scientific Affairs issued the following recommendations when prescribing antibiotics to a female patient who takes oral contraceptives:
  "The dentist should
  1) advise the patient of the potential risk of the antibiotic's reducing the effectiveness of the oral contraceptive
  2) recommend that the patient discuss with her physician the use of an additional nonhormonal means of contraception
  3) advise the patient to maintain compliance with oral contraceptives when concurrently using antibiotics."

*In general, dentists should provide treatment for acute infection irrespective of the stage of pregnancy.

Anxiolytics:

- Few anxiolytics are considered safe to use during pregnancy.
- Benzodiazepines, zaleplon, and zolpidem should be avoided.
- single, short-term exposure to nitrous oxide–oxygen (N2 O–O2) for less than 35 minutes is not thought to be associated with any human fetal anomalies
- chronic occupational exposure to N2 O–O 2 has been associated with spontaneous abortion and reduced fertility in humans.
- following guidelines are recommended if N2 O–O 2 is used during pregnancy
  1) Use of N2 O–O 2 inhalation should be minimized to 30 minutes.
  2) At least 50% oxygen should be delivered to ensure adequate oxygenation at all times.
  3) Appropriate oxygenation should be provided to avoid diffusion hypoxia at the termination of administration.
  4) Repeated and prolonged exposures to nitrous oxide are to be prevented.
  5) The second and third trimester are safer periods for treatment because organogenesis occurs during the first trimester.
- Female dental health care workers who are chronically exposed to nitrous oxide for more than 3 hours per week, when scavenging equipment is not used, have decreased fertility and increased rates of spontaneous abortion.

Nursing:

- The concern is that the administered drug may enter the breast milk and be transferred to the nursing infant
- The AAP concludes that "most drugs likely to be prescribed to the nursing mother should have no effect on milk supply or on infant wellbeing." (amount of drug excreted in the breast milk usually is not more than about 1% to 2% of the maternal dose)
- Drugs, or categories of drugs, are definitely contraindicated for nursing mothers.
  a- lithium, anticancer drugs
  b- radioactive pharmaceuticals
  c- phenindione.
- nursing mothers may take the drug just after breastfeeding and avoid nursing for 4 hours or longer (reduce drug concentration in the breast milk)

Treatment Planning Modifications

1) No technical modifications required except the following best delayed until after pregnancy
- full-mouth radiographs.
- Reconstruction.
- Crown and bridge procedures.
- aggressive surgery.

2) A prominent gag reflex may dictate a delay in certain dental procedures.

ORAL SURGERY

LECTURE 3 **ا.م.د.احمد فاضل ابراهيم القيسي**

Bleeding disorder

A number of procedures that are performed in dentistry may cause bleeding. Under normal circumstances, these procedures can be performed with little risk; however, patients whose ability to control bleeding is altered by congenital defects in coagulation factors, platelets, or blood vessels may be in grave danger unless the dentist identifies the problem before performing any dental procedure. In most cases, after a patient with a congenital bleeding problem has been identified, steps can be taken to greatly reduce the risks associated with dental procedures.

Hemophilia A:

The hemostatic abnormality in hemophilia A is caused by a deficiency or a defect of factor VIII. Factor VIII circulates in plasma bound to Von Willebrand factor (vWF). Unbound factor VIII is destroyed. Factor VIII was thought to be produced by endothelial cells and not by the liver, as most coagulation factors are. However, when disease was corrected by transplantation in several liver transplant recipients with hemophilia, it became clear that liver parenchymal cells also produce factor VIII.

Hemophilia A is inherited as an X-linked recessive trait. The defective gene is located on the X chromosome. An affected man will not transmit the disease to his sons; however, all of his daughters will be carriers of the trait because they inherit his X chromosome. A female carrier will transmit the disorder to half of her sons and the carrier state to half of her daughters. Severity of bleeding varies from kindred to kindred. Within a given kindred, the clinical severity of the disorder is constant; for example, relatives of people with severe hemophilia are likely to be affected severely. The mutation rate for the responsible gene is unusually high (up to 30%), which explains why a rare condition such as hemophilia A does not die out after several generations. Because of the high mutation rate of the responsible gene, a negative family history is of limited value in excluding the possibility of hemophilia A. The assay of factor VIII activity can be used to identify female carriers of the trait. About 35% of carriers will show a decrease in factor VIII (≈50% of normal factor VIII levels). Other carriers may have normal levels of factor VIII. Immunoassays for vWF can greatly improve the detection rate among carriers of hemophilia A. Polymorphic DNA probes are now available that are capable of detecting 90% of affected families and 96% or more of carriers. Hemophilia A can

manifest in women. This occurs in a mating between an affected male and a female carrier. Half of the daughters of such a mating would inherit two abnormal X chromosomes—one from the affected father and one from the carrier mother. These daughters would have homozygous hemophilia. In addition, hemophilia may occur in a minority of heterozygous carriers. Normal homeostasis requires at least 30% factor VIII activity. Symptomatic patients usually have factor VIII levels below 5%. Those with factor VIII levels between 5% and 30% have a mild form of the disease. Patients with levels between 1% and 5% have moderate disease, and severe forms of the disease occur when the level is less than 1% of normal. About 60% of cases of hemophilia are severe.

HEMOPHILIA B

In hemophilia B (Christmas disease), factor IX is deficient or defective. Hemophilia B is inherited as an X-linked recessive trait (F9 gene). Factor IX levels below 10% have been reported in a few women. Similar to hemophilia A, the disorder manifests primarily in males. Severe disease, in which affected patients have less than 1% of normal amounts of factor IX, is less common than in hemophilia A. Clinical manifestations of the two disorders are identical. Screening laboratory test results are similar for both diseases. Specific factor assays for factor IX establishes the diagnosis. Purified factor IX products are recommended for the treatment of minor and major bleeding. Recombinant factor IX is now available for clinical use.

Von Willebrand's disease

The most common inherited bleeding disorder is von Willebrand disease, which is caused by an inherited defect involving platelet adhesion. The cause of platelet dysfunction in von Willebrand disease is a deficiency or a qualitative defect in vWF.

The disease has several variants, depending on the severity of genetic expression. Most of the variants are transmitted as autosomal dominant traits (types 1 and 2). These variants of the disease tend to result in mild to moderate clinical bleeding problems. Type 1 is the most common form of von Willebrand disease. It accounts for about 70% to 80% of the cases. The greater the deficiency of vWF in type 1 disease, the more likely it is that signs and symptoms of hemophilia A will be found. Type 2 A is responsible for 15% to 20% of cases. The other variants of the disease are uncommon. Type 3, which is rare, is transmitted as an autosomal recessive trait that leads to severe deficiency of vWF and FVIII.

Variants of von Willebrand disease with a significant reduction in vWF or with a vWF that is unable to bind factor VIII may show signs and symptoms of hemophilia A, in addition to those associated with defective platelet adhesion.

in mild cases, bleeding occurs only after surgery or trauma. In the more severe cases—type 2N and type 3—spontaneous epistaxis or oral mucosal bleeding may be noted.

Thrombocytopenia:

It occurs when the platelet count that falls below the lower limit of normal, i.e., 150000/microliter (for adults). risks associated with thrombocytopenia range from no risk at all to bleeding risks and thrombosis. The correlation of severity of thrombocytopenia and bleeding risk is uncertain. Spontaneous bleeding can occur with a platelet count under 10000/microliter and surgical bleeding with counts below 50000/microL. Thrombocytopenia is associated with risk of thrombosis in conditions like heparin-induced thrombocytopenia (HIT), antiphospholipid antibody syndrome (APS), disseminated intravascular coagulation (DIC), thrombotic microangiopathy (TMA), paroxysmal nocturnal hemoglobinuria (PNH).

Infiltration and block injections of local anesthesia can be provided in patients with platelet counts above 30,000/µL. Also, most routine dental procedures can be performed. If the platelet count is below this level, routine dental treatment involving minor tissue injury should be delayed. For urgent or emergency dental needs, platelet replacement is indicated. If the platelet count is above 50,000/µL, extractions and dentoalveolar surgery can be performed. For more advanced surgery, the platelet count should be 80,000/µL and 100,000/µL or higher. Patients with platelet counts below these levels will need platelet replacement before undergoing the planned procedures.

Common causes of thrombocytopenia:

1- Primary immune thrombocytopenia (primary ITP). An autoimmune condition where antibodies are produced against platelets resulting in platelet destruction.

2- Drug-induced immune thrombocytopenia:

- Heparin-induced thrombocytopenia (HIT) - in this condition, anti-platelet antibodies activate platelets resulting in thrombosis (both arterial and venous)
- Quinine
- Sulfonamides, ampicillin, vancomycin, piperacillin
- Acetaminophen, ibuprofen, naproxen
- Cimetidine

3- Drug-induced non-immune thrombocytopenia. Drugs like valproic acid, daptomycin, linezolid cause thrombocytopenia by dose-dependent suppression of platelet production.

4- Infections:

    - Viral: HIV, hepatitis C, Ebstein-Barr virus, parvovirus, mumps, varicella, rubella, Zika viral infections can cause thrombocytopenia.
    - Sepsis causes bone marrow suppression.
    - Helicobacter pylori
    - Malaria

5- Hypersplenism due to chronic liver disease

6- Chronic alcohol abuse

7- Nutrient deficiencies (folate, vitamin B12, copper)

8- Autoimmune disorders like systemic lupus erythematosus, rheumatoid arthritis associated with secondary ITP

9- Pregnancy. Mild thrombocytopenia presents in gestational thrombocytopenia; moderate-severe thrombocytopenia can occur in preeclampsia and HELLP (hemolysis, elevated liver enzymes, low platelet count) syndrome

10- Aplastic anemia

11- Inherited thrombocytopenia. Often seen in children, rare in adults

    - Von Willebrand disease type 2
    - Alport syndrome
    - Fanconi syndrome.
    - Bernard–Soulier syndrome

Dental management of the patient with bleeding disorders

1- Patient Evaluation and Risk Assessment

• Evaluate and determine whether a bleeding disorder (e.g., hemophilia) exists.

• Obtain medical consultation if undiagnosed, poorly controlled, or if uncertain. Screen patients with bleeding history or clinical signs of a bleeding disorder with PT, PTT, TT, and platelet count.

2- Drugs

Analgesics Avoid aspirin, aspirin-containing compounds, and other NSAIDs; acetaminophen with or without codeine is suggested for most patients.
Antibiotics Not indicated unless acute infection is present.
Anesthesia Avoid block anesthetic injections in patients not on desmopressin, aminocaproic acid, or factor concentrates.
Anxiety No issues
Allergy Patients placed on factor VIII replacement need to be observed for signs and symptoms of allergy.

3- Bleeding

These patients are at great risk of bleeding from invasive dental procedures. Special precautions must be taken before invasive procedures. Patients with mild to moderate hemophilia can be managed using desmopressin and aminocaproic acid for many dental procedures. Factor VIII replacement is needed for patients with more severe hemophilia. Patients who are low responders for inhibitors (antibody response to factor VIII) require higher doses of factor VIII. Patients who are high responders are most difficult to manage and require activated factor VII, porcine factor VIII, steroids, or other special preparations such as prothrombin complex concentrates or activated prothrombin complex concentrations.

4- Consultation

The patient's hematologist must be consulted before any invasive dental procedures are performed. The severity of disease must be established. The presence of inhibitors and level of response to factor VIII need to be determined. Determine if the patient can be managed with desmopressin and aminocaproic acid. Establish the type and dosage of factor replacement needed for invasive dental procedures or surgery. Determine if the patient can be managed in the dental office or will require hospitalization.

5- Devices

Splints: may be constructed before multiple extractions or surgical procedures in patients with severe hemophilia.

Drugs: Avoid all drugs that may cause bleeding, such as aspirin and other NSAIDs, certain herbal medications, and over-the-counter drugs containing aspirin.

6- Emergencies

Excessive bleeding may occur after invasive dental procedures or surgery. Systemic and local means may be required to control the bleeding. Allergic reactions may occur in patients receiving factor replacement.

7- Follow-up

Patients should be seen and examined for signs of allergy or bleeding within 24 to 48 hours after surgical procedures

Blood dyscrasias

Disorders of the RBCs

1- Anemia

Anemia is the reduction in the oxygen carrying capacity of the blood, it is associated with the decreased number of circulating RBCs or abnormalities in the Hemoglobin (Hb) contained in the RBCs, which is the oxygen carrying molecule of the erythrocytes, it is also responsible for the transport of CO2. Hb is a heterogeneous group of proteins consisting of 4 globin chains and 4 haem (heme) groups. In anemia Hb level is below 12 g/dl in adult female and below 13 g/dl in adult male. Anemia is not a disease but rather a feature or symptom that results from many underlying causes.

Types of anemia:

• Deficiency anemias: Iron deficiency anemia; it is caused by blood loss, poor iron intake, poor iron absorption or increased demands for iron. It is more common in women than in men due to blood loss during menstruation and pregnancy. Vitamin B12 (cobalamin) deficiency, Folate (Folic acid) deficiency and *Pernicious anemia*; (*Vit. B12 and folic acid are needed for RBCs formation and growth within the bone marrow. Vit B12 is bound to gastric intrinsic factor secreted by the parietal cells and absorbed in the terminal ileum, deficiency of the intrinsic factor causes Pernicious anemia*).

• Hemolytic anemias: Hemoglobinopathies; these are inherited abnormalities of the Hb formation like Sickle cell anemia and Thalassemia. Inherited abnormal function or structure of erythrocytes; Erythrocyte metabolic defects as in Glucose-6-Phosphate Dehydrogenase deficiency (G6PD). And Erythrocyte membrane defects as in Spherocytosis, Ovalocytosis and Stomatocytosis. Damage to erythrocytes; which could be autoimmune, drug induced or infective. Worldwide Malaria is the most common cause of hemolytic anemia.

• Other anemias: Aplastic anemia; it is a pancytopenia with a non-functioning bone marrow, many cases are idiopathic but possible causes include: Chemical like Benzene, drugs, hepatitis virus, irradiation and graft versus host disease.

Anemia caused by bone marrow infiltration by abnormal cells; like in Leukemia and Multiple Myloma.

Anemia associated with systemic diseases; like in chronic inflammation and connective tissue diseases such as Rheumatoid Arthritis, Liver disease, Hypothyroidism, Hypopituitarism, Hypoadrenocorticism, Uremia and HIV infections.

Oral manifestations

Pale mucosa, oral ulcerations, angular cheilitis, glossitis and loss of papillae with atrophic changes in the oral mucosa.

patients with iron deficiency anemia may develop Plummer-Vinson syndrome and burning mouth symptoms.

patients with hemolytic anemia, there may be oral evidence of jaundice due to excessive red cells destruction, the trabecular pattern of bone may be affected due to hyperplasia of marrow elements so radiographs show enlarged marrow spaces and osteoporosis, the trabeculae between the teeth appear horizontal (stepladder).

Skull radiographs show hair on end appearance due to the new bone formation on the outer table of the skull. Vaso-occlusive events can lead to osteomyelitis, necrosis and peripheral neuropathy. Dental hypoplasia and delayed eruption of teeth often occur.

Dental management

1- Identification of the conditions associated with anemia through obtaining careful history, the questions should include history of dietary intake, malnutrition, alcohol or drug use, history of blood loss especially for women during menstruation and pregnancy. The clinician should also identify signs and symptoms of anemia and can also order some screening tests, if the results of one of the tests or more are abnormal, the patient should be referred for medical evaluation and treatment.

2- the clinician should ensure that the patient's underlying condition is under therapeutic control before proceeding with routine dental care. Patients with signs and symptoms of anemia and Hb level below 11 g/dl with abnormal heart rate or reduced oxygen saturation (below 91% in oximetry) are considered unstable and routine dental treatment should be deferred.

3- Local anesthesia (LA) is satisfactory for pain control, conscious sedation can be given only if there is supplemental oxygen, elective operations under general anesthesia (GA) are not carried out when Hb level is below 10 g/dl.
4- In patients with G6PD deficiency, certain drugs should be avoided since they can cause hemolysis, such as Sulfonamides (Sulfamethoxazole), Aspirin, Chloramphenicol and to a lesser extent Penicillin, Strepromycin and Isoniazide. Also, dental infections should be avoided and if they occur, they should be treated effectively.
5- In patients with Sickle cell anemia, routine dental care can be provided for stable patients during non-crisis period, appointments should be short and the procedures should be not complicated, oral infections should be avoided, LA without vasoconstrictor for routine dental care is used while for surgical procedures LA with vasoconstrictor 1:100000 can be used. Barbiturates and strong narcotics should be avoided and Diazepam used when sedation is needed, prophylactic Antibiotics for surgical procedures are used, liberal use of Salicylates should be avoided and pain control can be achieved with acetaminophen (Paracetamol) and Codeine. In general infection, dehydration, hypoxia, acidosis and cold should be avoided in patients with Sickle cell anemia because the can precipitate acute crisis.

2- Polycythemia

It is an expansion mainly in the red cell population, it may be primary and idiopathic associated with normal erythropoietin level (Polycythemia Rubra Vera) PRV. It can also be secondary to tumors that release erythropoietin hormone. PRV is a disease of elderly and of smokers, it has a slight male predilection. Diagnosis of Polycythemia is made when Hb level is above 16.5 g/dl and hematocrit 48% in women and when Hb is above 18.5 g/dl and hematocrit 52% in men.

Dental management

• LA regional blocks should be avoided if possible.
• Conscious sedation can be given.
• GA is allowed.
• Susceptibility to thrombosis and hemorrhage should be considered.
• Cytotoxic chemotherapy may cause oral complications that require management.

WBCs Disorders

1- Leukemia

Is cancer of the WBCs that affects the bone marrow and circulating blood. It involves exponential proliferation of lymphoid or myloid cells. Leukemias is classified by the clinical course into: acute and chronic, and by the cell of origin into: lymphoid or myloid (non-lymphoid). In acute leukemia there is a rapidly progressive disease that result from accumulation of immature, functionless WBCs in the bone marrow and blood, it is more common than chronic leukemia. While in the chronic leukemia there is slower onset and the cells are more mature. There are 4 types of leukemia with many subtypes:

1. Acute Lymphoblastic Leukemia ALL, it is the most common type in children.
2. Acute Mylogenous Leukemia AML, the most common type in adults.
3. Chronic Lymphocytic Leukemia CLL, the second most common type in adults.
4. Chronic Myloid Leukemia CML.

Oral manifestations

Are more common in acute leukemia than in chronic leukemia, they include:

• Localized or generalized gingival enlargement, caused by infiltration of immature WBCs, it occurs in about 35% of acute leukemias and 10% of the chronic leukemias.
• The gingiva bleeds easily, sometimes spontaneously oral hygiene measures and chemotherapy may cause resolution.
• Oral ulcerations.
• Recurrent oral infections, due to the immature WBCs and as a complication of chemotherapy.
• Localized mass of leukemic cells in the gingiva and other site of the oral cavity, it is termed Chloroma (Granulocytic Sarcoma).
• Pallor of oral mucosa.
• LAP.

2- Lymphoma

Lymphoma is a solid malignant tumor that originate in the lymph nodes or extranodal lymphoid tissues in any part of the body. Lymphoma comprises Hodgkin's lymphoma or disease and non-Hodgkin's lymphoma NHL. NHL is more common than the Hodgkin's type.

1. Hodgkin's disease; it is a neoplasm of B lymphocytes; it contains a characteristic tumor cell (Reed Sternberg cell). The cause is unknown but EBV may be implicated. It presents as a painless enlargement of non-tender lymph nodes involving head and neck, axillary, mediastinal or groin lymph nodes. Fever, night sweats, fatigue and weight loss may be experienced by the patient. The diagnosis is based on nodal biopsy and bone marrow aspirate. Medical management requires staging on the basis of history, physical examination, lab. Findings and imaging.
2. Non-Hodgkin's lymphoma; a large group of lymphoproliferative disorders of either B lymphocytes (more than 80% of the cases) or T lymphocytes origin. There are many types of NHL. The cause is unknown but some genetic factors and chromosomal abnormalities in addition to other environmental factors such as infection with EBV, irradiation and drugs were implicated as possible causative factors. The clinical presentation includes; LAP, fever, weight loss, abdominal or chest pain and extranodal tumors. The diagnosis is based on biopsy of the lymph nodes or extranodal tumor. Proper staging is required which consists of blood investigation, imaging and bone marrow biopsy.

Oral manifestations

• Cervical LAP.

• Intraoral tumors that may involve Waldeyer's ring (named after the German anatomist Heinrich Wilhelm Gottfried von Waldeyer Hartz), salivary glands, mandible, palate, gingiva or floor of the mouth.

• Oral ulcerations.

• Oral complications secondary to treatment include; burning mouth symptoms, xerostomia, infections, trismus, impaired craniomandibular growth and osteoradionecrosis.

Burkitt's Lymphoma

It is an aggressive type of B cell NHL. It is the most common lymphoma of children. Types of Burkitt's lymphoma:

1. the endemic or African type.
2. non-endemic type, occurs in western societies.

3. recently described type associated with HIV infected individuals.

Oral manifestations

Include; tumors of the maxilla or mandible that cause bone destruction, mobility of the teeth, pain and paresthesia. On radiograph it appears as an osteolytic lesion with poorly demarcated margins.

Multiple Myeloma

It is a lymphoproliferative disorder that results from overproduction of cloned malignant plasma cells resulting in bony lesions involving the skeletal system.

Oral manifestations

• Painful bony lesions, that appear as osteolytic punched out lesions which may be associated with cortical bone expansion.
• Extramedullary plasma cell tumor.
• Deposition of Amyloid in soft tissues like tongue.
• Osteonecrosis of the bone associated with Bisphosphonates treatment; it usually appears after surgery especially tooth extraction as a painful, non-healing socket. Treatment is directed to limiting the progression of necrosis through debridement, irrigation with antiseptics and antibiotics.

To minimize the likelihood of developing necrosis:

• Early treatment of any source of odontogenic infection preferably before starting treatment with Bisphosphonates.
• Non-surgical approaches are to be preferred.
• If extraction is required it should be as conservative as possible.
• The risk of necrosis should be discussed with the patient.

Dental management of WBCs Dyscrasias

• The clinician attempts to identify and recognize the presence of WBCs disorders through obtaining a thorough history about the signs and symptoms of these disorders, such as easy bruising or bleeding tendency, also family history of WBCs disorders.

• Thorough extraoral and intraoral examination of the head and neck, oral cavity and oropharynx to identify any abnormalities that are suggestive of WBCs disorders. Screening blood investigations may be needed and if the results are abnormal, the patient is referred for further evaluation and routine dental care can be deferred.

Dental management of patients with diagnosed Leukemia, Lymphoma and Multiple Myloma It involves the three phases of the medical therapy:

3. Pretreatment assessment and preparation of the patient: Full knowledge of the patient's condition is required, the aim of this phase is to prevent oral infections, all potential sources of infection must be eliminated through restorative, periodontal and surgical treatment preferably 3 weeks prior to medical treatment. Oral hygiene measures should be encouraged. When extraction is planned it should be as conservative as possible avoiding any hemostatic packing agents and attaining primary closure.

Prophylactic antibiotic is recommended before oral surgical procedure, 2 g oral Penicillin 1 hour before the procedure, 500 mg 4 times daily for 1 week.

Patients with platelet count below 50.000/mm^3 should not undergo oral surgical procedures unless correction by transfusion is carried out.

4. Oral health care during medical treatment: during treatment the patient is susceptible to many oral complications that require care:

• Mucositis; appear 7-10 days after initiation of treatment and resolve after it. The non-keratinized mucosa is more severely affected. Oral hygiene measures should be maintained to minimize infection, antiseptic and antimicrobial mouth washes e.g. Chlorhexidin are recommended, topical anesthetics and systemic analgesics can be given.

• Neutropenia and Infection; neutropenia leads to gingival inflammation, oral ulceration and infection which can be severe but with minimal clinical signs. Unusual bacterial infections, fungal and viral infections occur in patients with Leukemia, Lymphoma and Multiple Myloma on chemotherapy and require treatment. When oral infections develop, a specimen of the exudate should be sent for culture and antibiotic sensitivity tests.

• Bleeding; thrombocytopenia may case submucosal hemorrhage and sometimes spontaneous gingival bleeding, oral hygiene measures should be improved, when bleeding occurs local hemostatic measures should be used first like using pressure, gelatin sponge with thrombin or the use of oral antifibrinolytic agents. If these measures fail transfusion may be needed.

• Graft versus host disease; it occurs after bone marrow transplantation when immunologically active donor T cells react against host tissues, it can be acute (within 2-3 weeks) causing rash, mucosal ulcerations, increased liver enzymes and diarrhea. Or it could be chronic (3-12 months) producing features like Sjögren syndrome, scleroderma, lichenoid changes, xerostomia, mucositis, dysphagia and damage to liver. It can be prevented by corticosteroids and immunosuppressive drugs.
• Adverse effects of drugs; such as gingival overgrowth with patients taking Cyclosporine.
• Disturbance of growth and development; due to treatment with chemotherapy and radiotherapy during childhood leading to micrognathia, malocclusion and teeth abnormalities.

3. Posttreatment management: patients in remission state can have routine dental care while patients with poor prognosis should receive emergency care only. When invasive procedures are planned (e.g. oral surgery), platelet count and bleeding time should be investigated, the patient's physician should be consulted. In patients with surgically removed spleen, prophylactic antibiotic is needed, since they are at risk of bacterial infections, especially in the first 6 months after splenectomy. In patients with acute symptoms, routine dental care should be deferred. LA regional block should be avoided if possible, in patients with bleeding tendency. Conscious sedation can be given and GA is allowed.

ORAL SURGERY

LECTURE 4 **ا.م.د.احمد فاضل ابراهيم القيسي**

Thyroid Diseases

Thyroid disease in a patient who presents for dental treatment is a cause for concern on several fronts. Undiagnosed or poorly controlled thyroid disorders can be expected to compromise outcomes with otherwise perfectly appropriate dental management plans. Detection of early signs and symptoms of such disorders during the dentist's head and neck evaluation can lead to referral of the patient for medical evaluation and treatment. In some instances, such intervention may be lifesaving, and in others, quality of life can be improved and complications of certain thyroid disorders avoided, particularly in the context of delivery of dental care.

Patients with **hyperthyroidism** are predisposed to adverse interaction with epinephrine, life-threatening cardiac arrhythmias, congestive heart failure (CHF), and thyrotoxic crisis (thyroid storm, precipitated by infection or surgical procedures).

Further complications that may occur in patients with **hypothyroidism** are an exaggerated response to central nervous system (CNS) depressants (sedatives and narcotic analgesics) and myxedematous coma precipitated by CNS depressants, infection, or surgical procedures.

- **HYPERTHYROIDISM (THYROTOXICOSIS):**

The term thyrotoxicosis refers to an excess of T4 and T3 in the bloodstream. This excess may be the result of production by ectopic thyroid tissue, multinodular goiter, or thyroid adenoma or may be associated with subacute thyroiditis, pituitary disease involving the anterior portion of the gland, or ingestion of thyroid hormone (thyrotoxicosis factitia) or foodstuffs containing thyroid hormone. When thyrotoxicosis occurs, it is most commonly associated with Graves disease, toxic nodular goiter, or acute thyroiditis.

Clinical Presentation :

Direct and indirect effects of excessive thyroid hormones contribute to the clinical picture in Graves disease. The most common symptoms and signs are nervousness, fatigue, rapid heartbeat or palpitations, heat intolerance, and weight loss. The patient's skin is warm and moist and the complexion rosy; the patient may blush readily. Palmar erythema may be present, profuse sweating is common, and excessive melanin pigmentation of the skin is evident in many patients; however, pigmentation of the oral mucosa has not been reported. In addition, the patient's hair becomes fine and friable, and the nails soften.

ORAL MANIFESTATIONS:

In children, the teeth and jaws develop rapidly, and premature loss of deciduous teeth with early eruption of permanent teeth is common. Euthyroid infants of hyperthyroid mothers have been reported to have erupted teeth at birth. A few patients with thyrotoxicosis have been found to have a lingual "thyroid" consisting of thyroid tissue posterior to the foramen cecum , If the dentist detects a lingual thyroid, assessment by a physician is required before the mass is considered for surgical removal. Osteoporosis involving the alveolar bone may be an associated feature, and development of dental caries and periodontal disease may occur rapidly in these patients. In very early reports, a number of changes were reported affecting taste and smell.

Dental management of the patients with thyroid diseases :

1- **Identification:** Palpation and inspection of the thyroid gland should be included as part of the routine head and neck examination performed by the dentist. The anterior neck region should be inspected for indications of old surgical scars, and the posterior dorsal region of the tongue should be examined for a nodule, which could represent lingual thyroid tissue.
normal thyroid gland can be palpated in many patients. It may feel rubbery and may be more easily identified by having the patient swallow during the examination. An enlarged thyroid gland caused by hyperplasia (goiter) feels softer than the normal gland. Adenomas and carcinomas involving the gland are firmer on palpation and are usually seen as isolated swellings but may appear as multinodular growth. In patients with Hashimoto disease or, the gland is much firmer than normal.
Auscultation should be used to examine for a systolic or continuous bruit that can be heard over the hyperactive gland of thyrotoxicosis or Graves

disease as a result of engorgement of the gland's vascular system. If a thyroid abnormality is detected, even if the patient may appear euthyroid, a referral should be made for medical evaluation before dental treatment is rendered.

2- **Risk Assessment :** it based on the presence or absence of signs and symptoms, clinical features, recent thyroid function tests, and consultation with the physician. Persons undiagnosed or poorly treated hyperthyroid disease and affected older adults are at higher risk for adverse consequences of dental treatment

3- **In Case of Thyrotoxicosis :**

Recommendation

a- Antibiotics and Risk of Infection:

patients with extensive dental caries or periodontal disease should be treated after medical management of the thyroid problem has been instituted.

If acute oral infection occurs in a patient with uncontrolled hyperthyroid disease, consultation with the patient's physician is recommended before initiated dental therapy.

b- Bleeding:

There is little to no risk of bleeding abnormalities in patients with hyperthyroidism except in patients concurrently taking warfarin and propylthiouracil.

c- Capacity to Tolerate Dental Care:

thyrotoxic patient under medical management, dental treatment can proceed without alteration. However, patients with untreated or poorly treated thyrotoxicosis are susceptible to developing an acute medical emergency, called thyrotoxic crisis (thyroid storm).Clinical manifestations include restlessness, fever, tachycardia, pulmonary edema, tremor, sweating, stupor, and, finally, coma and death, if treatment is not provided. Of note, dental surgery performed in these patients may precipitate a thyrotoxic crisis. In addition, acute oral infection has been associated with such events.

d- Drug Considerations:

Use of epinephrine or other pressor amines (in local anesthetics or gingival retraction cords or to control bleeding) must be avoided in untreated or poorly treated thyrotoxic patients. However, well-managed (euthyroid) thyrotoxic patients with thyroid disease require no special consideration in this regard and may be given normal concentrations of these vasoconstrictors.Care must be taken with patients whose disease is being brought under control when the dentist plans to use nonselective beta-blockers. When epinephrine is given to

these patients, it is possible that blood pressure can be increased through inhibition of the vasodilatory action of epinephrine attained through blocking of β2 receptors. Clinical experience has shown that small amounts of epinephrine can be used safely in euthyroid patients. Use of more concentrated preparations of epinephrine (as in retraction cords and preparations used to control bleeding) should be avoided.

Adverse reactions to propylthiouracil include agranulocytosis and leukopenia. If these occur, the patient is at risk for serious infection. The physician should monitor the patient for these adverse reactions. The dentist can consult with the patient's physician or can order a complete blood count to rule out the presence of these complications before undertaking surgical procedures. It has been reported that propylthiouracil can induce sialolith formation. This drug also can increase the anticoagulant effects of warfarin. Aspirin and other nonsteroidal antiinflammatory drugs can increase the amount of circulating T4, making control of thyroid disease more difficult.

e- Emergencies:

If a thyrotoxic crisis occurs, the dentist must recognize the features, begin emergency treatment, and seek immediate medical assistance. The patient can be cooled with cold towels, given an injection of hydrocortisone (100–300 mg), and started on an IV infusion of hypertonic glucose (if equipment is available). Vital signs must be monitored and cardiopulmonary resuscitation initiated, if necessary. Immediate medical assistance should be sought, and when available, other measures such as antithyroid drugs and potassium iodide may be started.

- **HYPOTHYROIDISM**

Hypothyroidism may be congenital or acquired. The acquired form may result from failure of the thyroid gland or pituitary gland and commonly is caused by irradiation of the thyroid gland (RAI), surgical removal, or excessive antithyroid drug therapy.

Subclinical hypothyroidism is a prevalent condition that is characterized by elevated serum TSH concentration and normal serum T4 and T3. Subclinical hypothyroidism secondary to chronic autoimmune thyroiditis has a predictable clinical course.Spontaneous return to normal TSH values occurs in 5% to 6% of cases. Progression to overt hypothyroidism occurs at a rate of about 5% per year. Some patients report fatigue, weight gain, poor memory, poor ability to concentrate, and depressed feelings.

AUTOIMMUNE HYPOTHYROIDISM (HASHIMOTO THYROIDITIS)

This is the most common cause of primary hypothyroidism and it is an autoimmune disorder that manifests most often as an asymptomatic diffuse goiter. High titers of circulating thyroid autoantibodies and thyroid antigen–specific T cells are observed. It usually affects young and middle-aged women and is three to four times more frequent in women than men. By the time the diagnosis has been established, most patients are hypothyroid. A family history of Hashimoto thyroiditis or other autoimmune thyroid disorder often is reported. It may be associated with other autoimmune diseases such as pernicious anemia and type 1 diabetes mellitus.

Signs and Symptoms

Goiter is the clinical hallmark of Hashimoto thyroiditis. The goiter usually is moderate in size and rubbery firm in consistency, and it moves freely with swallowing. Patients may be euthyroid (normal function) during early phases of the disease. Early in the disease course, the thyroid becomes enlarged and firm and may have a nodular consistency. Over time, most patients develop hypothyroidism as lymphocytes replace functioning tissue. In a few cases, the patient develops transient hyperthyroidism, to be followed later by hypothyroidism.

CLINICAL PRESENTATION

Neonatal hypothyroidism is characterized by dwarfism; overweight; well-recognized facial features consisting of a broad flat nose, wide-set eyes, thick lips, and a large protruding tongue; poor muscle tone; pale skin; stubby hands; retarded

bone age; delayed eruption of teeth; malocclusions; a hoarse cry; an umbilical hernia; and mental retardation. All of these abnormalities can be prevented by early detection and treatment. The onset of hypothyroidism in older children and adults is manifested by characteristic changes in physical appearance: a dull expression; puffy eyelids; alopecia of the outer third of the eyebrows; palmar yellowing; dry and rough skin; and dry, brittle, and coarse hair, along with increased size of the tongue. Other features include slowing of physical and mental activity, slurred and hoarse speech, anemia, constipation, increased sensitivity to cold, increased capillary fragility, weight gain, muscle weakness, and deafness. Untreated patients with severe myxedema may develop hypothyroid coma, which usually is fatal.

ORAL MANIFESTATIONS

Hypothyroidism

Infants with hypothyroidism may present with thick lips, enlarged tongue, and delayed eruption of teeth with resulting malocclusion. Adults with acquired hypothyroidism can display an enlarged tongue and low salivary flow.

Hashimoto thyroiditis

It can be accompanied by salivary gland dysfunction, resulting in dry mouth. This may be due to the effect of cytokines in the autoimmune process or because of thyroid hormone dysfunctions. The pain associated with subacute painful thyroiditis may radiate to the ear, jaw, or occipital region. Hoarseness and dysphagia may be accompanying features. Patients may report palpitations, nervousness, and lassitude. On palpation, the thyroid is enlarged, firm, often nodular, and usually very tender.

Dental management :

1- Identification.
Patients with hypothyroidism should be identified when possible because their quality of life can be greatly improved with medical treatment. With detection early in childhood, permanent mental retardation can be avoided with appropriate medical management. In addition, oral complications of delayed eruption of teeth, malocclusion, enlarged tongue, and skeletal retardation can be prevented through early detection and medical treatment.

Recommendations:

2- Antibiotics and Risk of Infection. Acute oral infection in an uncontrolled hypothyroid patient could trigger a myxedema coma; such a patient should receive immediate consultation with the patient's physician as part of the management program.
3- Bleeding. There is little to no risk of bleeding abnormalities in patients with hypothyroidism.
4- Capacity to Tolerate Care. In general, patients with mild symptoms of untreated hypothyroidism are not in danger when receiving dental therapy. Also, when hypothyroid patients are under good medical care, no special problems in terms of dental management remain. However, patients with poorly controlled disease who have infection and older adults with myxedema are at great risk. A myxedematous coma can be precipitated by CNS depressants, surgical procedures, and infections; thus, the major concerns of dental management of patients with this condition are detection and referral for medical management before any dental treatment is rendered.
5- Drug Considerations. CNS depressants, sedatives, and narcotic analgesics may cause an exaggerated response in patients with mild to severe hypothyroidism. These drugs must be avoided in all patients with severe hypothyroidism and must be used with care (reduced dosage) in patients with mild hypothyroidism.
6- Emergencies. If myxedema coma occurs, the dentist should call for medical aid; while waiting for this assistance, the dentist can inject 100 to 300 mg of hydrocortisone, cover the patient to conserve heat, and apply cardiopulmonary resuscitation if indicated. When medical aid becomes available, parental levothyroxine is administered, and IV hypertonic saline and glucose are given as needed.

Complications

Studies suggested an association between thyroid disease (i.e. hypothyroidism or its treatment) and oral lichen planus.

Further more, medications (Radio Active Iodine) used to treat hyperthyroid conditions and thyroid cancer is associated with Acute risks like salivary gland swelling and pain and loss of taste. And longe term complications like recurrent sialoadenitis, hyposalivation, xerostomia, mouth pain, and dental caries

ORAL SURGERY

LECTURE **ا.م.د.احمد فاضل ابراهيم القيسي**

Adrenal gland disorders

The adrenal glands are small endocrine glands located bilaterally at the superior pole of each kidney. Each gland contains an outer cortex and an inner medulla. The adrenal medulla functions as a sympathetic ganglion and secretes catecholamines, primarily epinephrine. The adrenal cortex secretes several steroid hormones (aldosterone, glucocorticoids, androgens, and Cortisol).

Hyperadrenalism is characterized by excessive secretion of adrenal cortisol, mineralocorticoids, androgens, or estrogen in isolation or combination. The most common type of overproduction is due to glucocorticoid excess. When this is caused by pathophysiologic processes, the condition is known as Cushing disease.The term Cushing syndrome is a generalized state caused by excessive cortisol in the body, regardless of the cause

Adrenal insufficiency is divided into three categories: primary, secondary, and tertiary.

Primary adrenocortical insufficiency, also known as Addison disease, occurs when the adrenal cortex is destroyed or the gland is removed.

Secondary adrenocortical insufficiency is the consequence of pituitary disease or a lack of responsiveness of the adrenal glands to ACTH (corticotrophin) or caused by critical illness.

Tertiary adrenal insufficiency results from processes that impair function of the hypothalamus, which is most commonly caused by chronic use of corticosteroids.

Dental Management of Adrenal Gland Disorders

1- **Identification:**
   Any patient whose condition remains undiagnosed but who has cardinal signs and symptoms of adrenal disease should be referred to a physician for diagnosis and treatment.
2- **Risk Assessment:**
   The dentist should be aware that a past or present history of tuberculosis, histoplasmosis, or HIV infection increases the risk for primary adrenal disease (insufficiency) because opportunistic infectious agents may attack

the adrenal glands. In addition, adrenal crisis is more likely in patients with adrenal insufficiency who have the following comorbidities: malignancy, major traumatic injury, severe pain, infection or sepsis, liver cirrhosis, administration of medications that alter cortisol metabolism or production, recent emergency or hospitalization visits, or need for stress-related corticoid dose self-adjustments. If the dentist is uncertain of the functional reserve of the patient, laboratory testing and medical consultation are advised before the performance of an invasive or prolonged (>1 hour) procedure.

3- the dentist must consider the type and degree of adrenal dysfunction and the dental procedure planned.

4- **In Case of Hyperadrenalism:**

Patients with hyperadrenalism or who take corticosteroids for prolonged periods have an increased likelihood of having hypertension, diabetes, delayed wound healing, osteoporosis, and peptic ulcer disease. To minimize the risk of an adverse outcome, blood pressure should be taken at baseline and monitored during dental appointments. Blood glucose levels should be determined, and invasive procedures should be performed during periods of good glucose control. Follow-up appointments should be arranged to assess proper wound healing. Because osteoporosis has a relationship with periodontal bone loss, implant placement, and bone fracture, periodic measures of periodontal bone loss are indicated. Also, measures should be instituted that promote bone mineralization, and extensive neck manipulation should be avoided if osteoporosis is severe. Because of the risk of peptic ulceration, postoperative analgesics for long-term steroid users should not include aspirin and other nonsteroidal antiinflammatory drugs.

5- **In Case Of Adrenal Insufficiency**

- **Bleeding:**

Generally, this is not an issue. An exception is patients who take heparin or an other anticoagulant, which places them at increased risk for adrenal hemorrhage, postsurgical bleeding, and hypotension.

- **Blood Pressure.**

Monitoring of blood pressure throughout invasive dental procedures of patients who have adrenal insufficiency is critical for recognition of a developing adrenal crisis. During surgery, blood pressure should be evaluated at 5-minute intervals and before the patient leaves the office. A systolic blood pressure below 100 mm Hg or a diastolic pressure at or below 60 mm Hg represents hypotension. A diagnosis of hypotension dictates that the clinician must take corrective action. This includes proper

patient positioning (i.e., head lower than feet), fluid replacement, administration of vasopressors, and evaluation for signs of adrenal dysfunction versus hypoglycemia.

- **Capacity to Tolerate Care**:

  This patient population is potentially at risk for an adrenal crisis. The risk is highest in those with primary adrenal insufficiency, especially those who are undiagnosed or untreated. In contrast, patients who have secondary or tertiary adrenal insufficiency are at much lower risk.

  Patients at risk for adrenal crisis are those who have a fever, intercurrent illness, or sustained trauma or who are undergoing stressful surgical procedures or general anesthesia and have no, or extremely low, adrenal function because of primary or severe secondary adrenal insufficiency. It is recommended to delay treatment for these patients and any patient who is undiagnosed or untreated until the patient has been medically stabilized. Dentists should be aware that three factors influence the recommendation for supplemental corticosteroids: (1) type of adrenal insufficiency, (2) medical status and stability, and (3) level and type of stress.

  Currently, only patients with primary adrenal insufficiency are recommended to receive corticosteroid supplementation.Patients with well-controlled secondary adrenal insufficiency and those who take daily or alternate-day corticosteroids generally have enough exogenous and endogenous cortisol to handle routine dental procedures and surgery if their usual steroid dose (or parenteral dose equivalent) is taken the morning of the procedure.Thus, the recommendation is for patients to take their usual daily dose of steroid within 2 hours of the surgical procedure and that the surgeon, anesthetist, and nurses be advised of possible complications associated with the patient's adrenal state. Routine dental procedures do not stimulate cortisol production at levels comparable with those that occur during and after surgery and do not require supplementation, even in patients with controlled primary adrenal insufficiency.

  Patients undergoing surgery should be closely monitored for blood and fluid loss and for hypotension during the postoperative period. If hypotension appears during monitoring, IV fluids are to be given and additional doses of corticosteroid considered if fluid replacement fails to rectify the blood pressure. Patients are returned to their usual glucocorticoid dosage as soon as their vital signs are stabilized. Additional measures recommended to minimize the risk of adrenal crisis associated with surgical stress (Surgery should be scheduled in the morning when cortisol levels are highest, Proper stress reduction should be provided because fear and anxiety increase cortisol demand. Nitrous oxide–oxygen

inhalation and benzodiazepine sedation are helpful in minimizing stress and reducing cortisol demand. In contrast, reversal of and recovery from general anesthesia and extubation, and not the trauma of surgery itself, are major determinants of secretion of ACTH, cortisol, and epinephrine. Thus, general anesthesia increases glucocorticoid demand for these patients. Barbiturates also should be used cautiously because these drugs enhance the metabolism of cortisol and reduce blood levels of cortisol. In addition, inhibitors of corticosteroid production should be discontinued at least 24 hours before surgery, with the consent of the patient's physician. Surgeries that last longer than 1 hour are more stressful than shorter surgeries and should be considered major surgical procedures that can require the need for steroid supplementation.d

- **Drug Considerations and Interactions**. Inadequate pain control during the postoperative period increases the risk of adrenal crisis. Clinicians should provide good postoperative pain control by means of long-acting local anesthetics (e.g., bupivacaine) given at the end of the procedure. as the significant increases in cortisol levels generally are not seen before or during the operation but are increased in the postoperative period (5 hours after the procedure) and the rise in cortisol levels is blunted by the use of analgesics and midazolam, good pain control with local anesthesia and analgesics is recommended for these patients.
- **Emergency Action**. Immediate treatment during an adrenal crisis requires proper patient positioning (i.e., head lower than feet), fluid replacement, administration of vasopressors, administration of 100 mg of hydrocortisone or 4 mg of dexamethasone IV, and immediate transportation to a medical facility.

- **Oral Manifestations**

  Diffuse or focal brown macular pigmentation of the oral mucous membranes is a common finding in primary adrenal insufficiency. Pigmentation of sun-exposed skin in areas of friction generally occurs after the appearance of oral pigmentation and is accompanied by lethargy.Patients with secondary or tertiary adrenal insufficiency may be prone to delayed healing and may have increased susceptibility to infection but do not develop hyperpigmentation.

ORAL SURGERY

LECTURE **ا.م.د.احمد فاضل ابراهيم القيسي**

Diabetes Mellitus

Diabetes mellitus is a group of metabolic diseases characterized by high blood glucose levels (hyperglycemia) and the inability to produce and/or use insulin. The disease is defined by abnormal blood glucose levels and utilization and is classified by the American Diabetes Association (ADA) into four general types. Each type is distinguished by the underlying mechanism, and each type demonstrates different levels of glycemia

BOX 14.1 Current Classification of Diabetes

Type 1	• Beta cell destruction, usually leading to absolute insulin deficiency • Immune mediated: presence of islet cell or insulin antibodies that identify the autoimmune process, leading to beta cell destruction • Idiopathic: no evidence of autoimmunity
Type 2	• Insulin resistance with relative insulin deficiency or insulin secretory defect with insulin resistance
Other specific types	• Genetic defects of beta cell function or insulin action, diseases of exocrine pancreas, endocrinopathies, drug- or chemical-induced diabetes, infections, uncommon forms of immune-mediated diabetes, other genetic syndromes • Impaired fasting glucose (impaired glucose tolerance) • Abnormalities of fasting glucose (abnormal glucose tolerance)
Gestational	• Any degree of abnormal glucose tolerance during pregnancy diabetes

Persistent hyperglycemia leads to metabolic and vascular complications.The vascular complications include premature macrovascular disease and serious microvascular disease. The metabolic component involves the elevation of blood glucose associated with alterations in lipid protein metabolism, resulting from a relative or absolute lack of insulin.

A crucial aspect of care of dental patients who have diabetes is determination of the level of disease severity and the level of glycemic control, as well as the presence of complications from diabetes, so that appropriate dental treatment can be provided. Essential to this determination is knowledge of the patient's blood glucose level at the time that dental treatment is provided.

CLINICAL PRESENTATION

In patients with type 1 diabetes, the onset of symptoms is sudden and acute, often developing over days or weeks.Typically, the diagnosis is made in nonobese children or young adults younger than 40 years of age; however, it may occur at any age. Signs and symptoms include polydipsia, polyuria, polyphagia, weight loss, loss of strength, marked irritability, recurrence of bed wetting, drowsiness, malaise, and blurred vision. Patients also may present with ketoacidosis, which if severe is accompanied by vomiting, abdominal pain, nausea, tachypnea, paralysis, and loss of consciousness.Type 2 diabetes generally occurs after age 40 and more often affects obese individuals.The onset of symptoms in type 2 diabetes usually is insidious, and the cardinal manifestations and symptoms (polydipsia, polyuria, polyphagia, weight loss, and loss of strength) are less commonly seen. Other signs and symptoms related to the complications of diabetes include skin lesions, cataracts, blindness, hypertension, chest pain, and anemia. The rapid onset of myopia in an adult is highly suggestive of diabetes mellitus.

TABLE 14.3 Diagnostic Criteria for Diabetes Mellitus*

1. FPG ≥126 mg/dL (≥7.0 mmol/L) on two occasions. Fasting is defined as no caloric intake for at least 8 hours. This fasting glucose value is consistently associated with the risk for retinopathy.

or

2. Symptoms and signs of diabetes plus casual (random) plasma glucose concentration ≥200 mg/dL (11.1 mmol/L). *Casual* is defined as obtained at any time of day without regard to time since last meal. Many patients do not have obvious symptoms. The cardinal manifestations of diabetes include polyuria, polydipsia, and unexplained weight loss.

or

3. 2-Hour postload glucose ≥200 mg/dL (≥11.1 mmol/L) during an OGTT. The test should be performed as described by the WHO using a glucose load containing the equivalent of 75 g of anhydrous glucose dissolved in water.*

or

4. Glycosylated hemoglobin (by A_{1C} assay) ≥6.5%

TABLE 14.5 American Diabetes Association (ADA) and American College of Endocrinology (ACE): Targets for Glycemia Management

Parameter	Normal	ADA*	ACE
Premeal plasma glucose (mg/dL)	<100 (mean ≈90)	90–130	<110
Postprandial plasma glucose* (mg/dL)	<140	<180	<140
A_{1C}	4%–6%	<7%†	<6.5%

DENTAL MANAGEMENT

1- Any dental patient whose condition remains undiagnosed but who has the cardinal signs and symptoms of diabetes (i.e., polydipsia, polyuria, polyphagia, weight loss, and weakness) should be referred to a physician for diagnosis and treatment.

2- Patients with findings suggestive of diabetes (headache, dry mouth, marked irritability, repeated skin infection, blurred vision, paresthesias, progressive periodontal disease, multiple periodontal abscesses, loss of sensation) should be referred to a clinical laboratory or to a physician for screening tests to determine if diabetes mellitus type 1 or type 2 or another type of diabetes is responsible for their symptoms.

3- Patients with an estimated fasting blood glucose level of 126 mg/100 mL or higher should be referred to a physician for medical evaluation and treatment, if indicated. Those with a 2-hour postprandial blood glucose level of 200 mg/100 mL or higher also should be referred.

4- Patients who are obese, who are older than 45 years of age, or who have close relatives with diabetes should be screened routinely (at least at 3-year intervals) for any indication of hyperglycemia that may reveal the onset of diabetes.

5- Women who have given birth to large babies (birth weight >9 lb) or who have had multiple spontaneous abortions or stillbirths also should be screened once a year for diabetes.

6- All patients with diagnosed diabetes must be identified by history, and the type of medical treatment they are receiving must be established.

7- The type of diabetes (type 1, type 2, other) should be determined and the presence of complications noted.

8- Patients who are being treated with insulin should be asked how much insulin they use, how often they inject themselves each day, the frequency of insulin reactions and when the last one occurred, how often the patient self-monitors her or his blood glucose levels.
9- Patients with abnormal pulse rate and rhythm or elevated blood pressure should be approached with caution. As the risk for serious cardiovascular events increases substantially in those patients
10- **Analgesics**: Aspirin and nonsteroidal antiinflammatory drugs can potentially enhance the efficacy of some oral hypoglycemic agents (sulfonylureas) and enhance hypoglycemia; thus, they should be used judiciously.
11- **Prophylactic Antibiotics**: The decision to use antibiotic prophylaxis or coverage typically involves consultation with the patient's physician and is related to poor glycemic control. Patients who have brittle diabetes (in which control is very difficult to achieve) or who require a high dosage of insulin (in type 1 diabetes) and are undergoing an invasive procedure may be at increased risk for postoperative infection. However, prophylactic antibiotics usually are not indicated. If the patient develops an infection, appropriate systemic antibiotics may be given.
12- **The risk for infection in patients with diabetes**, in theory, the rsik is directly related to fasting blood glucose levels, presence of infecting organisms, and invasiveness of dental procedures. As indicated by data for general surgery procedures, if the fasting blood glucose level is below 206 mg/100 mL, increased risk is not predicted. However, if the fasting blood glucose level is between 207 and 229 mg/100 mL, the risk is predicted to be increased by 20% if surgical procedures are being performed. Additionally, if the fasting blood glucose level rises to above 230 mg/100 mL, an 80% increased risk of infection postoperatively has been reported.

13- **Management of infection:** Patients who are receiving insulin usually require additional insulin, which should be prescribed by their physicians. Non–insulin-controlled patients may need more aggressive medical management of their diabetes, which may include insulin during this period. The dentist should treat infection aggressively by incision and drainage, extraction, pulpotomy, warm rinses, and antibiotics. Antibiotic sensitivity testing is recommended for patients with brittle diabetes and for those who require a high insulin dosage for control. For these patients, penicillin therapy can be initiated. Then, if the clinical response is poor, a more effective antibiotic can be selected on the basis of results of antibiotic

sensitivity testing. Attention also should be paid to the patient's electrolyte balance and to fluid and dietary needs.

14- **Anesthetics:** For most patients with diabetes, routine use of local anesthetic with 1:100,000 epinephrine is well tolerated. However, epinephrine has a pharmacologic effect that is opposite that of insulin, so blood glucose could rise with the use of epinephrine. In diabetic patients with hypertension, history of recent \MI, or cardiac arrhythmia, caution may be indicated with use of epinephrine. Guidelines for these patients are similar to those for patients with cardiovascular conditions.

15- **Complications:** A patient with diabetes who is receiving good medical management and demonstrates good glycemic control without serious complications such as renal disease, hypertension, or coronary atherosclerotic heart disease can undergo any indicated dental treatment. If diabetes is under good control, even cardiac transplantation can be safely performed.In patients with diabetes who have serious medical complications

16- **Insulin shock:** A major goal in the dental management of patients with diabetes who are being treated with insulin is to *prevent insulin shock during the dental appointment*. Patients should be told to take their usual insulin dosage **and** to eat normal meals before the appointment, which usually is best scheduled in the morning. When such a patient arrives, the dentist should confirm that the patient has taken insulin and has eaten breakfast. In addition, patients should be instructed to tell the dentist whether at any time during the appointment they are experiencing symptoms of an insulin reaction. A source of sugar such as orange juice, cake icing, or nondiet soft drink must be available in the dental office to be given to the patient if symptoms of an insulin reaction develop

17- **Consultation**: Patients who have not seen their physician recently (within the previous 6 months), who have had frequent episodes of insulin shock, or who report signs and symptoms of diabetes may have disease that is unstable. These patients should be referred to their physicians for evaluation, or their physicians should be consulted to establish their current status.

18- **Diet:** Any patient with diabetes who is going to undergo extensive periodontal or oral surgery procedures other than single simple extractions should be given special dietary instructions for after surgery. It is important that the total caloric content and the protein–carbohydrate– fat ratio of the diet remain the same so that control of the disease and proper blood glucose balance are maintained. One suggestion is to have the patient use a blender

to prepare his or her usual diet so that it can be ingested with minimum discomfort; alternatively, special food supplements in a liquid form may be used.

19- **A protocol for intravenous sedation**: it often involves fasting before the appointment (i.e., nothing by mouth after midnight), using only half the usual insulin dose, and then supplementing with intravenous glucose during the procedure. Patients with well-controlled diabetes may be given general anesthesia if necessary. However, management with local anesthetics is preferable, especially in outpatient office settings.

20- **Devices:** If an insulin pump is being used by the patient, ensure that it is working properly. Antibiotic prophylaxis is not indicated.

21- **Drugs:** Some patients with type 1 diabetes who are being treated with large doses of insulin (in some cases, type 2 diabetes) experience periods of extreme hyperglycemia and hypoglycemia (brittle diabetes) even when given the best of medical management. For these patients, close consultation with the physician is required before any dental treatment is started. Certain drugs used in dentistry can alter blood glucose and interfere with the action of several drugs used to treat diabetes (insulin). See Table below.

TABLE 14.7 Noninsulin Antidiabetic Drugs

Class Drug	Mechanism of Action (Target Tissue)	Principal Adverse Effects	Drug Interaction(s)
SULFONYLUREAS			
Administer 30 minutes before meals.			
First Generation			
Chlorpropamide (Diabinese, Insulase) Acetohexamide (Dymelor) Tolazamide (Tolinase) Tolbutamide (Orinase)	Enhance insulin secretion (beta cells)	Hypoglycemia, weight gain, hyperinsulinemia	Salicylates and ketoconazole increase hypoglycemia.
Second-Generation			
Glipizide (Glucotrol, Glucotrol XL) Glyburide (Micronase, Glynase, DiaBeta) Glimepiride (Amaryl)	Enhance insulin secretion (beta cells)	Hypoglycemia, weight gain, hyperinsulinemia	Corticosteroids decrease action.
BIGUANIDES			
Administer with meals. Metformin (Foramet)	Reduce glucose production*	GI disturbances (abdominal pain, nausea, diarrhea), lactic acidosis	—
α-GLUCOSIDASE INHIBITORS			
Administer just before meals. Acarbose (Precose) Miglitol (Glyset)	Delay carbohydrate digestion (gut)	GI disturbances (abdominal pain, nausea, diarrhea), liver function test elevation	—
THIAZOLIDINEDIONES GLITAZONES			
Administer with meals. Pioglitazone (Actos) Rosiglitazone (Avandia)	Improves insulin sensitivity (fat, muscle)	Headache, weight gain, flatulence Causes or exacerbates heart failure, decreased hemoglobin or hematocrit	—
SODIUM GLUCOSE CO-TRANSPORTER 2 INHIBITORS (SLGT2 AGENTS)			
Dapagliflozin (Farxiga), Canagliflozin (Invokana), empagliflozin (Jardiance)	Selectively inhibiting this co-transporter, which is expressed almost exclusively in the proximal, convoluted tubule in the kidney	Headache, weight gain, flatulence	May increase hypoglycemia
GLINIDES			
Administer 15 minutes before meals. Repaglinide (Prandin) Nateglinide (Starlix)	Enhance insulin secretion (beta cells)	Hypoglycemia (less than sulfonylureas), weight gain, hyperinsulinemia, hypersensitivity, increased uric acid levels	Increased risk of hypoglycemia with salicylates, nonselective beta blockers, NSAIDs Metabolism may be inhibited by azoles, erythromycin
INCRETIN (GLP-1) ANALOGUES			
Administer 15 minutes before meals. Exenatide (Byetta) *Injected subcutaneously* Liraglutide (Victoza) *Injected subcutaneously*	Enhance insulin secretion (beta cells), delay gastric emptying (gut), suppress prandial glucagon secretion	GI adverse effects (nausea, vomiting, diarrhea)	—
AMYLIN ANALOGUE			
Administer before meals. Pramlintide (Symlin) injected subcutaneously	Aids absorption of glucose by slowing gastric emptying (gut), promotes satiety (hypothalamic receptors)	GI disturbances, headache	Avoid anticholinergics that alter GI motility. Can delay absorption of oral medications; administer oral hypoglycemic agents 1–2 hr after Symlin
DIPEPTIDYL PEPTIDASE-4 INHIBITORS			
Administer once daily regardless of meals. Linagliptin (Tradjenta)	Inhibits enzymatic breakdown of GLP-1 and GIP; increases insulin secretion; decreases glucagon secretion (pancreas)	Runny nose, headache	Hypoglycemia may occur when combined with insulin or sulfonylurea drugs.
Saxagliptin (Onglyza)		Peripheral edema	—
Sitagliptin (Januvia)		Headache	
COMBINATION DRUGS			
Some combination drugs include glyburide and metformin (Glucovance), glipizide and metformin (Metaglip), and pioglitazone hydrochloride and glimepiride (Duetact).			

Oral Complications and Manifestations

Oral complications of poorly controlled diabetes mellitus may include xerostomia; bacterial, viral, and fungal infections (including candidiasis); poor wound healing; increased incidence and severity of caries; gingivitis and periodontal disease; periapical abscesses; and burning mouth symptoms.

Oral findings in patients with uncontrolled diabetes most likely relate to excessive loss of fluids through urination, altered response to infection; microvascular changes; and, possibly, increased glucose concentrations in saliva.

The effects of hyperglycemia lead to increased amounts of urine, which deplete the extracellular fluids and reduce the secretion of saliva, resulting in dry mouth. A high percentage of patients with diabetes present with xerostomia and low levels of salivary calcium, phosphate, and fluoride.

Saliva glucose levels are elevated in persons with uncontrolled and controlled diabetes. Several studies have reported increased incidence and severity of gingival inflammation, periodontal abscess, and chronic periodontal disease in patients with diabetes.

Diabetes results in enhanced inflammatory responses, depressed wound healing, and small blood vessel changes that contribute to an increased risk for periodontitis. Thus, it is not surprising that adults with uncontrolled diabetes have more severe manifestations of periodontal disease than do adults without diabetes. Periodontal disease found in these young adults (older than 30 years of age) usually is asymptomatic and typically remains undetected. Overall, periodontal disease is more severe and more frequent in patients with poorly controlled diabetes.

Caries appears to be more significant in patients with diabetes who have poor glycemic control.

Oral fungal infections, including candidiasis and the more rare mucormycosis, may be noted in the patient with uncontrolled diabetes. The general consensus is that healing is delayed in persons with uncontrolled diabetes and that they are more prone to various oral infections after undergoing surgical procedures.

Oral lesions are more common in patients with diabetes. A significantly higher percentage of oral lesions, especially candidiasis, traumatic ulcers, lichen planus, and delayed healing, have been noted in patients with type 1 diabetes, compared with a control population. Altered immune system function contributes to the appearance of these lesions in diabetes.

Diabetic neuropathy may lead to oral symptoms of paresthesias and tingling, numbness, burning, or pain caused by pathologic changes involving nerves in the oral region. Diabetes has been associated with oral burning symptoms. Early diagnosis and treatment of diabetes may lead to regression of these symptoms, but in long-standing cases, the changes may be irreversible.

The drug used for treatment lik Metformin is associated with a metallic taste.

-The End-

ORAL SURGERY

LECTURE 4 **ا.م.د.احمد فاضل ابراهيم القيسي**

Thyroid Diseases

Thyroid disease in a patient who presents for dental treatment is a cause for concern on several fronts. Undiagnosed or poorly controlled thyroid disorders can be expected to compromise outcomes with otherwise perfectly appropriate dental management plans. Detection of early signs and symptoms of such disorders during the dentist's head and neck evaluation can lead to referral of the patient for medical evaluation and treatment. In some instances, such intervention may be lifesaving, and in others, quality of life can be improved and complications of certain thyroid disorders avoided, particularly in the context of delivery of dental care.

Patients with **hyperthyroidism** are predisposed to adverse interaction with epinephrine, life-threatening cardiac arrhythmias, congestive heart failure (CHF), and thyrotoxic crisis (thyroid storm, precipitated by infection or surgical procedures).

Further complications that may occur in patients with **hypothyroidism** are an exaggerated response to central nervous system (CNS) depressants (sedatives and narcotic analgesics) and myxedematous coma precipitated by CNS depressants, infection, or surgical procedures.

- **HYPERTHYROIDISM (THYROTOXICOSIS):**

The term thyrotoxicosis refers to an excess of T4 and T3 in the bloodstream. This excess may be the result of production by ectopic thyroid tissue, multinodular goiter, or thyroid adenoma or may be associated with subacute thyroiditis, pituitary disease involving the anterior portion of the gland, or ingestion of thyroid hormone (thyrotoxicosis factitia) or foodstuffs containing thyroid hormone. When thyrotoxicosis occurs, it is most commonly associated with Graves disease, toxic nodular goiter, or acute thyroiditis.

Clinical Presentation :

Direct and indirect effects of excessive thyroid hormones contribute to the clinical picture in Graves disease. The most common symptoms and signs are nervousness, fatigue, rapid heartbeat or palpitations, heat intolerance, and weight loss. The patient's skin is warm and moist and the complexion rosy; the patient may blush readily. Palmar erythema may be present, profuse sweating is common, and excessive melanin pigmentation of the skin is evident in many patients; however, pigmentation of the oral mucosa has not been reported. In addition, the patient's hair becomes fine and friable, and the nails soften.

ORAL MANIFESTATIONS:

In children, the teeth and jaws develop rapidly, and premature loss of deciduous teeth with early eruption of permanent teeth is common. Euthyroid infants of hyperthyroid mothers have been reported to have erupted teeth at birth. A few patients with thyrotoxicosis have been found to have a lingual "thyroid" consisting of thyroid tissue posterior to the foramen cecum , If the dentist detects a lingual thyroid, assessment by a physician is required before the mass is considered for surgical removal. Osteoporosis involving the alveolar bone may be an associated feature, and development of dental caries and periodontal disease may occur rapidly in these patients. In very early reports, a number of changes were reported affecting taste and smell.

Dental management of the patients with thyroid diseases :

1- **Identification:** Palpation and inspection of the thyroid gland should be included as part of the routine head and neck examination performed by the dentist. The anterior neck region should be inspected for indications of old surgical scars, and the posterior dorsal region of the tongue should be examined for a nodule, which could represent lingual thyroid tissue.
normal thyroid gland can be palpated in many patients. It may feel rubbery and may be more easily identified by having the patient swallow during the examination. An enlarged thyroid gland caused by hyperplasia (goiter) feels softer than the normal gland. Adenomas and carcinomas involving the gland are firmer on palpation and are usually seen as isolated swellings but may appear as multinodular growth. In patients with Hashimoto disease or, the gland is much firmer than normal.
Auscultation should be used to examine for a systolic or continuous bruit that can be heard over the hyperactive gland of thyrotoxicosis or Graves

disease as a result of engorgement of the gland's vascular system. If a thyroid abnormality is detected, even if the patient may appear euthyroid, a referral should be made for medical evaluation before dental treatment is rendered.

2- **Risk Assessment :** it based on the presence or absence of signs and symptoms, clinical features, recent thyroid function tests, and consultation with the physician. Persons undiagnosed or poorly treated hyperthyroid disease and affected older adults are at higher risk for adverse consequences of dental treatment

3- **In Case of Thyrotoxicosis :**

Recommendation

a- Antibiotics and Risk of Infection:

patients with extensive dental caries or periodontal disease should be treated after medical management of the thyroid problem has been instituted.

If acute oral infection occurs in a patient with uncontrolled hyperthyroid disease, consultation with the patient's physician is recommended before initiated dental therapy.

b- Bleeding:

There is little to no risk of bleeding abnormalities in patients with hyperthyroidism except in patients concurrently taking warfarin and propylthiouracil.

c- Capacity to Tolerate Dental Care:

thyrotoxic patient under medical management, dental treatment can proceed without alteration. However, patients with untreated or poorly treated thyrotoxicosis are susceptible to developing an acute medical emergency, called thyrotoxic crisis (thyroid storm).Clinical manifestations include restlessness, fever, tachycardia, pulmonary edema, tremor, sweating, stupor, and, finally, coma and death, if treatment is not provided. Of note, dental surgery performed in these patients may precipitate a thyrotoxic crisis. In addition, acute oral infection has been associated with such events.

d- Drug Considerations:

Use of epinephrine or other pressor amines (in local anesthetics or gingival retraction cords or to control bleeding) must be avoided in untreated or poorly treated thyrotoxic patients. However, well-managed (euthyroid) thyrotoxic patients with thyroid disease require no special consideration in this regard and may be given normal concentrations of these vasoconstrictors.Care must be taken with patients whose disease is being brought under control when the dentist plans to use nonselective beta-blockers. When epinephrine is given to

these patients, it is possible that blood pressure can be increased through inhibition of the vasodilatory action of epinephrine attained through blocking of β2 receptors. Clinical experience has shown that small amounts of epinephrine can be used safely in euthyroid patients. Use of more concentrated preparations of epinephrine (as in retraction cords and preparations used to control bleeding) should be avoided.

Adverse reactions to propylthiouracil include agranulocytosis and leukopenia. If these occur, the patient is at risk for serious infection. The physician should monitor the patient for these adverse reactions. The dentist can consult with the patient's physician or can order a complete blood count to rule out the presence of these complications before undertaking surgical procedures. It has been reported that propylthiouracil can induce sialolith formation. This drug also can increase the anticoagulant effects of warfarin. Aspirin and other nonsteroidal antiinflammatory drugs can increase the amount of circulating T4, making control of thyroid disease more difficult.

e- Emergencies:

If a thyrotoxic crisis occurs, the dentist must recognize the features, begin emergency treatment, and seek immediate medical assistance. The patient can be cooled with cold towels, given an injection of hydrocortisone (100–300 mg), and started on an IV infusion of hypertonic glucose (if equipment is available). Vital signs must be monitored and cardiopulmonary resuscitation initiated, if necessary. Immediate medical assistance should be sought, and when available, other measures such as antithyroid drugs and potassium iodide may be started.

- **HYPOTHYROIDISM**

Hypothyroidism may be congenital or acquired. The acquired form may result from failure of the thyroid gland or pituitary gland and commonly is caused by irradiation of the thyroid gland (RAI), surgical removal, or excessive antithyroid drug therapy.

Subclinical hypothyroidism is a prevalent condition that is characterized by elevated serum TSH concentration and normal serum T4 and T3. Subclinical hypothyroidism secondary to chronic autoimmune thyroiditis has a predictable clinical course.Spontaneous return to normal TSH values occurs in 5% to 6% of cases. Progression to overt hypothyroidism occurs at a rate of about 5% per year. Some patients report fatigue, weight gain, poor memory, poor ability to concentrate, and depressed feelings.

AUTOIMMUNE HYPOTHYROIDISM (HASHIMOTO THYROIDITIS)

This is the most common cause of primary hypothyroidism and it is an autoimmune disorder that manifests most often as an asymptomatic diffuse goiter. High titers of circulating thyroid autoantibodies and thyroid antigen–specific T cells are observed. It usually affects young and middle-aged women and is three to four times more frequent in women than men. By the time the diagnosis has been established, most patients are hypothyroid. A family history of Hashimoto thyroiditis or other autoimmune thyroid disorder often is reported. It may be associated with other autoimmune diseases such as pernicious anemia and type 1 diabetes mellitus.

Signs and Symptoms

Goiter is the clinical hallmark of Hashimoto thyroiditis. The goiter usually is moderate in size and rubbery firm in consistency, and it moves freely with swallowing. Patients may be euthyroid (normal function) during early phases of the disease. Early in the disease course, the thyroid becomes enlarged and firm and may have a nodular consistency. Over time, most patients develop hypothyroidism as lymphocytes replace functioning tissue. In a few cases, the patient develops transient hyperthyroidism, to be followed later by hypothyroidism.

CLINICAL PRESENTATION

Neonatal hypothyroidism is characterized by dwarfism; overweight; well-recognized facial features consisting of a broad flat nose, wide-set eyes, thick lips, and a large protruding tongue; poor muscle tone; pale skin; stubby hands; retarded

bone age; delayed eruption of teeth; malocclusions; a hoarse cry; an umbilical hernia; and mental retardation. All of these abnormalities can be prevented by early detection and treatment. The onset of hypothyroidism in older children and adults is manifested by characteristic changes in physical appearance: a dull expression; puffy eyelids; alopecia of the outer third of the eyebrows; palmar yellowing; dry and rough skin; and dry, brittle, and coarse hair, along with increased size of the tongue. Other features include slowing of physical and mental activity, slurred and hoarse speech, anemia, constipation, increased sensitivity to cold, increased capillary fragility, weight gain, muscle weakness, and deafness. Untreated patients with severe myxedema may develop hypothyroid coma, which usually is fatal.

ORAL MANIFESTATIONS

Hypothyroidism

Infants with hypothyroidism may present with thick lips, enlarged tongue, and delayed eruption of teeth with resulting malocclusion. Adults with acquired hypothyroidism can display an enlarged tongue and low salivary flow.

Hashimoto thyroiditis

It can be accompanied by salivary gland dysfunction, resulting in dry mouth. This may be due to the effect of cytokines in the autoimmune process or because of thyroid hormone dysfunctions. The pain associated with subacute painful thyroiditis may radiate to the ear, jaw, or occipital region. Hoarseness and dysphagia may be accompanying features. Patients may report palpitations, nervousness, and lassitude. On palpation, the thyroid is enlarged, firm, often nodular, and usually very tender.

Dental management :

1- Identification.
Patients with hypothyroidism should be identified when possible because their quality of life can be greatly improved with medical treatment. With detection early in childhood, permanent mental retardation can be avoided with appropriate medical management. In addition, oral complications of delayed eruption of teeth, malocclusion, enlarged tongue, and skeletal retardation can be prevented through early detection and medical treatment.

Recommendations:

2- Antibiotics and Risk of Infection. Acute oral infection in an uncontrolled hypothyroid patient could trigger a myxedema coma; such a patient should receive immediate consultation with the patient's physician as part of the management program.
3- Bleeding. There is little to no risk of bleeding abnormalities in patients with hypothyroidism.
4- Capacity to Tolerate Care. In general, patients with mild symptoms of untreated hypothyroidism are not in danger when receiving dental therapy. Also, when hypothyroid patients are under good medical care, no special problems in terms of dental management remain. However, patients with poorly controlled disease who have infection and older adults with myxedema are at great risk. A myxedematous coma can be precipitated by CNS depressants, surgical procedures, and infections; thus, the major concerns of dental management of patients with this condition are detection and referral for medical management before any dental treatment is rendered.
5- Drug Considerations. CNS depressants, sedatives, and narcotic analgesics may cause an exaggerated response in patients with mild to severe hypothyroidism. These drugs must be avoided in all patients with severe hypothyroidism and must be used with care (reduced dosage) in patients with mild hypothyroidism.
6- Emergencies. If myxedema coma occurs, the dentist should call for medical aid; while waiting for this assistance, the dentist can inject 100 to 300 mg of hydrocortisone, cover the patient to conserve heat, and apply cardiopulmonary resuscitation if indicated. When medical aid becomes available, parental levothyroxine is administered, and IV hypertonic saline and glucose are given as needed.

Complications

Studies suggested an association between thyroid disease (i.e. hypothyroidism or its treatment) and oral lichen planus.

Further more, medications (Radio Active Iodine) used to treat hyperthyroid conditions and thyroid cancer is associated with Acute risks like salivary gland swelling and pain and loss of taste. And longe term complications like recurrent sialoadenitis, hyposalivation, xerostomia, mouth pain, and dental caries

ORAL SURGERY

LECTURE **ا.م.د.احمد فاضل ابراهيم القيسي**

Adrenal gland disorders

The adrenal glands are small endocrine glands located bilaterally at the superior pole of each kidney. Each gland contains an outer cortex and an inner medulla. The adrenal medulla functions as a sympathetic ganglion and secretes catecholamines, primarily epinephrine. The adrenal cortex secretes several steroid hormones (aldosterone, glucocorticoids, androgens, and Cortisol).

Hyperadrenalism is characterized by excessive secretion of adrenal cortisol, mineralocorticoids, androgens, or estrogen in isolation or combination. The most common type of overproduction is due to glucocorticoid excess. When this is caused by pathophysiologic processes, the condition is known as Cushing disease.The term Cushing syndrome is a generalized state caused by excessive cortisol in the body, regardless of the cause

Adrenal insufficiency is divided into three categories: primary, secondary, and tertiary.

Primary adrenocortical insufficiency, also known as Addison disease, occurs when the adrenal cortex is destroyed or the gland is removed.

Secondary adrenocortical insufficiency is the consequence of pituitary disease or a lack of responsiveness of the adrenal glands to ACTH (corticotrophin) or caused by critical illness.

Tertiary adrenal insufficiency results from processes that impair function of the hypothalamus, which is most commonly caused by chronic use of corticosteroids.

Dental Management of Adrenal Gland Disorders

1- **Identification:**
   Any patient whose condition remains undiagnosed but who has cardinal signs and symptoms of adrenal disease should be referred to a physician for diagnosis and treatment.
2- **Risk Assessment:**
   The dentist should be aware that a past or present history of tuberculosis, histoplasmosis, or HIV infection increases the risk for primary adrenal disease (insufficiency) because opportunistic infectious agents may attack

the adrenal glands. In addition, adrenal crisis is more likely in patients with adrenal insufficiency who have the following comorbidities: malignancy, major traumatic injury, severe pain, infection or sepsis, liver cirrhosis, administration of medications that alter cortisol metabolism or production, recent emergency or hospitalization visits, or need for stress-related corticoid dose self-adjustments. If the dentist is uncertain of the functional reserve of the patient, laboratory testing and medical consultation are advised before the performance of an invasive or prolonged (>1 hour) procedure.

3- the dentist must consider the type and degree of adrenal dysfunction and the dental procedure planned.

4- **In Case of Hyperadrenalism:**

Patients with hyperadrenalism or who take corticosteroids for prolonged periods have an increased likelihood of having hypertension, diabetes, delayed wound healing, osteoporosis, and peptic ulcer disease. To minimize the risk of an adverse outcome, blood pressure should be taken at baseline and monitored during dental appointments. Blood glucose levels should be determined, and invasive procedures should be performed during periods of good glucose control. Follow-up appointments should be arranged to assess proper wound healing. Because osteoporosis has a relationship with periodontal bone loss, implant placement, and bone fracture, periodic measures of periodontal bone loss are indicated. Also, measures should be instituted that promote bone mineralization, and extensive neck manipulation should be avoided if osteoporosis is severe. Because of the risk of peptic ulceration, postoperative analgesics for long-term steroid users should not include aspirin and other nonsteroidal antiinflammatory drugs.

5- **In Case Of Adrenal Insufficiency**

- **Bleeding:**

Generally, this is not an issue. An exception is patients who take heparin or an other anticoagulant, which places them at increased risk for adrenal hemorrhage, postsurgical bleeding, and hypotension.

- **Blood Pressure.**

Monitoring of blood pressure throughout invasive dental procedures of patients who have adrenal insufficiency is critical for recognition of a developing adrenal crisis. During surgery, blood pressure should be evaluated at 5-minute intervals and before the patient leaves the office. A systolic blood pressure below 100 mm Hg or a diastolic pressure at or below 60 mm Hg represents hypotension. A diagnosis of hypotension dictates that the clinician must take corrective action. This includes proper

patient positioning (i.e., head lower than feet), fluid replacement, administration of vasopressors, and evaluation for signs of adrenal dysfunction versus hypoglycemia.

- **Capacity to Tolerate Care**:

  This patient population is potentially at risk for an adrenal crisis. The risk is highest in those with primary adrenal insufficiency, especially those who are undiagnosed or untreated. In contrast, patients who have secondary or tertiary adrenal insufficiency are at much lower risk.

  Patients at risk for adrenal crisis are those who have a fever, intercurrent illness, or sustained trauma or who are undergoing stressful surgical procedures or general anesthesia and have no, or extremely low, adrenal function because of primary or severe secondary adrenal insufficiency. It is recommended to delay treatment for these patients and any patient who is undiagnosed or untreated until the patient has been medically stabilized. Dentists should be aware that three factors influence the recommendation for supplemental corticosteroids: (1) type of adrenal insufficiency, (2) medical status and stability, and (3) level and type of stress.

  Currently, only patients with primary adrenal insufficiency are recommended to receive corticosteroid supplementation.Patients with well-controlled secondary adrenal insufficiency and those who take daily or alternate-day corticosteroids generally have enough exogenous and endogenous cortisol to handle routine dental procedures and surgery if their usual steroid dose (or parenteral dose equivalent) is taken the morning of the procedure.Thus, the recommendation is for patients to take their usual daily dose of steroid within 2 hours of the surgical procedure and that the surgeon, anesthetist, and nurses be advised of possible complications associated with the patient's adrenal state. Routine dental procedures do not stimulate cortisol production at levels comparable with those that occur during and after surgery and do not require supplementation, even in patients with controlled primary adrenal insufficiency.

  Patients undergoing surgery should be closely monitored for blood and fluid loss and for hypotension during the postoperative period. If hypotension appears during monitoring, IV fluids are to be given and additional doses of corticosteroid considered if fluid replacement fails to rectify the blood pressure. Patients are returned to their usual glucocorticoid dosage as soon as their vital signs are stabilized. Additional measures recommended to minimize the risk of adrenal crisis associated with surgical stress (Surgery should be scheduled in the morning when cortisol levels are highest, Proper stress reduction should be provided because fear and anxiety increase cortisol demand. Nitrous oxide–oxygen

inhalation and benzodiazepine sedation are helpful in minimizing stress and reducing cortisol demand. In contrast, reversal of and recovery from general anesthesia and extubation, and not the trauma of surgery itself, are major determinants of secretion of ACTH, cortisol, and epinephrine. Thus, general anesthesia increases glucocorticoid demand for these patients. Barbiturates also should be used cautiously because these drugs enhance the metabolism of cortisol and reduce blood levels of cortisol. In addition, inhibitors of corticosteroid production should be discontinued at least 24 hours before surgery, with the consent of the patient's physician. Surgeries that last longer than 1 hour are more stressful than shorter surgeries and should be considered major surgical procedures that can require the need for steroid supplementation.d

- **Drug Considerations and Interactions**. Inadequate pain control during the postoperative period increases the risk of adrenal crisis. Clinicians should provide good postoperative pain control by means of long-acting local anesthetics (e.g., bupivacaine) given at the end of the procedure. as the significant increases in cortisol levels generally are not seen before or during the operation but are increased in the postoperative period (5 hours after the procedure) and the rise in cortisol levels is blunted by the use of analgesics and midazolam, good pain control with local anesthesia and analgesics is recommended for these patients.
- **Emergency Action**. Immediate treatment during an adrenal crisis requires proper patient positioning (i.e., head lower than feet), fluid replacement, administration of vasopressors, administration of 100 mg of hydrocortisone or 4 mg of dexamethasone IV, and immediate transportation to a medical facility.

- **Oral Manifestations**

  Diffuse or focal brown macular pigmentation of the oral mucous membranes is a common finding in primary adrenal insufficiency. Pigmentation of sun-exposed skin in areas of friction generally occurs after the appearance of oral pigmentation and is accompanied by lethargy.Patients with secondary or tertiary adrenal insufficiency may be prone to delayed healing and may have increased susceptibility to infection but do not develop hyperpigmentation.

ORAL SURGERY

LECTURE **ا.م.د.احمد فاضل ابراهيم القيسي**

Diabetes Mellitus

Diabetes mellitus is a group of metabolic diseases characterized by high blood glucose levels (hyperglycemia) and the inability to produce and/or use insulin. The disease is defined by abnormal blood glucose levels and utilization and is classified by the American Diabetes Association (ADA) into four general types. Each type is distinguished by the underlying mechanism, and each type demonstrates different levels of glycemia

BOX 14.1 Current Classification of Diabetes

Type 1	• Beta cell destruction, usually leading to absolute insulin deficiency • Immune mediated: presence of islet cell or insulin antibodies that identify the autoimmune process, leading to beta cell destruction • Idiopathic: no evidence of autoimmunity
Type 2	• Insulin resistance with relative insulin deficiency or insulin secretory defect with insulin resistance
Other specific types	• Genetic defects of beta cell function or insulin action, diseases of exocrine pancreas, endocrinopathies, drug- or chemical-induced diabetes, infections, uncommon forms of immune-mediated diabetes, other genetic syndromes • Impaired fasting glucose (impaired glucose tolerance) • Abnormalities of fasting glucose (abnormal glucose tolerance)
Gestational	• Any degree of abnormal glucose tolerance during pregnancy diabetes

Persistent hyperglycemia leads to metabolic and vascular complications.The vascular complications include premature macrovascular disease and serious microvascular disease. The metabolic component involves the elevation of blood glucose associated with alterations in lipid protein metabolism, resulting from a relative or absolute lack of insulin.

A crucial aspect of care of dental patients who have diabetes is determination of the level of disease severity and the level of glycemic control, as well as the presence of complications from diabetes, so that appropriate dental treatment can be provided. Essential to this determination is knowledge of the patient's blood glucose level at the time that dental treatment is provided.

CLINICAL PRESENTATION

In patients with type 1 diabetes, the onset of symptoms is sudden and acute, often developing over days or weeks.Typically, the diagnosis is made in nonobese children or young adults younger than 40 years of age; however, it may occur at any age. Signs and symptoms include polydipsia, polyuria, polyphagia, weight loss, loss of strength, marked irritability, recurrence of bed wetting, drowsiness, malaise, and blurred vision. Patients also may present with ketoacidosis, which if severe is accompanied by vomiting, abdominal pain, nausea, tachypnea, paralysis, and loss of consciousness.Type 2 diabetes generally occurs after age 40 and more often affects obese individuals.The onset of symptoms in type 2 diabetes usually is insidious, and the cardinal manifestations and symptoms (polydipsia, polyuria, polyphagia, weight loss, and loss of strength) are less commonly seen. Other signs and symptoms related to the complications of diabetes include skin lesions, cataracts, blindness, hypertension, chest pain, and anemia. The rapid onset of myopia in an adult is highly suggestive of diabetes mellitus.

TABLE 14.3 Diagnostic Criteria for Diabetes Mellitus*

1. FPG ≥126 mg/dL (≥7.0 mmol/L) on two occasions. Fasting is defined as no caloric intake for at least 8 hours. This fasting glucose value is consistently associated with the risk for retinopathy.

or

2. Symptoms and signs of diabetes plus casual (random) plasma glucose concentration ≥200 mg/dL (11.1 mmol/L). *Casual* is defined as obtained at any time of day without regard to time since last meal. Many patients do not have obvious symptoms. The cardinal manifestations of diabetes include polyuria, polydipsia, and unexplained weight loss.

or

3. 2-Hour postload glucose ≥200 mg/dL (≥11.1 mmol/L) during an OGTT. The test should be performed as described by the WHO using a glucose load containing the equivalent of 75 g of anhydrous glucose dissolved in water.*

or

4. Glycosylated hemoglobin (by A_{1C} assay) ≥6.5%

TABLE 14.5 American Diabetes Association (ADA) and American College of Endocrinology (ACE): Targets for Glycemia Management

Parameter	Normal	ADA*	ACE
Premeal plasma glucose (mg/dL)	<100 (mean ≈90)	90–130	<110
Postprandial plasma glucose* (mg/dL)	<140	<180	<140
A_{1C}	4%–6%	<7%†	<6.5%

DENTAL MANAGEMENT

1- Any dental patient whose condition remains undiagnosed but who has the cardinal signs and symptoms of diabetes (i.e., polydipsia, polyuria, polyphagia, weight loss, and weakness) should be referred to a physician for diagnosis and treatment.

2- Patients with findings suggestive of diabetes (headache, dry mouth, marked irritability, repeated skin infection, blurred vision, paresthesias, progressive periodontal disease, multiple periodontal abscesses, loss of sensation) should be referred to a clinical laboratory or to a physician for screening tests to determine if diabetes mellitus type 1 or type 2 or another type of diabetes is responsible for their symptoms.

3- Patients with an estimated fasting blood glucose level of 126 mg/100 mL or higher should be referred to a physician for medical evaluation and treatment, if indicated. Those with a 2-hour postprandial blood glucose level of 200 mg/100 mL or higher also should be referred.

4- Patients who are obese, who are older than 45 years of age, or who have close relatives with diabetes should be screened routinely (at least at 3-year intervals) for any indication of hyperglycemia that may reveal the onset of diabetes.

5- Women who have given birth to large babies (birth weight >9 lb) or who have had multiple spontaneous abortions or stillbirths also should be screened once a year for diabetes.

6- All patients with diagnosed diabetes must be identified by history, and the type of medical treatment they are receiving must be established.

7- The type of diabetes (type 1, type 2, other) should be determined and the presence of complications noted.

8- Patients who are being treated with insulin should be asked how much insulin they use, how often they inject themselves each day, the frequency of insulin reactions and when the last one occurred, how often the patient self-monitors her or his blood glucose levels.
9- Patients with abnormal pulse rate and rhythm or elevated blood pressure should be approached with caution. As the risk for serious cardiovascular events increases substantially in those patients
10- **Analgesics**: Aspirin and nonsteroidal antiinflammatory drugs can potentially enhance the efficacy of some oral hypoglycemic agents (sulfonylureas) and enhance hypoglycemia; thus, they should be used judiciously.
11- **Prophylactic Antibiotics**: The decision to use antibiotic prophylaxis or coverage typically involves consultation with the patient's physician and is related to poor glycemic control. Patients who have brittle diabetes (in which control is very difficult to achieve) or who require a high dosage of insulin (in type 1 diabetes) and are undergoing an invasive procedure may be at increased risk for postoperative infection. However, prophylactic antibiotics usually are not indicated. If the patient develops an infection, appropriate systemic antibiotics may be given.
12- **The risk for infection in patients with diabetes**, in theory, the rsik is directly related to fasting blood glucose levels, presence of infecting organisms, and invasiveness of dental procedures. As indicated by data for general surgery procedures, if the fasting blood glucose level is below 206 mg/100 mL, increased risk is not predicted. However, if the fasting blood glucose level is between 207 and 229 mg/100 mL, the risk is predicted to be increased by 20% if surgical procedures are being performed. Additionally, if the fasting blood glucose level rises to above 230 mg/100 mL, an 80% increased risk of infection postoperatively has been reported.

13- **Management of infection:** Patients who are receiving insulin usually require additional insulin, which should be prescribed by their physicians. Non–insulin-controlled patients may need more aggressive medical management of their diabetes, which may include insulin during this period. The dentist should treat infection aggressively by incision and drainage, extraction, pulpotomy, warm rinses, and antibiotics. Antibiotic sensitivity testing is recommended for patients with brittle diabetes and for those who require a high insulin dosage for control. For these patients, penicillin therapy can be initiated. Then, if the clinical response is poor, a more effective antibiotic can be selected on the basis of results of antibiotic

sensitivity testing. Attention also should be paid to the patient's electrolyte balance and to fluid and dietary needs.

14- **Anesthetics:** For most patients with diabetes, routine use of local anesthetic with 1:100,000 epinephrine is well tolerated. However, epinephrine has a pharmacologic effect that is opposite that of insulin, so blood glucose could rise with the use of epinephrine. In diabetic patients with hypertension, history of recent \MI, or cardiac arrhythmia, caution may be indicated with use of epinephrine. Guidelines for these patients are similar to those for patients with cardiovascular conditions.

15- **Complications:** A patient with diabetes who is receiving good medical management and demonstrates good glycemic control without serious complications such as renal disease, hypertension, or coronary atherosclerotic heart disease can undergo any indicated dental treatment. If diabetes is under good control, even cardiac transplantation can be safely performed.In patients with diabetes who have serious medical complications

16- **Insulin shock:** A major goal in the dental management of patients with diabetes who are being treated with insulin is to *prevent insulin shock during the dental appointment*. Patients should be told to take their usual insulin dosage **and** to eat normal meals before the appointment, which usually is best scheduled in the morning. When such a patient arrives, the dentist should confirm that the patient has taken insulin and has eaten breakfast. In addition, patients should be instructed to tell the dentist whether at any time during the appointment they are experiencing symptoms of an insulin reaction. A source of sugar such as orange juice, cake icing, or nondiet soft drink must be available in the dental office to be given to the patient if symptoms of an insulin reaction develop

17- **Consultation**: Patients who have not seen their physician recently (within the previous 6 months), who have had frequent episodes of insulin shock, or who report signs and symptoms of diabetes may have disease that is unstable. These patients should be referred to their physicians for evaluation, or their physicians should be consulted to establish their current status.

18- **Diet:** Any patient with diabetes who is going to undergo extensive periodontal or oral surgery procedures other than single simple extractions should be given special dietary instructions for after surgery. It is important that the total caloric content and the protein–carbohydrate– fat ratio of the diet remain the same so that control of the disease and proper blood glucose balance are maintained. One suggestion is to have the patient use a blender

to prepare his or her usual diet so that it can be ingested with minimum discomfort; alternatively, special food supplements in a liquid form may be used.

19- **A protocol for intravenous sedation**: it often involves fasting before the appointment (i.e., nothing by mouth after midnight), using only half the usual insulin dose, and then supplementing with intravenous glucose during the procedure. Patients with well-controlled diabetes may be given general anesthesia if necessary. However, management with local anesthetics is preferable, especially in outpatient office settings.

20- **Devices:** If an insulin pump is being used by the patient, ensure that it is working properly. Antibiotic prophylaxis is not indicated.

21- **Drugs:** Some patients with type 1 diabetes who are being treated with large doses of insulin (in some cases, type 2 diabetes) experience periods of extreme hyperglycemia and hypoglycemia (brittle diabetes) even when given the best of medical management. For these patients, close consultation with the physician is required before any dental treatment is started. Certain drugs used in dentistry can alter blood glucose and interfere with the action of several drugs used to treat diabetes (insulin). See Table below.

TABLE 14.7 Noninsulin Antidiabetic Drugs

Class Drug	Mechanism of Action (Target Tissue)	Principal Adverse Effects	Drug Interaction(s)
SULFONYLUREAS			
Administer 30 minutes before meals.			
First Generation			
Chlorpropamide (Diabinese, Insulase) Acetohexamide (Dymelor) Tolazamide (Tolinase) Tolbutamide (Orinase)	Enhance insulin secretion (beta cells)	Hypoglycemia, weight gain, hyperinsulinemia	Salicylates and ketoconazole increase hypoglycemia.
Second-Generation			
Glipizide (Glucotrol, Glucotrol XL) Glyburide (Micronase, Glynase, DiaBeta) Glimepiride (Amaryl)	Enhance insulin secretion (beta cells)	Hypoglycemia, weight gain, hyperinsulinemia	Corticosteroids decrease action.
BIGUANIDES			
Administer with meals. Metformin (Foramet)	Reduce glucose production*	GI disturbances (abdominal pain, nausea, diarrhea), lactic acidosis	—
α-GLUCOSIDASE INHIBITORS			
Administer just before meals. Acarbose (Precose) Miglitol (Glyset)	Delay carbohydrate digestion (gut)	GI disturbances (abdominal pain, nausea, diarrhea), liver function test elevation	—
THIAZOLIDINEDIONES GLITAZONES			
Administer with meals. Pioglitazone (Actos) Rosiglitazone (Avandia)	Improves insulin sensitivity (fat, muscle)	Headache, weight gain, flatulence Causes or exacerbates heart failure, decreased hemoglobin or hematocrit	—
SODIUM GLUCOSE CO-TRANSPORTER 2 INHIBITORS (SLGT2 AGENTS)			
Dapagliflozin (Farxiga), Canagliflozin (Invokana), empagliflozin (Jardiance)	Selectively inhibiting this co-transporter, which is expressed almost exclusively in the proximal, convoluted tubule in the kidney	Headache, weight gain, flatulence	May increase hypoglycemia
GLINIDES			
Administer 15 minutes before meals. Repaglinide (Prandin) Nateglinide (Starlix)	Enhance insulin secretion (beta cells)	Hypoglycemia (less than sulfonylureas), weight gain, hyperinsulinemia, hypersensitivity, increased uric acid levels	Increased risk of hypoglycemia with salicylates, nonselective beta blockers, NSAIDs Metabolism may be inhibited by azoles, erythromycin
INCRETIN (GLP-1) ANALOGUES			
Administer 15 minutes before meals. Exenatide (Byetta) *Injected subcutaneously* Liraglutide (Victoza) *Injected subcutaneously*	Enhance insulin secretion (beta cells), delay gastric emptying (gut), suppress prandial glucagon secretion	GI adverse effects (nausea, vomiting, diarrhea)	—
AMYLIN ANALOGUE			
Administer before meals. Pramlintide (Symlin) injected subcutaneously	Aids absorption of glucose by slowing gastric emptying (gut), promotes satiety (hypothalamic receptors)	GI disturbances, headache	Avoid anticholinergics that alter GI motility. Can delay absorption of oral medications; administer oral hypoglycemic agents 1–2 hr after Symlin
DIPEPTIDYL PEPTIDASE-4 INHIBITORS			
Administer once daily regardless of meals. Linagliptin (Tradjenta)	Inhibits enzymatic breakdown of GLP-1 and GIP; increases insulin secretion; decreases glucagon secretion (pancreas)	Runny nose, headache	Hypoglycemia may occur when combined with insulin or sulfonylurea drugs.
Saxagliptin (Onglyza)		Peripheral edema	—
Sitagliptin (Januvia)		Headache	
COMBINATION DRUGS			
Some combination drugs include glyburide and metformin (Glucovance), glipizide and metformin (Metaglip), and pioglitazone hydrochloride and glimepiride (Duetact).			

Oral Complications and Manifestations

Oral complications of poorly controlled diabetes mellitus may include xerostomia; bacterial, viral, and fungal infections (including candidiasis); poor wound healing; increased incidence and severity of caries; gingivitis and periodontal disease; periapical abscesses; and burning mouth symptoms.

Oral findings in patients with uncontrolled diabetes most likely relate to excessive loss of fluids through urination, altered response to infection; microvascular changes; and, possibly, increased glucose concentrations in saliva.

The effects of hyperglycemia lead to increased amounts of urine, which deplete the extracellular fluids and reduce the secretion of saliva, resulting in dry mouth. A high percentage of patients with diabetes present with xerostomia and low levels of salivary calcium, phosphate, and fluoride.

Saliva glucose levels are elevated in persons with uncontrolled and controlled diabetes. Several studies have reported increased incidence and severity of gingival inflammation, periodontal abscess, and chronic periodontal disease in patients with diabetes.

Diabetes results in enhanced inflammatory responses, depressed wound healing, and small blood vessel changes that contribute to an increased risk for periodontitis. Thus, it is not surprising that adults with uncontrolled diabetes have more severe manifestations of periodontal disease than do adults without diabetes. Periodontal disease found in these young adults (older than 30 years of age) usually is asymptomatic and typically remains undetected. Overall, periodontal disease is more severe and more frequent in patients with poorly controlled diabetes.

Caries appears to be more significant in patients with diabetes who have poor glycemic control.

Oral fungal infections, including candidiasis and the more rare mucormycosis, may be noted in the patient with uncontrolled diabetes. The general consensus is that healing is delayed in persons with uncontrolled diabetes and that they are more prone to various oral infections after undergoing surgical procedures.

Oral lesions are more common in patients with diabetes. A significantly higher percentage of oral lesions, especially candidiasis, traumatic ulcers, lichen planus, and delayed healing, have been noted in patients with type 1 diabetes, compared with a control population. Altered immune system function contributes to the appearance of these lesions in diabetes.

Diabetic neuropathy may lead to oral symptoms of paresthesias and tingling, numbness, burning, or pain caused by pathologic changes involving nerves in the oral region. Diabetes has been associated with oral burning symptoms. Early diagnosis and treatment of diabetes may lead to regression of these symptoms, but in long-standing cases, the changes may be irreversible.

The drug used for treatment lik Metformin is associated with a metallic taste.

-The End-

Pulmonary diseases

Respiratory tract consists of: **Upper respiratory tract**; nose, paranasal sinuses, pharynx and larynx and **Lower respiratory tract**; trachea, bronchi, bronchioles and the lungs (respiratory bronchioles, alveolar ducts alveolar sacs and alveoli).

Chronic obstructive pulmonary diseases (COPD)

It is a general term for pulmonary disorders characterized by chronic airflow limitation from the lungs that is not fully reversible. The two most common diseases classified as COPD are chronic bronchitis and emphysema.

Chronic bronchitis

It is defined as excessive tracheobronchial mucous production causing chronic cough and sputum production for at least 3 months in at least 2 consecutive years.

Pathological changes:
- Thickened bronchial walls.
- Inflammatory cell infiltrate.
- Increased size of the mucous glands and goblet cell hyperplasia.
- Narrowing of the small airways, increased sputum production and collapse of the peripheral airways.
- Obstruction of the airflow is on inspiration and expiration.

Emphysema

It is the distention of the air spaces distal to the terminal bronchioles because of the destruction of the alveolar walls and septa.

Pathological changes:
Injury to the epithelium causes the release of the inflammatory mediators that attract neutrophils which release the enzyme (elastase) causing destruction of the alveolar walls. The obstruction is caused by the collapse of these unsupported and enlarged air spaces on expiration.

Etiology

1. Genetic susceptibility.
2. Smoking.
3. Pollution.
4. Absence of alpha1-antitrypsin.

Clinical presentation

Chronic bronchitis; chronic cough with copious sputum, patients are usually overweight, cyanotic, edematous and breathless (blue bloaters).
Patients have frequent respiratory infections, it may progress to Cor pumonale (right sided heart failure).
Emphysema; dyspnea on exertion, non-productive cough, patients are barrel chested, weight loss, expiration with pursing lips to forcibly exhale the air (pink puffers).

Diagnosis

- Measuring forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) by spirometry. FEV1/FVC ratio of less than 70% indicates COPD.
- Arterial blood gas measurement. In chronic bronchitis there is increased partial pressure of CO2 (PCO2) and reduced partial pressure of O2 (PO2), while in emphysema there is relatively normal PCO2 and reduced PO2.
- Chest radiographs.

Medical management

- Smoking cessation and elimination of exposure to pollutants.
- Exercise and good nutrition.
- Prevention of infection.
- Low flow supplemental oxygen when PO2 is 88% or less.
- Medical treatment which include among other drugs:
  - ✓ Bronchodilators: The inhaled agents are **short- and long-acting anticholinergics** (e.g., ipratropium, tiotropium) that reduce glandular mucus and relax smooth muscle by blocking acetylcholine at the muscarinic receptors, **short-and long-acting β2-adrenergic agonists** bronchodilators that relax smooth muscle and **inhaled corticosteroids**.
  - ✓ Phosphodiesterase inhibitors like theophylline.
  - ✓ phosphodiesterase-4–selective inhibitors (e.g., roflumilast, cilomilast).
  - ✓ Antibiotics for pulmonary infections.

Dental management

1. Obtaining thorough history and examination for the presence of COPD.
2. Encourage patients who smoke to quit.
3. Assessment of the severity of the disease and the degree of control.
4. Patients with signs and symptoms of COPD; shortness of breath, respiratory tract infection or reduced oxygen saturation (less than

91% by oximetry) should be referred for medical evaluation and treatment and dental treatment should be deferred.
5. Stable patients should be treated in upright or semisupine position and to avoid anything that could further depress respiration.
6. Local anesthesia is satisfactory but bilateral inferior dental nerve or palatal nerve block should be avoided.
7. Avoid rubber dam application.
8. Low flow oxygen (2-3 L/min.) should be considered when oxygen saturation is reduced below 95%.
9. Avoid Nitrous oxide (N2O) in severe COPD.
10. Avoid Barbiturates and narcotics (respiratory depression), antichoinergic and antihistamine drugs (drying of the mucous membrane and increased tenacity of mucous), because patients with chronic bronchitis may be already taking these types of drugs and concurrent administration may cause additive effects.
11. Patients who are treated with corticosteroids may need supplementation.
12. Patients may have hypertension and coronary heart disease and must be managed accordingly.
13. Avoid macrolide antibiotics (e.g. erythromycin) and Ciprofloxacin in patients taking theophylline to avoid toxicity (symptoms include: anorexia, nausea, vomiting, nervousness, headache, agitation and cardiac arrhythmia.
14. Avoid outpatient GA, it should be in hospital setting.

Oral complications and manifestations

- Chronic smokers may exhibit increased likelihood for halitosis, extrinsic tooth stains, nicotine stomatitis, periodontal diseases premalignant oral lesions and oral cancer.
- Theophylline has been associated with Steven-Johnson syndrome.

Asthma

Is a chronic inflammatory respiratory disease that is associated with increased airway hyperreactivity, resulting in episodes of dyspnea, cough and wheezing.
The exact cause is not completely understood but it is multifactorial.

Types:

- Extrinsic (allergic or atopic); the most common (35%), it is an exaggerated inflammatory response that is triggered by inhaled seasonal allergens such as pollens, dust, house mites, and animal danders. it is IgE mediated sensitization and is generally seen in children and young adults.
- Intrinsic (non-allergic), it occurs in about 30% of patients. This form of asthma generally is seen in middle-aged adults, and its

onset is associated with endogenous factors such as emotional stress, gastroesophageal acid reflux, or vagally mediated responses.
- Drug induced (salicylates, NSAIDs, cholinergic drugs, ACE inhibitors and β-adrenergic blocking drugs).
- Exercise induced.
- Infectious asthma, which may occur due to viral or fungal infections.

Some authors believe that asthma is of two types only; Extrinsic and Intrinsic, and that both types can be precipitated by drugs, exercise and infections.

The obstruction of the air flow occurs as a result of bronchospasm, inflammation of the bronchial mucosa, mucous hypersecretion and sputum plugging.

Clinical presentation

Include reversible episodes of breathlessness (dyspnea), wheezing, cough, chest tightness, tachypnea and expiratory wheezing. The onset is sudden.
Based on the frequency of attacks and the level of pulmonary function, asthma is classified as:
- Mild; brief attacks less than 2 days/week, forced expiratory volume in 1 second (FEV1) is more than 80%. It is either intermittent or persistent.
- Moderate; several days/week, limited exercise tolerance, affect sleep and FEV1 between 60%-80%.
- Severe; frequent daily exacerbations, nocturnal asthma, exercise intolerance and FEV1 less than 60%.

Medical management

- Education of the patients and involving them in prevention or elimination of precipitating factors.
- Antiasthmatic drugs are selected according to the types and severity of asthma and whether the drug is prophylactic or therapy.

Antiasthmatic drugs include:
- ✓ Anti-inflammatory; (inhaled) like corticosteroids and Leukotriene inhibitors for prophylaxis of chronic asthma. Corticosteroids rarely produce systemic effects especially when the maximum daily dose does not exceed 1.5 mg/day for Beclomethasone dipropionate or equivalent.
- ✓ β2-adrenergic agonists; short acting β2-adrenergic agonists are used to relieve acute asthma, they cause bronchodilatation in 5 minutes or less.
- ✓ Methylxanthines; like Theophylline.
- ✓ Anticholinergic drugs

- ✓ Systemic corticosteroids: reserved for asthma unresponsive to inhaled corticosteroids and bronchodilators and for use during the recovery phase of a severe acute attack.

Dental management

The goal is to prevent acute asthmatic attack of the dental patient.

1. Identify the patient by history, the dentist must determine type and severity of asthma, precipitating factors, level of control (frequency, time of day, severity of attacks, management, medications taken and the necessity of hospitalization).
2. Precipitating factors must be avoided.
3. For patients with severe unstable asthma, routine dental care should be postponed and medical consultation is sought.
4. Always ask the patients to bring their bronchodilator inhalers with them, in patients with history of moderate or severe asthma, a prophylactic inhalation before dental treatment may be considered.
5. Drugs:
   - Avoid Aspirin and other NSAIDs, use Paracetamol.
   - Avoid Barbiturates and narcotics (may precipitate an attack and cause respiratory depression).
   - In patients taking Theophylline, macrolide antibiotics (e.g. Erythromycin) and Ciprofloxacin should be avoided. Also Cimetidine is discontinued 24 hours before i.v. sedation.
   - Patients taking corticosteroids may need supplementation.
   - Patients with Leukotriene modifying drugs may have prolonged INR and bleeding tendency due to impaired liver metabolism.
6. Provide stress free environment, if pre or intra-operative sedation is required, N2O is the best or small dose of Diazepam. In children Hydroxyzine or Ketamine can be used.
7. Local anesthesia is used in dental treatment. Asthmatic patients may react to sulfites (sulphites) used as a preservative in vasoconstrictor containing local anesthesia.

Management of asthmatic attack

1. Recognize signs and symptoms of acute attack:
   - Inability to finish sentences with one breath.
   - Tachypnea, more than 25 breaths/min.
   - Tachycardia, more than 100 beats/min.
   - Diaphoresis.
   - Accessory muscle usage.
   - Ineffectiveness of bronchodilators to relieve dyspnea.
   - Paradoxical pulse.
2. Stop the procedure.
3. Administer fast acting bronchodilator (Ventolin) at the first sign of the attack.

4. Corticosteroids and long acting beta2 agonists provide delayed response.
5. Subcutaneous injection of adrenalin 0.3-0.5 ml 1:1000.
6. Positive-flow oxygenation.
7. Repeat bronchodilators if needed.
8. Monitor vital signs.
9. Seek immediate medical assistance.

Oral complications and manifestations

- Nasal symptoms, allergic rhinitis and mouth breathing.
- Altered nasorespiratory function leading to increased upper anterior facial height, higher palatal vault, greater overjet and crossbite.
- Beta agonists may cause decreased salivary flow leading to increased incidence of gingivitis and dental caries.
- Patients taking beta agonists and Theophylline may have increased risk of gastroesophageal acid reflux leading to enamel erosion.
- Patients with prolonged use of corticosteroid inhalers may have candidiasis although rare.
- Headache and facial pain are frequent in patients taking antileukotrienes or Theophylline.

Tuberculosis (TB)

It is caused by infection with Mycobacterium tuberculosis and it is primarily a disease of the pulmonary system, although any organ of the body can be involved. Other species of mycobacteria are encountered such as; M. avium complex.
Mycobacterium tuberculosis is an acid fast, non motile, intracellular rod which is obligate aerobic.
The organism is contracted by infected air borne droplets of mucus and saliva during coughing, sneezing and talking. Another mode of transmission is by ingestion of contaminated milk.
Oral tissues may be infected by their own sputum.

Clinical presentation

In primary TB, there are few manifestations in 90% of the infected people. Primary pulmonary TB is seen most often in infants and children; however, cavitation is rare in these age groups, and children generally do not actively produce or expectorate sputum while the usual form of disease found in adults is called secondary or re-infection TB, which occurs with delayed reactivation of persistent dormant viable bacilli and probably represents relapse of a previous infection. Progression of the disease is associated with underlying conditions that cause depression of the immune system.

Symptoms include; cough, lassitude, malaise, anorexia, weight loss, fever, night sweats, sputum is mucoid but may become purulent, hemoptysis and dyspnea.
10-20% of the cases have extrapulmonary signs and symptoms which may include; LAP, back pain, GIT disturbance, dysuria and hematuria.

Some of the common sequelae of TB include:
- Pleurisy and pleural effusion.
- Meningitis.
- Miliary or disseminated TB.
- Isolated organ involvement other than that of the lung may occur, with the pericardium, peritoneum, kidneys, adrenal glands, and bone (known as Pott's disease **[named after Percivall Pott, 1714-1788, a British surgeon]**when occurring in the spine) commonly affected, the tongue and other tissues of the oral cavity also are involved, albeit infrequently.

Laboratory tests and investigations

1. Tuberculin skin test (TST)(Mantoux); which is a standardized purified protein derivative (PPD) test, its limitation is that it is negative in the first 6-8 weeks of incubation, it is also limited in immunocompromised patients. 10-25% of infected individuals have negative skin test.
2. Interferon gamma release assays (IGRA); performed on fresh whole blood, are diagnostic tests that may be used in place of the TST (except in children younger than 5 years of age). IGRAs measure the person's immune reactivity to white blood cells infected with M. tuberculosis, which release interferon-γ when mixed with antigens from the mycobacteria. These assays are advantageous because they can detect recent infections, results are available within 24 hours, and previous bacillus Calmette-Guérin (BCG) vaccination does not cause a false-positive result. Like the TST, however, they cannot discriminate active from latent infection
3. Chest radiographs are helpful but not pathognomonic, they show cavitation and hilar LAP.
4. Microscopic examination of the sputum for the presence of acid fast bacilli.
5. Culture or direct molecular tests that identify the M. tuberculosis or other species from body fluids and tissues.

Medical management

1. Patient education and compliance.
2. Appropriate selection of the drugs.
3. Multiple antibiotics like isoniazid, Rifampin and Pyrazinamide for sufficient time (usually 6 months), but this can be extended for 12-24 months in cases of drug resistance.

4. During treatment sputum culture should be tested for drug resistant bacteria (most threatening feature of the disease).

- **Multidrug Resistant TB (MDR-TB):** is defined by the WHO as resistant to the two strongest antituberculosis drugs, isoniazid and rifampin, it is the most threatening feature of TB, it occurs in HIV-infected persons and in many countries where TB is endemic.

Dental management

Many patients with infectious diseases cannot be identified, therefore all patients should be treated as though they are potentially infected and standard precautions for infection control should be strictly followed.

1. Patients with clinically active sputum-positive TB

1. Consult with the physician before treatment.
2. Perform urgent care only.
3. Treatment, especially when handpiece is required, should be done in hospital setting (proper sterilization, isolation, gloves, mask, gown and special ventilation) and not as out patient.
4. Children under 6 years receiving TB therapy can be treated as normal non-infectious patients and in outpatient setting, because cavitary disease is rare and due to the inability of children to cough up sputum effectively.
5. Patients older than 6 years are treated in hospital setting and only urgent care performed.

2. Patients with past history of TB

Fortunately relapse is rare but this is not the case in patients with inadequate treatment and those immunocompromised.

1. Obtain careful good history about treatment duration.
2. A good review of systems is essential.
3. Obtain history of periodic physical examination and chest radiograph to rule out reactivation or relapse.
4. Consult the physician if:
   a. History of inadequate duration of treatment (less than 6 months).
   b. Lack of appropriate follow up after recovery.
   c. Signs and symptoms of the disease.
5. Treat as normal patient with standard precautions of infection control, if the patient is free of clinically active disease.

3. Patients with positive tuberculin test

Those patients in the absence of signs and symptoms of active disease are considered to have latent TB and are not infectious.

1. Evaluation by a physician to rule out active disease.
2. Verify receiving Isoniazid for 9 months for prophylaxis.
3. Treat as normal patients with standard precautions of infection control.

4. <u>Patients with signs and symptoms suggestive of TB</u>

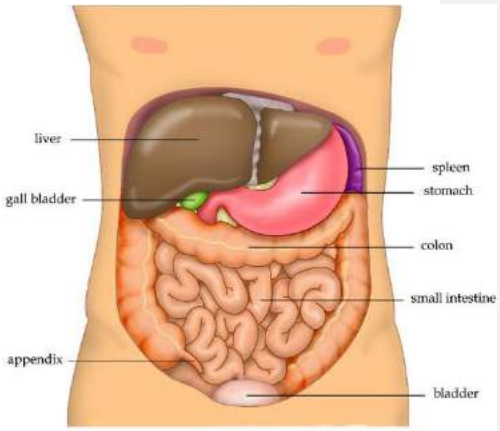
1. Any dental treatment should be postponed, the patient should be referred for medical treatment.
2. If urgent care is necessary, treatment is the same as in the first group.

Oral complications and manifestations

- Painful, deep irregular ulcer on the dorsum of the tongue, the palate, lips, buccal mucosa and gingiva may be affected.
- Granular, nodular or leukoplakic mucosal lesions.
- Cervical and submandibular lymph node infections (enlarged and abscessed).
- Osteomyelitis when the infection extends to the bone.
- Rifampin can cause leukopenia, hemolytic anemia and thrombocytopenia resulting in increased incidence of infection, delayed healing and gingival bleeding.
- Isoniazid, Rifampin and Pyrazinamide can cause hepatotoxicity so Acetaminophen containing drugs should be avoided.

Oral Surgery **د.ياسر رياض الخناق**

LIVER DISEASE



The liver is an essential organ located in the right upper quadrant of the abdomen, below the diaphragm, it has a number of key functions, including metabolism of the byproducts of food, detoxification of drugs, conversion of nitrogenous substances to be excreted by the kidneys, formation of blood clotting factors, metabolism of bilirubin, processing of lipids from the intestines, and storage of glycogen. Obviously then, clinical consequences of liver dysfunction manifest in loss of these functions.

Liver dysfunction may be attributed to a number of causes, including acquired infections and other pathologic conditions, as well as drug use.
Impairment of liver function can lead to abnormalities in many biochemical functions performed by the liver, such as synthesis of coagulation factors and drug metabolism, and dental patients with acute or chronic liver disease may be adversely affected.
Ultimately, serious end-stage liver dysfunction or cirrhosis may result.
Cirrhosis is the consequence of long-term damage to the liver tissues. This condition is irreversible and leads to fibrosis, resulting in jaundice, ascites, and portal hypertension, as well as significant liver dysfunction.
Obviously, liver disorders in persons presenting for treatment are of significant clinical interest to dentists in the context of the proper management of such patients. In this lecture, the two most common liver disorders and main causes of cirrhosis, hepatitis and alcoholic liver disease, are presented.

HEPATITIS

General Description

Hepatitis is inflammation of the liver that may result from infectious or other causes. Examples of hepatitis with infectious causes are viral hepatitis and that associated with infectious mononucleosis, secondary syphilis, and tuberculosis.

Also, noninfectious hepatitis can result from excessive or prolonged use of toxic substances, including drugs (i.e., acetaminophen, halothane, ketoconazole, methyldopa, and methotrexate) or, more commonly, alcohol.

Because the several types of hepatitis have various degrees of impact on dental treatment.

Viral hepatitis

Viral hepatitis is a collective term describing liver inflammation or hepatitis caused by a group of several different viruses.

Is the most common form of infectious hepatitis, there are 5 distinctive types of hepatitis viruses; A, B, C, D and E, they all target the liver.

The clinical manifestations of the five forms of viral hepatitis are quite similar, and the diseases can be distinguished from each other only by serologic assays.

Hepatitis A virus (HAV) and hepatitis E virus (HEV) are forms of *infectious hepatitis*; they are spread largely by the fecal–oral route, are associated with poor sanitary conditions, are highly contagious, occur in outbreaks as well as sporadically, and cause self-limited hepatitis only.

Hepatitis B virus (H BV), hepatitis C virus (HCV), and hepatitis D virus (HDV) are forms of *serum hepatitis*, are spread largely by parenteral routes and less commonly by intimate or sexual exposure, and are not highly contagious. They are capable of leading to chronic infection and, ultimately, to cirrhosis and hepatocellular carcinoma.

Cases of an acute viral hepatitis–like syndrome that cannot be identified as being caused by a known hepatitis virus; this syndrome has been called acute non-A, non-B, non-C, non-D, non-E (non–A-E) hepatitis or acute hepatitis of unknown cause. Despite many attempts, the viral etiology of non–A-E hepatitis remains unproved.

Pathophysiology and Complications

The pathogenesis of the liver injury in viral hepatitis is not well understood. None of the five primary agents seems to be directly cytopathic. The hepatocyte injury in viral hepatitis suggest that **immune responses**, particularly cytotoxic T-cell responses to viral antigens expressed may be the major effectors of injury. Other proinflammatory cytokines, natural killer cell activity, and antibody dependent cellular cytotoxicity also may play roles in cell injury and inflammation during acute hepatitis virus infection.

Recovery from hepatitis virus infection usually is accompanied by the appearance of rising titers of antibody against viral envelope antigens, such as anti-HAV, anti-HBs, anti-HCV-E1 and anti-HCV-E2, and anti-HEV; these antibodies may provide at least partial immunity to reinfection.

Clinical Presentation

Acute hepatitis

is highly variable and ranges in severity from a transient, asymptomatic infection to severe or fulminant disease. The disease may be self-limited with complete resolution, run a relapsing course, or lead to chronic infection. The **incubation period** ranges from 2 to 20 weeks. During this phase, virus becomes detectable in blood, but serum aminotransferase and bilirubin levels are normal, and antibody is not detected.

Patients classically exhibit three phases of acute viral hepatitis:

1. **Prodromal (preicteric) phase** is usually precedes the onset of jaundice by 1 or 2 weeks and consists of nonspecific anorexia, intermittent nausea, vomiting, fatigue, myalgia, malaise, fever, and vague right upper quadrant pain. Virus specific antibody first appears during this phase. Viral titers generally are highest at this point, and serum aminotransferase levels start to increase.
2. **Icteric phase** is heralded by the onset of clinical jaundice, manifested by a yellow-brown cast to the conjunctivae, skin, oral mucosa, and urine (dark urine). Many of the nonspecific prodromal symptoms may subside, but gastrointestinal (GI) manifestations (e.g., anorexia, nausea, vomiting, right upper quadrant pain) may increase, especially early in this phase. In more severe cases Hepatomegaly and splenomegaly (HSM)frequently are noted. This phase lasts 2 to 8 weeks and is part of the clinical course in at least 70% of patients infected with HAV, 30% of those acutely infected with HBV, and 25% to 30% of patients acutely infected with HCV.
   Serum bilirubin levels (total and direct) rise, and aminotransferase levels generally are higher than10 times the upper limit of normal, at least at the onset.
3. **Convalescence or recovery (posticteric) phase** symptoms disappear, but hepatomegaly and abnormal liver function values may persist for a variable period. This phase can last for weeks or months, with longer recovery times for HBV and HCV with usual recovery (clinical and biochemical) within approximately 4 months after the onset of jaundice. Neutralizing antibodies usually appear during the icteric phase and rise to high levels during convalescence.

Complications of acute viral hepatitis include chronic infection, fulminant hepatic failure, and Cirrhosis.

Commented [WU1]: **Incubation period** is the time elapsed between exposure to a pathogenic organism, a chemical, or radiation, and when symptoms and signs are first apparent

Commented [WU2]: Bilirubin is a degradation product of hemoglobin and one of the major constituents of bile, to which it confers the characteristic yellowish color. Bilirubin normally is transported to the liver by way of the plasma. In the liver, it conjugates with glucuronic acid, and then it is excreted into the intestine, where it aids in the emulsification of fats and stimulates peristalsis.1,3a In the presence of liver disease, bilirubin tends to accumulate in the plasma as a consequence of decreased liver metabolism and transport.

Chronic hepatitis, generally defined as illness of at least 6 months' duration, develops in approximately 2% to 7% of adults with hepatitis B and in 50% to 85% of adults with hepatitis C.

Acute liver failure or fulminant hepatitis

serious complication of acute viral hepatitis occurs in 1% to 2% of patients with symptomatic acute hepatitis, perhaps most commonly with hepatitis B and hepatitis D and least commonly with hepatitis C.

It characterized by massive hepatocellular destruction and a mortality rate of approximately 80%. The condition occurs more commonly among older adults and patients with chronic liver disease. Coinfection or superinfection with HBV and HDV or infection by a single hepatitis virus can cause fulminant disease.

The disease is called fulminant if evidence of hepatic encephalopathy appears; however, the initial symptoms (changes in personality, aggressive behavior, or abnormal sleep patterns) may be subtle or misunderstood.

Commented [WU3]: Encephalopathy is a general term that means brain disease, damage, or malfunction.

Cirrhosis

Liver cell necrosis and inflammation followed by fibrosis, regeneration and vascular derangement. Liver function deteriorates and blood flow through the organ is obstructed.

It is a common sequel to hepatitis C and alcohol related damage.

Clinical features include; jaundice, ascites, anemia, gastrointestinal hemorrhage, HSM, spider nevi, opaque nails, gynecomastia, liver failure, bleeding tendencies, portal hypertension and hepatic encephalopathy.

Laboratory findings

- **Serum Transaminases** including; Alanine aminotransferase (ALT) (formerly serum glutamatepyruvate transaminase [SGPT]) {Normal range 7-56 IU/L} and Aspartate aminotransferase (AST) (formerly serum glutamate-oxaloacetate transaminase [SGOT]) {Normal range 0-35 IU/L}, these enzymes are released from damaged hepatocytes. ALT is more sensitive indicator of liver damage because it is found only in liver cells. AST is also found in heart, skeletal muscle, pancreas, kidney and RBCs.
- **Plasma bilirubin** level; normal value is less than 1 mg/100ml. jaundice is evident when bilirubin level approaches 2.5mg/100ml.
- **Serum alkaline phosphatase**; it is non-specific test may be normal or slightly elevated. Normal value is 9 to 85 IU/L.
- **WBC count** is increased.
- **Prothrombin time** may be elevated.

- **Diagnosis** is through **serological tests**:

Hepatitis A→ IgM-specific anti-HAV arises early in the disease and persists for only 4 to 12 months

→IgG anti-HAV develops in all patients infected with the virus, is first detectable shortly before the onset of symptoms; titers then rise to high levels, which persist for life.

Hepatitis B→ HBsAg (infectious). Anti-HBsAg (recovery)
Anti-HBcAg (acute infection and Previous infection).
HBeAg (infectious). Anti-HBeAg (clearing\cleared infection).

Hepatitis C→ HCV RNA (infectivity). Anti-HCV (previous infection).

Hepatitis D→ Anti-HDV and HDAg.
Hepatitis E→ Anti-HEV

PREVENTION

Prevention Through Active Immunization

The risk of viral hepatitis is reduced by receiving active immunization. At present, vaccines are available for HAV, for HBV and for combination hepatitis A and B. The HAV vaccines are safe, highly immunogenic, and recommended for patients 2 years of age and older.

The vaccines for prevention of HBV infection are administered in three doses over a 6-month period and produce an effective antibody response in more than 90% of adults and 95% of infants, children, and adolescents.

Vaccination are advocated for persons at high risk for contracting HBV infection table No.1. At the top of the list are health care workers, including dentists, for whom inoculation with the vaccine is strongly recommended.

A current strategy to interrupt HBV transmission in all age groups includes
(1) prevention of perinatal HBV infection,
(2) routine vaccination of all infants, and
(3) vaccination of selected adolescents and adults not vaccinated as infants.

Table No.1

Persons at Substantial Risk for Hepatitis B Who Should Receive Vaccine
Individuals with occupational risk
Health care workers
Public safety workers
Clients and staff of institutions for developmentally disabled individuals
Hemodialysis patients
Recipients of certain blood products
Household contacts and sex partners of HBV carriers
Adoptees from countries where HBV infection is endemic
International travelers
Illicit drug users
Sexually active homosexual and bisexual men (men who have sex with men)
Sexually active heterosexual men and women (who have multiple partners)
Inmates of long-term correctional facilities

Commented [WU4]: The duration of immunity and the need for booster doses remain controversial. Current information based on experience with the plasma-derived HBV indicates that immunity remains effective for more than 10 years. Current guidelines published by the Centers for Disease Control and Prevention (CDC) Advisory Committee on Immunization Practices recommend booster doses only for persons who did not respond to the primary vaccine series.

Prevention Through Passive Immunization

Treatment of viral hepatitis can be accomplished by administering early postexposure immune globulins or postexposure hepatitis B vaccine. Immune serum globulin is derived from a pool of antibodies collected from human plasma that is free of HBsAg, HCV, and HIV. This sterile solution contains antibodies against both hepatitis A and hepatitis B. Another type of immune globulin is called hepatitis B immune globulin (HBIG).

It is specially prepared from preselected plasma that is high in titers of anti-HBs. Administration of both immune globulin and HBIG is safe, but they interact adversely with live attenuated vaccines (i.e., measles, mumps, rubella [MMR] vaccine) if given within 5 months of each other.

TREATMENT

- As with many viral diseases, therapy basically is **palliative and supportive**. Bedrest and fluids may be prescribed, especially during the acute phase. A nutritious and high calorie diet is advised.
- Alcohol and drugs **metabolized by the liver** are not to be ingested.
- Viral **antigen and ALT** levels should be monitored for 6 months to determine whether the hepatitis is resolving.
- Chronic hepatitis is treated by administration of **interferon (alfa-2b)** (3–10 million units given three times weekly for 6 months to 1 year).
- **Corticosteroids** usually are reserved for patients with fulminant hepatitis.
- **Liver transplantation** is a last resort for patients who develop cirrhosis.

Dental management

1. **Identification of potential or actual carriers** of HBV, HCV and HDV is very difficult through history.
2. **All patients with viral hepatitis** must be managed as though they are **potentially infectious**, so standard precautions for infection control should be implemented.
3. It is recommended that all dental health care workers should receive **vaccination against HBV** and implement **standard precautions** during the care of all dental patients.
4. Patients with **active hepatitis**:
   - Should be referred for medical treatment, only urgent dental treatment should be provided with strict adherence to standard precautions of infection control and preferably in isolated operatory.
   - Aerosols should be minimized.
   - Drugs that are metabolized in liver should be avoided.
   - If surgery is necessary prothrombin time (PT) and bleeding time should be obtained.

5. Patients with **history of hepatitis**:
   - Since most patients are unaware that they have had hepatitis, identification of carriers is very difficult, and requesting screening tests for every patient is not practical.The only method for providing protection is to adopt a strict program of clinical asepsis for all patients.
   - If knowing the type of hepatitis virus through history is not possible, screening test can be ordered for the presence of HBsAg or Anti-HCV.
6. Patients at **high risk of HBV or HCV infection**:
   - Patients are at high risk of hepatitis B or hepatitis C are listed in table No.1.
   - **Screening** is recommended for HBsAg and Anti-HCV.
   - Patients who are carriers might have chronic active hepatitis leading to bleeding problems or metabolism problems that require treatment modification.
   - If **accidental needle stick occurs**, knowing if the patient is HBsAg positive or HCV positive is very important to determine the need for immunoglobulin, vaccine and follow up medical care.
7. Patients who are **hepatitis carriers:**
   - Standard precautions of infection control should be followed.
   - Such patients may have chronic active hepatitis with compromise liver function. Liver function tests can be ordered and consultation with physician may be needed.
8. Patients with **signs and symptoms of hepatitis**
   - Only emergency dental care should be provided in isolated operatory with adherence to the standard precautions of infection control.
   - Routine dental care should be postponed and the patient referred to the physician.
9. **Dentists who are hepatitis** virus carriers:
   - If the dentist is found to be positive for blood transmissible virus, exposure-prone procedures should not be performed, or strict adherence to aseptic technique should be followed to prevent transmission.
   - Periodic retesting is necessary.
10. **Postexposure protocols for percutaneous or permucosal exposures to blood.:**
   - In case of percutaneous or permucosal exposure through needle stick or puncture wound contaminated with blood from an individual who is HBsAg, the risk of infection may approach 30%.
   - ❖ If the exposed is **vaccinated;** a test to evaluate Anti-HBsAg should be done:
   - If inadequate levels→ HB immunoglobulin + vaccine booster should be administered.
   - If adequate levels→ nothing further is required.
   - ❖ If the exposed is **not vaccinated**→ HB immunoglobulin + initiation of vaccination is recommended.
   - For HCV; no postexposure protocol or vaccine is available.

11.Drug administration:

- Patients who are completely recovered from viral hepatitis, no special considerations are needed.
- In patients with *chronic active hepatitis with impaired liver function*; drugs that are metabolized in the liver should be avoided if possible or reduced doses used (Aspirin, Acetaminophen, Lidocaine, Ibuprofen, Ampicillin, Tetracycline, Metronidazol) table No.2. A quantity of 3 cartridges of 2% Lidocaine (120mg) is considered limited.

Table No.1

Dental Drugs Metabolized Primarily by the Liver
Local Anesthetics*
Lidocaine (Xylocaine)
Mepivacaine (Carbocaine)
Prilocaine (Citanest)
Bupivacaine (Marcaine)
Analgesics
Aspirin‡
Acetaminophen (Tylenol, Datril)‡
Codeine‡
Meperidine (Demerol)‡
Ibuprofen (Motrin)†
Sedatives
Diazepam (Valium)‡
Barbiturates‡
Antibiotics
Ampicillin
Tetracycline
Metronidazole§
Vancomycin§

Oral manifestations and complications

- **Abnormal bleeding**; results from abnormal synthesis of blood clotting factors, abnormal polymerization of fibrin, inadequate fibrin stabilization, excessive fibrinolysis, or thrombocytopenia associatedwith splenomegaly that accompanies chronic liver disease. Before surgery platelet count, PT and INR should be evaluated.
- Hepatocellular carcinoma rarely metastasize to the jaws.

Alcoholic liver disease

Alcoholism is a chronic addiction to ethanol in which a person craves and uncontrollably consumes ethanol and becomes tolerant to its intoxicating effect. It is the most common drug of abuse.

Alcohol is CNS depressant and it impairs the capacity to reason, it eventually interferes with cerebellar function causing ataxia, motor incoordination and unconsciousness.

Excessive alcohol consumption causes alcoholic liver disease and ultimately cirrhosis of the liver and worsens other liver disorders such as viral hepatitis.

The lack of treatment of alcohol abuse leads to significant morbidity and mortality rates.

An alcoholic is a man who drinks regularly 30 units/week or a woman who drinks 20 units/week.

Alcohol is hepatotoxic and its metabolite, acetyl aldehyde, is fibrinogenic.

Clinical presentation Pathophysiology and Complications

Alcohol has a deleterious effect on neural development, the corticotropin-releasing hormone system, metabolism of neurotransmitters, and the function of neurotransmitter receptors causing sensory and motor disturbances.

Prolonged abuse of alcohol contributes to malnutrition (folic acid deficiency), anemias, and decreased immune function.

The pathologic effects of alcohol on the liver are expressed as one of three disease entities. These conditions may exist alone but commonly appear in combination.

1. **Fatty liver** is the earliest change seen in alcoholic liver disease, characterized by presence of a fatty infiltrate with no visible manifestations except liver enlargement; it is considered completely reversible,
2. **Alcoholic hepatitis** is diffuse inflammatory condition of the liver is characterized by destructive cellular changes, some of which may be irreversible. For the most part, alcoholic hepatitis is considered a reversible condition. The clinical presentation of alcoholic hepatitis often is **nonspecific** and may include features such as nausea, vomiting, anorexia, malaise, weight loss, and fever. More **specific** findings include hepatomegaly, splenomegaly, jaundice, ascites, ankle edema, and spider angiomas.
3. **Cirrhosis** most serious form of alcoholic liver disease, which generally is considered an irreversible condition characterized by progressive fibrosis and abnormal regeneration of liver architecture in response to chronic injury or insult (i.e., prolonged and heavy use of ethanol).

It may remain asymptomatic for many years until sufficient destruction of the liver parenchyma has occurred to produce clinical evidence of hepatic failure.Ascites, spider angiomas, ankle edema, and jaundice may be the earliest manifestations, but frequently hemorrhage from esophageal varices is the initial sign with rapid progression to hepatic encephalopathy, coma, and death.

Laboratory findings

Laboratory findings in alcoholic liver disease range in significance from minimal abnormalities caused by a fatty liver to manifestations of alcoholic hepatitis and cirrhosis.

- Increased bilirubin, AST, ALT, gamma-glutamyl transpeptidase, amylase, uric acid, triglycerides and cholesterol.
- Leukopenia (or Leukocytosis) and anemia.
- Thrombocytopenia (due to splenomegaly).
- Increased prothrombin time (PT), partial thromboplastin time (PTT) and thrombin time.

Medical management

1. **Identification through physical examination** to evaluate impaired organ systems, includes a search for evidence of liver failure, GI bleeding, cardiac arrhythmia, and glucose or electrolyte imbalance.
2. **Gradual withdrawal from alcohol**, abrupt withdrawal leads to loss of appetite, tachycardia, anxiety, insomnia, hallucinations, disorientation, impaired memory and attention and agitation.
3. High protein, high calorie and low sodium diet and vitamin supplementation.
4. Management of CNS depression caused by withdrawal.
5. Education of the patient about alcoholism.
6. Management of complications.
7. End stage cirrhosis requires liver transplantation.

Dental management

1. **Detection of such patients** by:
   - History; the patient should be asked about the type, quantity, frequency, pattern and consequences of alcohol use, also family history of alcoholism.
   - Clinical examination for signs and symptoms of alcoholic liver disease.
   - Alcohol odor on breath.
   - Information from family members or friends.
2. A patient with **untreated alcoholic liver disease** is not a candidate for elective, outpatient dental care and should be referred to a physician.
3. In patients with **history of alcohol liver disease or alcohol abuse**, a physician should be consulted to verify the patient's current status, medications, laboratory values (if present) and contraindications for medications and surgery.
4. If signs and symptoms of alcoholic liver disease are present the dentist can request some screening tests before surgical procedures; complete blood count (CBC) with differential, AST, ALT, platelet count, thrombin time, PT and INR. **Abnormal results should be discussed with the physician**.

5. **Treatment considerations**; 3 major dental treatment considerations apply for patients with alcoholic liver disease:
   - **Bleeding tendencies**; can be managed with the assistance of physician, this may entail the use of local hemostatic agents, fresh frozen plasma, vitamin K, platelets and antifibrinolytic agents.
   - The **unpredictable metabolism of drugs**;

The dose of drug in table No.1 may need to be adjusted when treating patients with chronic alcoholism (e.g., half the regular adult dose may be appropriate if cirrhosis or alcoholic hepatitis is present), or a specific agent or class of drugs may be contraindicated as advised by the patient's physician.

In **mild to moderate liver disease**, enzymatic induction is likely to have occurred leading to increased tolerance to LA, sedatives, hypnotics and GA, thus larger doses may be needed to attain the desirable effects of these drugs.

In **cirrhosis or alcoholic hepatitis** avoid drugs that are metabolized in the liver or using half dose particularly if aminotransferases level (AST and ALT) is 4 times more than normal, serum bilirubin is more than 2mg/dl, serum albumin lower than 35g/L, with signs of ascites and encephalopathy or malnutrition.

   - The **risk of infection or spread of infection**; because these patients have reduced reticuloendothelial capacity and altered cell mediated immune function, but antibiotic prophylaxis is not needed unless there is an ongoing infection. Consultation with the physician regarding the use of antibiotic may be considered especially in patients with moderate or severe liver disease.

6. Liver enzyme induction and CNS effects of alcohol in patients with alcoholism can require use of **increased amounts of local anesthetic or additional anxiolytic procedures.**

Oral complications and manifestations

1. Poor oral hygiene and caries which is due to neglect.
2. Impaired gustatory function.
3. Nutritional deficiency can result in anemia causing glossitis, loss of papillae, angular and labial cheilitis.
4. Bleeding tendencies cause spontaneous gingival bleeding, mucosal ecchymoses and petechiae.
5. Alcohol breath odor.
6. Jaundiced mucosal tissues.

7. Sialoadenosis; bilateral painless swelling of the parotid glands, it is attributed to demyelinating polyneuropathy that results in abnormal sympathetic signaling, abnormal acinar protein secretion and acinar cytoplasmic swelling.
8. Oral squamous cell carcinoma, alcohol abuse and smoking are major predisposing factors.
9. Bruxism and dental attrition.
10. Xerostomia

أسأل الله تعالى
ان يرحمنا وإياكم
وأن يرفع هذه الغمه
عن العالم أجمع

Renal Diseases

- Functions of kidney: Regulate fluid volume , excrete nitrogenous waste, synthesize erythropoietin & vit D, and drug metabolism.

- Renal failure: the ability of the kidney to perform its functions has been deteriorated this indicates inability of the kidneys to maintain normal hemostasis.
- Acute renal failure(ARF): sudden decline in renal function usually reversible if treated early. Can be caused by renal hypo perfusion in hemorrhage, trauma, renal disease, drug damage, and prostatic hypertrophy or tumor that lead to obstruction of renal outflow.
- Chronic renal failure(CRF): irreversible, slowly progressive renal damage persisting for more than 3 months. Can be caused by DM, Hypertension, and chronic glomerulonephritis.

- Chronic renal disease can be compensated by structural and functional hypertrophy of surviving nephrons, when chronic renal insufficiency (CRI) ensues, CRI inevitably progresses to ESRD.
- End-stage renal disease(ESRD): Chronic deterioration of nephrons resulting in uremia and can lead to death.

- Clinical features of CRF: hypertension, heart failure, anorexia, drowsiness lead to coma, anemia, splenomegaly, leukopenia, thirst, polyuria, glycosuria, and hyperparathyroidism.

- Laboratory findings in CRF:
- There will be increase in serum creatinine level (normal values is 0.6-1.2 mg/dl), Blood urea nitrogen is above 20mg/dl, and there will be increase in the level of serum potassium and phosphate and decrease in serum calcium.

❑ Medical management of CRF&ESRD

1. Conservative care:{dietary modification, use of vit D that decrease parathyroid hormones levels to inhibit bone resorption, avoidance of nephrotoxic drugs(NSAIDs) or agents metabolized by the kidney (penicillin, cephalosporin and erythromycins), and the use of erythropoietin for treatment of anemia}.

2. Dialysis: artificially filters the blood become essential if renal function deteriorates to ESRD. It can be accomplished by

➢ Peritoneal dialysis: not required anticoagulation and reduced likelihood of infectious disease transmission

➢ Hemodialysis: required anticoagulation to prevent clotting, patients are at risk for contracting hepatitis B,C, and HIV.

3. Renal transplant: strongly recommended for all patients with ESRD, patient require immunosuppression to prevent rejection.

- Oral manifestations of CRF & ESRD

1. Pallor of oral mucosa related to anemia
2. Pigmentation of oral mucosa (red-orange discoloration) caused by deposition of carotene-like pigments
3. Xerostomia and parotid infections
4. Candidiasis
5. Dysgeusia (altered or metallic taste results from a high urea content)
6. Petechia and ecchymosis of oral mucosa
7. Gingival bleeding and gingival enlargement as a result of cyclosporine.
8. Oral ulcers , lichen planus lesions
9. Enamel hypoplasia
10. Central giant cell granuloma(brown tumor) radiolucent jaw lesions , lytic bone lesions result from hyperparathyroidism
11. Uremic stomatitis.

Dental management of patient with CRF or ESRD

- ✓ **<u>Under conservative care</u>**
- ➢ Consult with physician
- ➢ Avoid dental treatment if disease is unstable, poorly controlled or advanced.
- ➢ Screen for bleeding disorder before surgery (bleeding time, platelet count, HG,HCT), hemorrhage attributed to abnormal platelet aggregation and adhesiveness and defective platelet production dictate that the dentist must have local hemostatic agents available(topical thrombin & suture)

- Monitor blood pressure closely
- Pay attention to good surgical technique to decrease the risks of excessive bleeding and infection.
- Avoid nephrotoxic drugs(acyclovir, aspirin, NSAIDs, and tetracycline)
- Adjust dosage of drugs metabolized primarily by the kidney such as penicillin & cephalosporin.
- Aggressively manage orofacial infection with culture and sensitivity tests and antibiotics

- The dentist should consult physician to assess the need for antibiotics when invasive procedure are planned. Host defense compromised by nutritional deficiencies and changes in the production and function of WBC so individuals with CRF & ESRD are more susceptible to infection.
- Consider corticosteroid supplementation , if patients with ESRD have been taking large dose of corticosteroids its possible that they may have adrenal hypofunction, to avoid adrenal crisis the dental clinician may need to supplement the patient's corticosteroid dose before complex dental procedures are performed.

✓ **Peritoneal dialysis** presents no additional problems in dental management

✓ Patient receiving **hemodialysis**

- Same as conservative care recommendations
- Consult with physician about the risk of infective endarteritis or endocarditis occur even when preexisting cardiac defects are absent.
- Avoid blood pressure cuff and IV medications in arm with shunt.

- Avoid dental care on day of treatment (especially within first 6 hours afterward) best to treat on day after.
- Determination of the status of hemostasis is important before oral surgery is performed as hemodialysis tend to aggravate bleeding tendencies through physical destruction of platelet and the use of heparin. Screening tests include aPTT and platelet count.
- Consider corticosteroid supplementation as indicated
- Assess the status of liver function because of increased risk for carrier state of hepatitis B & C and HIV. Accordingly all patients should be treated with the use of standard infection control procedures.

✓ **<u>Patient with renal transplant</u>**

❖ Dental aspects

- Immunosuppression : increase risk of infection
- Excessive bleeding caused by adverse effects of immunosuppressant drugs that cause bone marrow suppression with resultant leukopenia, thrombocytopenia and anemia.

- Gingival hyperplasia caused by cyclosporine
- Increased incidence of cancer like lip squamous cell carcinoma and lymphoma as a complication of intense immunosuppression .
- Adrenal gland suppression, poor wound healing, osteoporosis, DM, hypertension, and increased risk of infection (all of them are adverse effects of corticosteroids)

❖ Dental management

- Stable graft period

➢ Consultation with physician

➢ Maintain effective oral hygiene procedures

➢ Initiate active recall program every 3-6 months

➢ Treat all new dental diseases

➢ Use universal precautions in controlling infection

➢ Have staff vaccinated against HBV

➢ Avoid infection:

❑ Medical consultation regarding the need for antibiotic prophylaxis

❑ Screening tests (WBC count)

➢ Avoid excessive bleeding:

❑ Screening tests (bleeding time, INR, PT, aPTT, and platelet count)

❑ Special precautions (local measures like suturing and topical hemostatic agents)

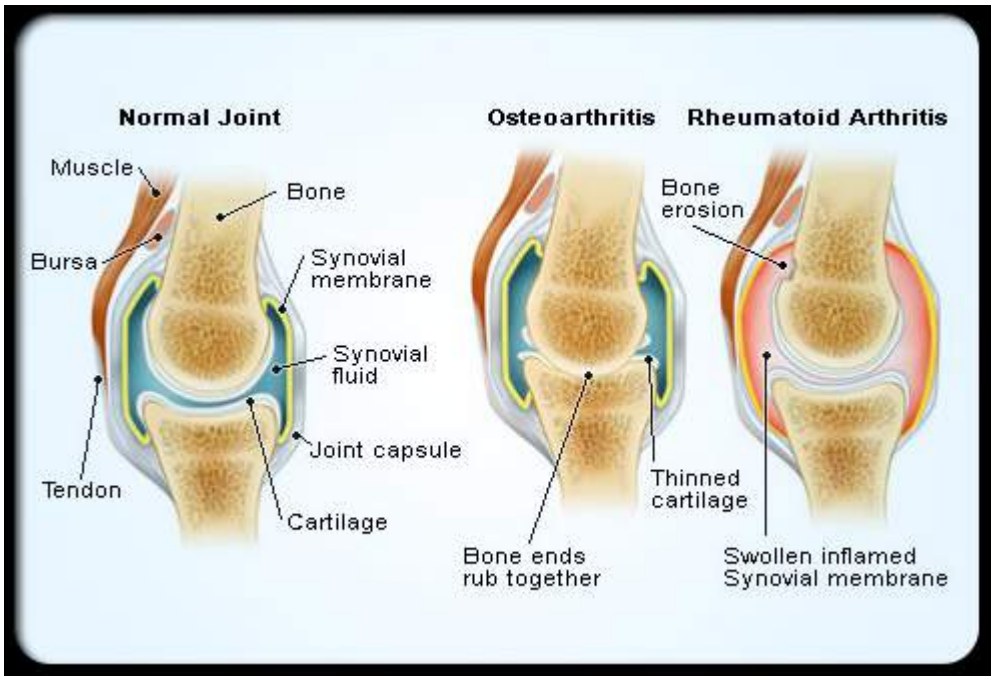
➢ Alter drug selection or reduce the dosage (Avoid drugs that are toxic to the kidney like NSAIDs)

➢ Establish need for steroid supplementation

➢ Monitor blood pressure for patients taking cyclosporine or prednisone; if blood pressure increases above baseline established, refer for medical evaluation.

Rheumatologic and Connective Tissue Disorders

Arthritis is a nonspecific term that means “inflammation of the joints.”
Arthritic disease a group of disorders of the rheumatic diseases that affect bones, joints, and muscles. more common types include rheumatoid arthritis, osteoarthritis, systemic lupus erythematosus, juvenile arthritis, Sjögren's syndrome, gout, ankylosing spondylitis (chronic inflammatory disorder of the sacroiliac joints) .



RHEUMATOID ARTHRITIS

DEFINITION,INCIDENCE & PREVALENCE;

Rheumatoid arthritis (RA) is an autoimmune disease of unknown origin that is characterized by symmetric inflammation of joints, especially of the hands, feet, and knees. Severity of the disease varies widely from patient to patient and from time to time within the same patient. the prevalence of the disease range from 1% to 2% of the population. Disease onset usually occurs between ages 35 and 50 years and is more in women than men by a 3:1 ratio. Other factors, such as socioeconomic status, education, and psychosocial stress, have been suggested to play predisposing roles.

Etiology;

The cause of RA is unknown; however, evidence shows an interrelationship of infectious agents, genetics, and autoimmunity. One theory suggests that a viral agent alters the immune

system in a genetically predisposed individual, leading to destruction of synovial tissues. Many persons who develop RA have a genetic predisposition that occurs in the form of a tissue marker called HLA-DR4; however, not everyone with this tissue type develops the disease .

Pathophysiology and Complications

Abnormality of RH involves microvascular endothelial cell activation and injury. Primary changes occur within the synovium, which is the inner lining of the joint capsule. Edema of the synovium occurs, followed by thickening and folding. This excessive tissue, composed of proliferative and invasive granulation tissue, is referred to as pannus , In addition, marked infiltration of lymphocytes and plasma cells into the capsule occurs. Eventually, granulation tissue covers the articular surfaces and destroys the cartilage and subchondral bone through enzymatic activity, This process also extends to the capsule and ligaments, causing distention and rupture. New bone or fibrous tissue then is deposited, resulting in fusion or loss of mobility.

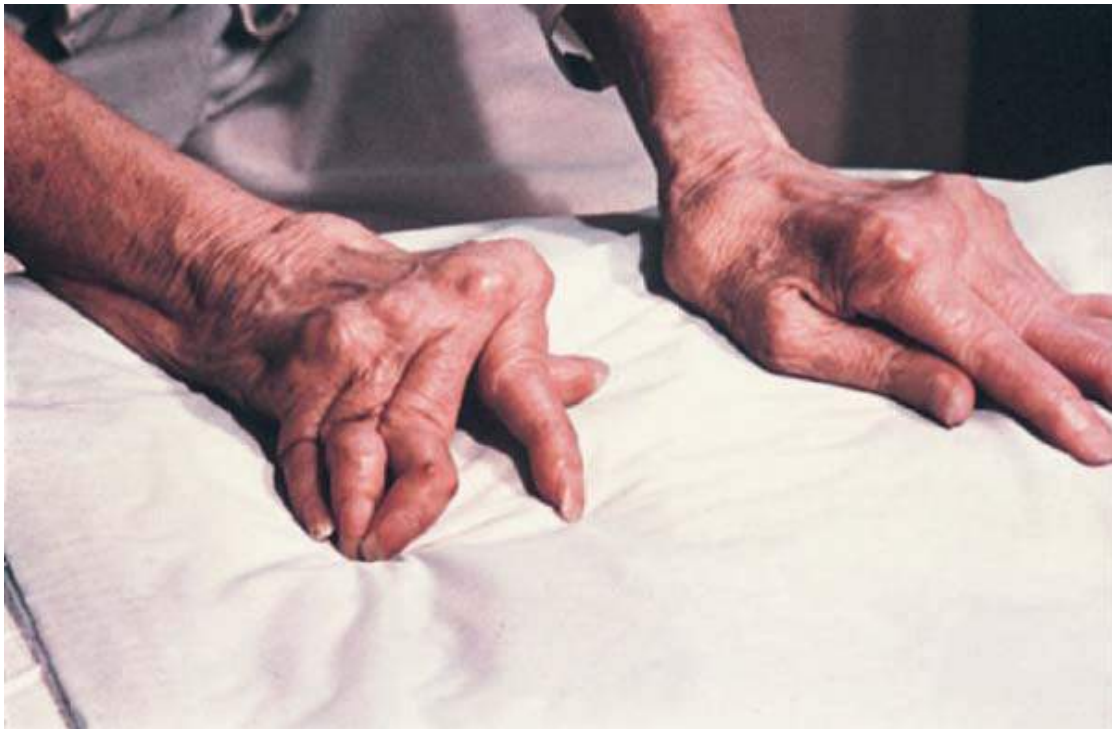
A likely sequence of events begins with a synovitis that stimulates immunoglobulin G (IgG) antibodies.These antibodies form antigenic aggregates in the joint space, leading to the production of rheumatoid factor (autoantibodies). Rheumatoid factor then complexes with IgG complement, a process that produces an inflammatory reaction that injures the joint space. The most progressive period of the disease occurs during the earlier years; thereafter, it slows. The course and severity of RA are unpredictable, but the disorder is characterized by remissions and exacerbations. For most patients, however, the disease is a sustained, lifelong problem that can be controlled or modified to allow a normal or nearly normal life. The life expectancy of persons with severe RA is shortened by 10 to 15 years. This increased mortality rate usually is attributed to infection, pulmonary and renal disease, and gastrointestinal bleeding.

Many complications may accompany RA. Included among these are; digital gangrene, skin ulcers, muscle atrophy, keratoconjunctivitis sicca (Sjögren's syndrome), TMJ involvement, pulmonary interstitial fibrosis, pericarditis, amyloidosis, anemia, thrombocytopenia, neutropenia, and splenomegaly (Felty's syndrome).

CLINICAL PRESENTATION

Signs and Symptoms

The usual onset of RA is gradual and the disorder is preceded by a prodromal phase of general fatigue and weakness with joint and muscle aches. Characteristically, these symptoms come and go over varying periods. Then, painful joint swelling, especially of the hands and feet, occurs in several joints and progresses to other joints in a symmetric fashion. Joint involvement persists and gradually progresses to immobility, contractures, subluxation, deviation, and other deformities. Characteristic features include pain in the affected joints aggravated by movement, generalized joint stiffness after inactivity, and morning stiffness that lasts longer than 1 hour. The joints most commonly affected are fingers, wrists, feet, ankles, knees, and elbows. Multiple joint changes noted in the hands include a symmetric spindle-shaped swelling of the proximal interphalangeal (PIP) joints, with dorsal swelling . The TMJ is reported to be involved in up to 75% of patients.



Hands of a patient with advanced rheumatoid arthritis. (From Damjanov I. Pathology for the Health

Comparison of Rheumatoid Arthritis and Osteoarthritis

RHEUMATOID ARTHRITIS	OSTEOARTHRITIS
Multiple symmetric joint involvement Usually one or two joints (or groups) involved	Usually one or two joints
Significant joint inflammation	Joint pain usually without inflammation
Morning joint stiffness for longer than 1 hour	Morning joint stiffness lasting less than 15 minuet
Symmetric, spindle-shaped swelling of proximal interphalangeal joints joints and subluxation of metacarpophalangeal joints .	Heberden's nodes of distal interphalangeal joints .
Systemic manifestations (fatigue, weakness, malaise	No systemic involvement

The American Rheumatism Association has developed criteria for the diagnosis and classification of RA to be used in clinical trials and epidemiologic studies (Box -1-). For the diagnosis of RA to be made, four of seven criteria must be met.

BOX- 1-

Criteria for the Diagnosis of Rheumatoid Arthritis [*]

- Morning stiffness
- Arthritis of three or more joint areas
- Arthritis of hand joints
- Symmetric arthritis
- Rheumatoid nodules
- Serum rheumatoid factor
- Radiographic changes

Modified from Arnett FC, Edworthy SM, Bloch DA, et al. Arthritis Rheum 1988;31:315-324.

* At least four must be present for a diagnosis of rheumatic arthritis

Laboratory Findings

No laboratory tests are pathognomonic or diagnostic of RA, although they are used in conjunction with clinical findings to confirm the diagnosis.

1- an increased erythrocyte sedimentation rate(ESR).
2- the presence of C-reactive protein.
3- a positive rheumatoid factor in 85% of affected patients.
4- a hypochromic/microcytic anemia.

Diagnosis:

By definition, the diagnosis of RA cannot be made until the disease has been present for at least several weeks.

Although extra-articular manifestations may dominate in some patients, documentation of an inflammatory synovitis is essential for a diagnosis. Inflammatory synovitis can be documented by demonstration of synovial fluid leukocytosis, defined as white blood cell (WBC) counts greater than 2000/mm , histologic evidence of synovitis, or radiographic evidence of characteristic erosions.

MEDICAL MANAGEMENT

The treatment approach to RA is, by necessity, palliative because no cure as yet exists for the disease. Treatment goals are to reduce joint inflammation and swelling, relieve pain and stiffness, and facilitate and encourage normal function. These goals are accomplished through a basic treatment program that consists of patient education, rest, exercise, physical therapy, and aspirin or other nonsteroidal anti-inflammatory drugs (NSAIDs).

Drugs for the management of RA have been divided into two groups: those used primarily for the control of joint pain and swelling, and those used to limit joint damage

All NSAIDs can cause a qualitative platelet defect that may result in prolonged bleeding, especially when given in high doses. The effects of aspirin are irreversible for the life of the platelet (10 to 12 days); thus,this effect continues until new platelets have replaced the old. The effect of the other NSAIDs on platelets is reversible and lasts only as long as the drug is present in the plasma .

- Drugs Used in the Management of Rheumatoid Arthritis and Systemic Lupus Erythematosus Dental and Oral Considerations

1- SALICYLATES : Aspirin, Ascriptin, Bufferin, Empirin Prolonged bleeding but not usually clinically significant

2- NONSTEROIDAL ANTI-INFLAMMATORY DRUGS : Ibuprofen, Indomethacin, naproxen, Piroxicam, Indocin, Feldene, Voltaren, Prolonged bleeding but not usually clinically significant; oral ulceration, stomatitis

3- CYCLOOXYGENASE (COX)-2 INHIBITORS

Celecoxib and Rofecoxib prolong use of these drugs cause myocardial infarction .

4- TUMOR NECROSIS FACTOR-INHIBITORS: Etanercept,Infliximab

5- INJECTABLE GLUCOCORTICOIDS :Triamcinolone hexacetonide,

Prednisolone ,Methylprednisolone acetate,produce Adrenal suppression,and masking of oral infection.

6- DISEASE-MODIFYING ANTIRHEUMATIC DRUGS (DMARDs), which are commonly employed in the treatment of patients with RA, are classified in various groups, each of which consists of multiple drugs (e.g., antimalarials, penicillamine, gold compounds)

-Antimalarial agents ;Hydroxychloroquine, Quinine, Chloroquine (Plaquenil)

-Penicillamine (Cuprimine, Depen) .those drugs has adverse effects include sever eye damage & blue black intra oral pigmentation .limited use.

-Gold compounds ;Increased infections, delayed healing, prolonged bleeding, oral ulceration, proteinuria,neutropenia & thrombocytopenia may result .

Dental Management of the Patient With Rheumatoid Arthritis

1. Short appointments
2. Ensurance of physical comfort
a. Frequent position changes
b. Comfortable chair position
c. Physical supports as needed (pillows, towels, etc.)
3. Drug considerations
a. Aspirin and NSAIDs—bleeding may be increased but usually is not clinically significant
b. Gold salts, penicillamine, antimalarials, immunosuppressives—get complete blood cell count with differential, bleeding time; treat stomatitis symptomatically
c. Corticosteroids—adrenal suppression possible
4. Joint prosthesis—prophylactic antibiotics (cephalosporin or clindamycin)
5. Technical treatment modification dictated by patient's disabilities
6. Temporomandibular joint pain/dysfunction—sudden occlusal changes possible
a. Decrease jaw function
b. Soft, diet
c. Moist heat or ice to face/jaw
d. Medication as directed by physician
e. Occlusal appliance to decrease joint loading
f. Consideration of surgery for persistent pain or dysfunction

Prosthetic joint Joint prosthesis

The need for antibiotic prophylaxis to prevent prosthetic joint infection (PJI) is controversial. Patients with high risk of PJI may require prophylactic antibiotic:

- Immunosuppressed patients.
- Type-1 diabetes mellitus.
- First 2 years after joint replacement.
- Previous PJI (prosthetic joint infection).
- Malnourished patients.
- Hemophilia.

The suggested antibiotic prophylaxis regimens are:

- Amoxicillin or Cephalexin 2g orally 1 hour before dental procedure. Patients unable to take oral medications; 1g Cefazolin or 2g Ampicillin i.m. or i.v. 1 hour before the dental procedure.
- Patients allergic to Penicillin; Clindamycin 600mg orally 1 hour before the dental procedure or i.v. in patients unable to take oral medications.

1. technical treatment modifications dictated by patients' disabilities e.g. patients with TMJ problems should not be subjected to prolonged procedures.

Oral Complications and Manifestations

The most significant complication of the oral and maxillofacial complex in RA is TMJ involvement, which is found in up to 45% to 75% of patients with RA. This may present as bilateral preauricular pain, tenderness, swelling, stiffness, and decreased mobility of the TMJ, or it may be asymptomatic. Periods of remission and exacerbation may occur, as with other joint involvement. Fibrosis or bony ankylosis can occur. Clinically, patients may present with tenderness over the lateral pole of the condyle, crepitus,limited opening, and radiographic evidence of structural change. Radiographic changes initially may show increased joint space. Later, these changes are primarily erosive and can involve both the condyles and the fossa. A particularly disturbing event is the development of an anterior open bite, caused by destruction of the condylar heads and loss of condylar height . This sudden retrognathia and anterior open bite can be severe and has been reported to cause obstructive sleep apnea. Although palliative treatment such as interocclusal splints, physical therapy, and medication may prove to be helpful, surgical intervention often becomes necessary to decrease pain, improve appearance, or restore function.

OSTEOARTHRITIS

DEFINITION

Incidence and Prevalence

Osteoarthritis (OA, degenerative joint disease). Almost everyone older than 60 years of age develops OA to some degree. OA is the leading cause of disability among the elderly. [1]
OA, which is considered a regional disease, usually affects often used joints such as hips, knees, feet, spine, and hands. The TMJ also is affected. Women twice than men; , first appearing after the age of 40. Racial differences have been noted in the prevalence of OA and in the pattern of joint involvement.

Etiology

Although the exact cause of OA is not known, many factors are now believed to be a significance Preexisting structural joint abnormalities, intrinsic aging, metabolic factors, genetic predisposition, obesity leading to overloaded joints, and macrotrauma or microtrauma are considered causative or contributory factors in the origin of the disease.

CLINICAL PRESENTATION

Signs and Symptoms

The primary symptom of OA is pain localized to one or two joints. The pain is dull ache accompanied by stiffness that is typically worse in the morning or after a period of inactivity. The pain and stiffness usually last no longer than 15 minutes. Joint noises or grinding sounds (crepitus) may be detected with movement. Redness and swelling usually are not associated with OA.
Radiographic signs of OA include narrowing of the joint space, articular surface irregularities and remodeling, and osteophytes . Symptoms often are not well correlated with radiographic signs.

Oral Complications and Manifestations:

The usual finding in patients with OA of the TMJ is onset of unilateral preauricular pain with stiffness after a period of inactivity that decreases with mild activity. Severe pain may be elicited on wide opening, and pain occurs with normal function and worsens during the day. Adjacent muscle spasm may occur , Crepitus is a common finding in the affected joint.

Dental Management of the Patient With Osteoarthritis

1. Short appointments
2. Physical comfort of patient
   a. Frequent position change
   b. Comfortable chair position

c. Physical supports as needed (pillow)
d. Drug considerations

3. Aspirin and NSAIDs
4. Joint prosthesis—generally, prophylactic antibiotics are not required unless diabetes mellitus or immunosuppression is present; if so, use cephalosporin or clindamycin
5. Technical treatment modifications dictated
6. Temporomandibular joint pain/dysfunction usually self-limiting
a. Painless jaw function encouraged
b. Soft diet
c. Moist heat or ice to face/jaw
d. Acetaminophen, aspirin, or nonsteroidal anti-inflammatory drugs (NSAIDs) for analgesia
e. Occlusal appliance to decrease joint loading
f. Surgery consideration for persistent pain or dysfunction

AIDS AND HIV INFECTION

Aetiology ;

the disease is caused by RETROVIRUS (human immunodeficiency) virus.

the chief mode of transmission by :

1- male homosexual.

2-intra venous drug abuse.

3-blood & blood products can contain the virus & many hemophilias have acquired the disease.

4-infected women to her fetus.

((the incubation period of AIDs is variable from 11-14 years.)) .

IMMUNOLOGY

the virus directly infect the lymphocytes & other cells which carry the CD4 MARKER

the virus depresses the number of

T HELPER (CD4) cells & reverses the ratio of helper to suppresser lymphocyte .

so it's depressed the cell mediated immunity.

the HIV also can bind (infect) some monocytes cells which they express the CD4 MARKER (RECEPTOR).

IMMUNOLOGY

antibody is produced in response to the presence of the virus, but not protective & the virus not eliminated from the body.

HIV also attack the CNS ,cells of which carry receptors for the virus.

RISK OF TRANSSMISION

HIV can be isolated from blood, semen,breast milk ,tears and saliva .most patient exposed to the virus shows antibody to this virus with or without symptoms .

laboratory screening test for this virus include ;

ELISA (enzyme- linked immunosorbent assay) is sensitive but high rate of false-positive so need a second ELISA ,and then by another ***test WESTERN BLOT ANALYSIS*** . This combination is accurate in 99% .

PCR (polymerase chain reaction) for direct detection of the virus but it is expensive.

RISK OF TRANSMMISION

high risk of exposure include the following ;

1- high level of viremia in the source patient .

2- exposure to large volume of infected fluid .

3- deep penetrating injury with sharp device covered with blood from infected patient .

4- needlestick injury during injection of infected patient .

Post exposure anti retroviral prophylaxsis should consist of two anti viral agents which are used to restore immune dysfunction by inhibiting viral replication ***(1)NRTIs (nucleoside reverse transcriptase inhibitors) and (2)PIs(protease inhibitors).***

RISK OF TRANSMMISION

the exposed dental health care worker should be followed with antibody testing for HIV infection at base line, 6weeks ,12 weeks .6 months.

CLINICAL ASPECTS

The full syndrome characterized by multiple

Infections bacteria ,fungi, parasites & viruses . the infection are main cause of death .

The involving of CNS include depression ,dementia & death .

Thrombocytopenic purpura or disease resembling lupus erythmatosus.

The course of the disease may be modified to some degree by drugs like ZIDOVUDINE & treatment of infection .

ORAL MANIFESTATION

I- CANDIDOSES;

Oral thrush can be seen in 70% of patient .

This may present in young adult male not receiving any drugs ,strongly suggestive of HIV infection .

II-VIRAL INFECTIONS;

- herpetic stomatitis is common .other orofacial zoster ,cytomegalovirus,epstein barr virus & papilloma virus.

III-BACTERIAL INFECTION ;

klebsiella pneumoniae ,enterobactria & E-coli . In the later stage of the disease there may be oral lesion secondary to systemic infections .

Periodontitis & acute ulcerative gingivitis are also well recognised feature .

IV -DEEP MYCOSIS;

such as histoplasmosis which may cause oral ulcerative lesion & also can destroyed the adrenal glands causing addison's disease & hyper pigmentation .

V – HAIRY LEUKOPLAKIA ;

it's hair- like filaments.

Microscopically ;The lesion appear as hyperkeratosis with an irregular surface or hair like extension.

It has no pre -malignant potential ,but 80% of patients with this lesion develop AIDS within 3 years.

VI – TUMOURS;

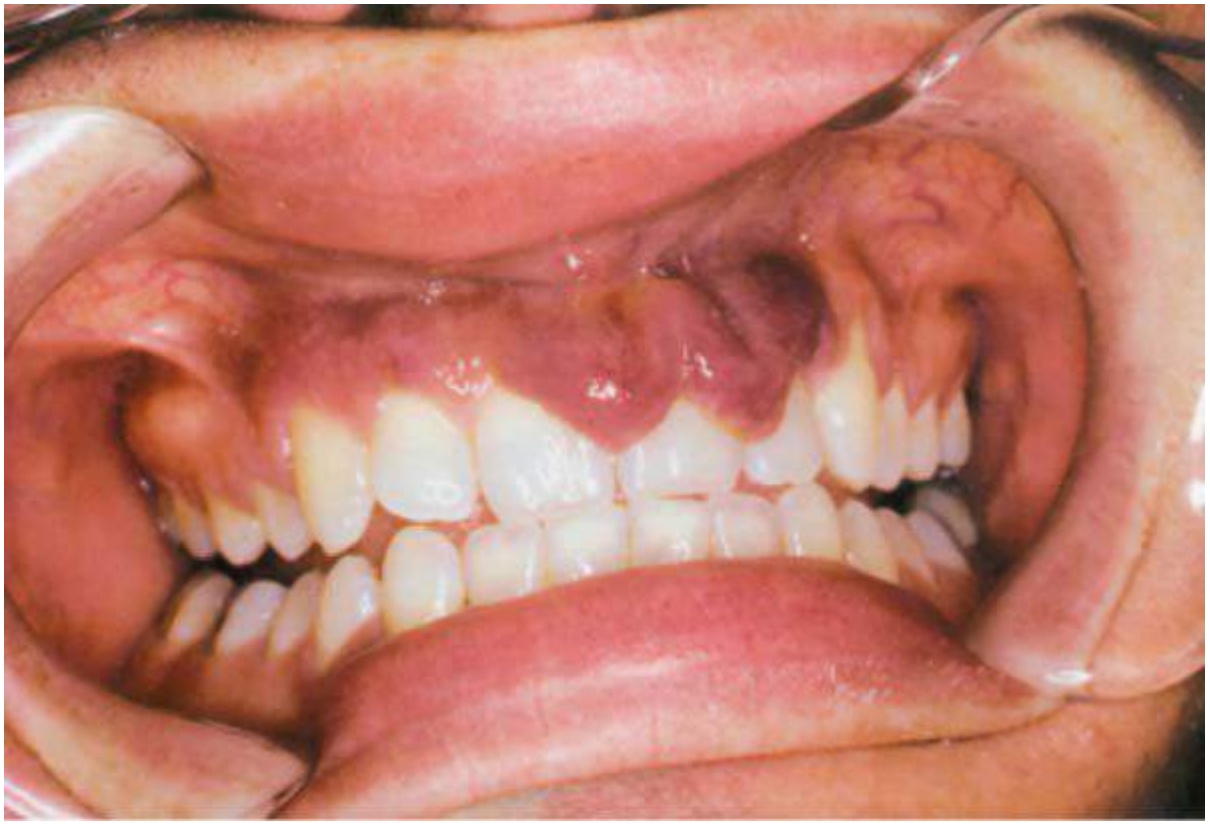
- kaposi's sarcoma (in the hard palate).it's purplish area or nodule which bleed readily .it's pathognomonic of AIDS ,unless there is any immunosupressive condition .

- non-Hodgkin lymphoma,

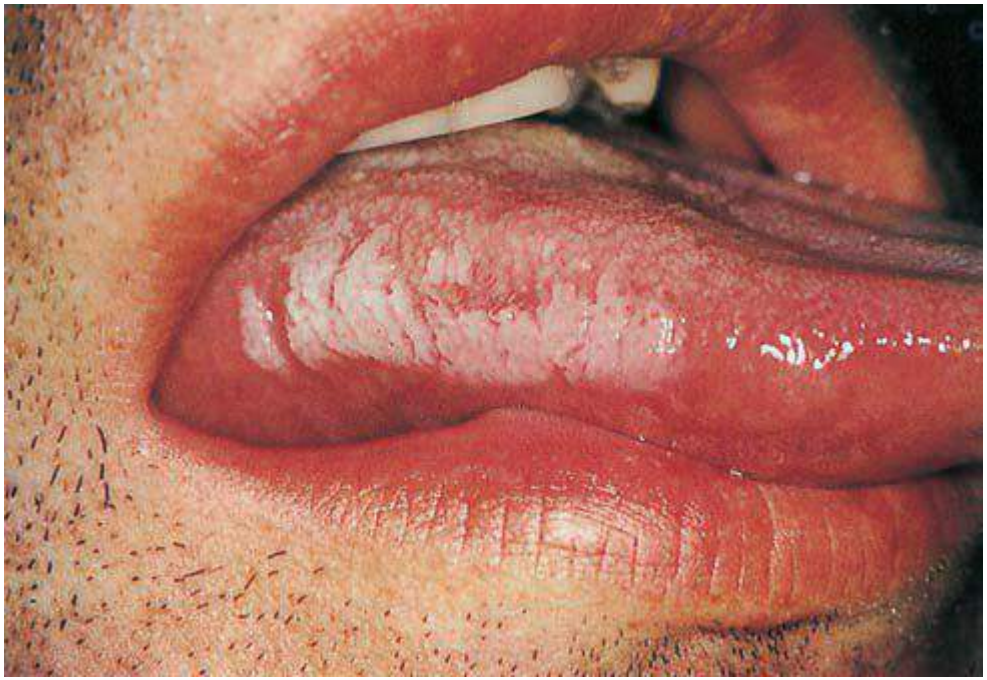
  Unlike non-AIDS patient those tumours are particularly common in head & neck .

- Squamous cell carcinoma

KAPOSI SARCOMA



HAIRY LEUKOPLAKIA



VII- LYMPHADENOPATHY ;

cervical lymphadenopathy is the most common head & neck manifestation of HIV

VIII- SALIVERY GLAND DISEASE;

-PAROTITIS .

-A SJOGREN SYNDROM

DENTAL MANAGEMENTS

1- ASYMPTOMATIC PATIENT .

2- SYMPTOMATIC PATIENT .

(HAS APPORTUNISTIC INFECTION).

3- PATIENT WITH THROMBOCYTPENIA

4- ANY LESION IN THE ORAL CAVITY SHOULD BE DIAGNOSED .

5- MEDICAL CONSULTATION ,BLOOD FILM ,DIFFRENTIAL WCC " NEUTROPHIL LAWER THAN 400 CELL/CUBIC MM" .

6- PATIENT WITH SEVER SYMPTOMES.

ASYMPOMATIC PATIENT

Patients who have been exposed to the virus & HIV seropositive but asymptomatic may receive all indicated dental treatment (CD4+cell count more than 400).

symptomatic patient , for early stages of AIDS ,with advanced immunosuppression and neutropenia (CD4+cell count lower than 200 and or neutrophil count lower than 500/cubic mm) have increased susceptibility to opportunistic infection & need prophylactic drugs for invasive procedure. Any source of oral or dental infection should be eliminated . chlorhexidine mouth wash may be helpful.

Patient with sever thrombocytopenia below 50 000/cubic mm ,may required special measures (platelet replacement) before any surgical treatment including scaling & polishing .

PAIENT WITH SEVERE SYMPTOMS

Sever symptoms of AIDS managed by treatment of their more urgent dental needs to prevent pain & infection , with deferment of extensive restorative procedures .

PATIENTS ON RADIOTHERAPY AND CHEMOTHERAPY

The effective management of the patient with cancer often requires a team approach that involves; dental, medical, surgical, radiotheraputic, chemotherapeutic, reconstructive, and psychiatric consideration.

Cancer could affect any organ of the body such as breast, lung, G.I.T., urinary system, bone, prostate, skin, oral cavity, endocrine system.

❖ *Lines of Treatment of Cancers:*

1- Surgery
2- Radiotherapy
3- Chemotherapy
4- Combination of The Above Lines.
5- Cytotoxic Drugs, Endocrine Drugs.

❖ *PATIENTS ON RADIOTHERAPY*

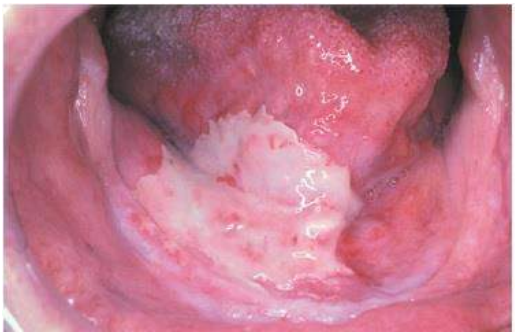
The primary goal of radiotherapy is to destroy and damage the tumor cells, but in fact the normal cells which lies at the line of radiation will be destroyed also and the area that exposed to radiation will be compromised. Radiotherapy of the head and neck region has a direct relation with our job as dentists.

❖ Radiation Effects on Normal Tissues in the Path of the External Beam:

1. MUCOSA (mucositis)

• Epithelial changes (atrophy), causing mucositis.

Mucositis defined as an inflammation of the oral mucosa, results from the direct cytotoxic effects of radiation on rapidly dividing oral epithelium. It develops more often in non keratinized mucosa (buccal and labial mucosa, ventral tongue) and adjacent to metallic restorations by the end of the second week of radiation therapy (if the dose is 200 cGy per week).



Extensive mucositis that developed from the effects of radiation on the oral mucosa.

2. MUSCLE

• Muscular dysfunction; it's a late complication of radiotherapy it results from Fibrosis and vascular damage of muscles of mastication and limitation in the mouth opening, which should be maintained through physiotherapy.

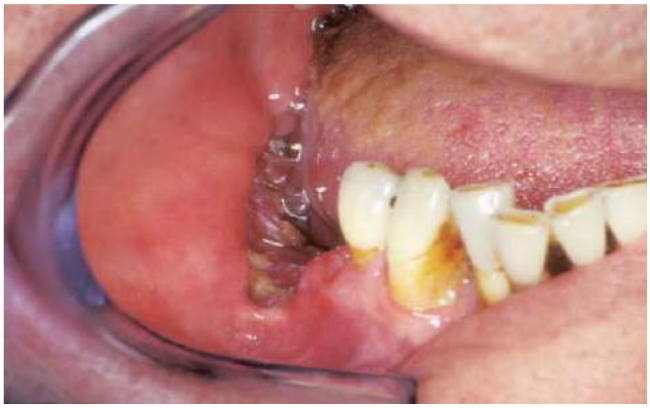
The patient also should perform daily stretching exercises to relieve trismus and apply local warm moist heat. One exercise is for the patient to place a given number of tongue blades in the mouth at least three times a day for 10-minute intervals. By slowly increasing the number of tongue blades, muscle stretching will occur, and more normal function will ensue.

3. BONE

• Decreased numbers of osteocytes and osteoblasts, decreased blood flow and the patient becomes more susceptible to develop an osteoradionecrosis.

Osteoradionecrosis ORN results from radiation-induced (hypocellularity, hypovascularity, and ischemia) in the jaws. Most cases result from damage to tissues overlying the bone rather than from direct damage to the bone. Accordingly, soft tissue necrosis usually precedes ORN and is variably present at the time of diagnosis. Risk is greatest in posterior mandibular sites for patients whose jaws have been treated with in excess of 6500 cGy, and who have undergone a traumatic procedure (e.g., extraction). Risk is greater for dentate patients than for edentulous patients, and periodontal disease enhances risk. Spontaneous ORN also occurs. This risk continues throughout a patient's life time.

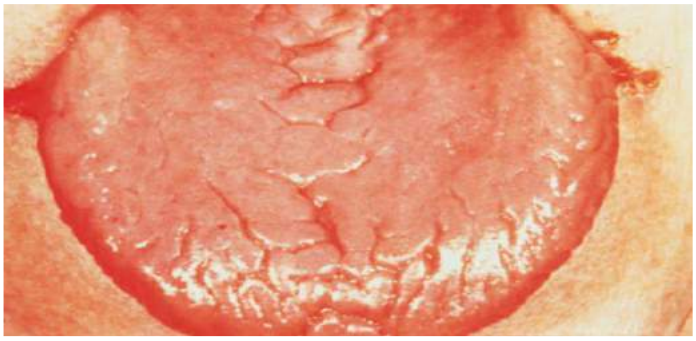
Note: (the amount of radiation absorbed by the tissue is called the radiation dose. before 1985 dose was measured in a unit called rad, but now the unit of radiation dose is called Gray (Gy) one Gy is equal to 100 rads , one centigray (cGy) is the same as one rad .



Osteoradionecrosis. Exposed necrotic bone in the posterior mandible edentulous ridge of a patient who previously received radiation therapy to the head and neck region.

4. SALIVARY GLANDS

• Atrophy of acini, vascular changes and chronic inflammation, fibrosis, leading to xerostomia, due to head and neck radiation therapy. Management by frequent drinking of water, using salivary substitutes, sugarless chewing gum, lozenges and drugs like parasympathomimetic drugs to stimulate salivary flow.

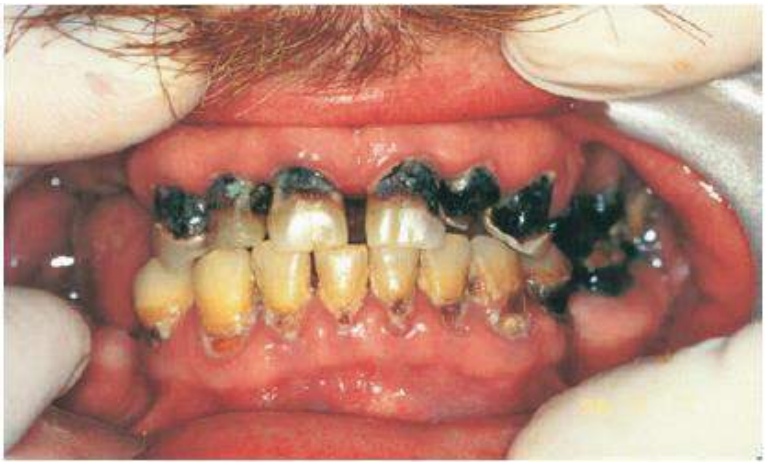


Severe xerostomia that developed from the effects of radiation on the oral mucosa. Note the angular cheilitis.

5. TEETH

Radiation caries: Radiation caries is estimated to occur 100 times more often in patients who have received head and neck radiation than in normal individuals due to hyposalivation. It can progress within months, advancing toward pulpal tissue and resulting in periapical infection that extends to surrounding irradiated bone. Extensive infection and necrosis may result. Management required patient education, oral hygiene instruction, fluoride application and frequent dental visits and early restoration of the teeth.

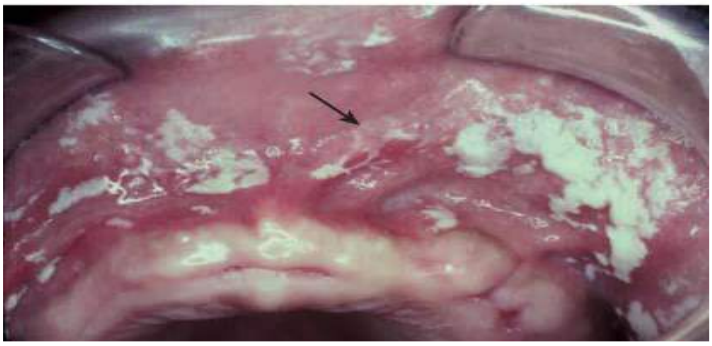
Sensitivity of the teeth increased due to hypo salivation.



Extensive cervical caries in a patient who received radiotherapy.

6. SECONDARY INFECTION

Occurs as a result of immunosuppression and reduced salivary flow, when the WBC count falls below 2000 cell/ml the immune system is less able to manage these infections.



Oral candidiasis (pseudomembranous form) in a patient undergoing chemotherapy. *Arrow* indicates lesions of pseudomembranous candidiasis.

Other effects of radiation include loss of taste result from damage of the microvilli of taste cells, the patient complain of bitter test, most of the patient will restore taste within 3-4 months after therapy. Zinc supplementation is reported to improve taste sensation (zinc sulfate).

DENTAL MANAGEMENT:

A. Prior to Radiotherapy:

Before the radiotherapy the patient should be examined and a treatment plane is done for a full mouth treatment taking in consideration the following:

1. **Symptomatic non vital teeth should be endodontically treated** at least 1 week before initiation of head and neck radiotherapy. However, dental treatment of asymptomatic teeth even with periapical involvement can be delayed.
2. Teeth which are indicated for extraction should be extracted
   - ❖ **Indications of extraction:**

i. Non restorable teeth with poor or hopeless prognosis, acute infection, or severe periodontal disease that may predispose the patient to complications (e.g., sepsis, osteoradionecrosis) should be extracted.
ii. Presence of periapical inflammation
iii. Broken-down, nonrestorable, nonfunctional, or partially erupted tooth in a patient who is noncompliant with oral hygiene measures.
iv. Patient lack of interest in saving tooth/teeth
v. Inflammatory (e.g., pericoronitis), infectious, or malignant osseous disease associated with questionable tooth

3. The **Guidelines for extraction of teeth**:

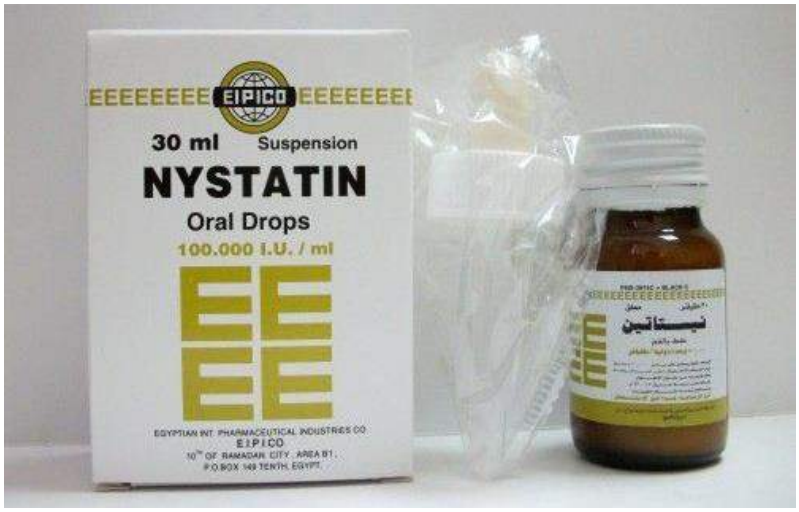
- Perform extraction with minimal trauma
- At least 2 weeks, ideally 3 weeks before initiation of radiation therapy to get enough time for healing.
- Trim bone at wound margins to eliminate sharp edges
- Obtain primary closure.
- Avoid intra-alveolar hemostatic packing agents that can serve as a nidus of microbial growth.

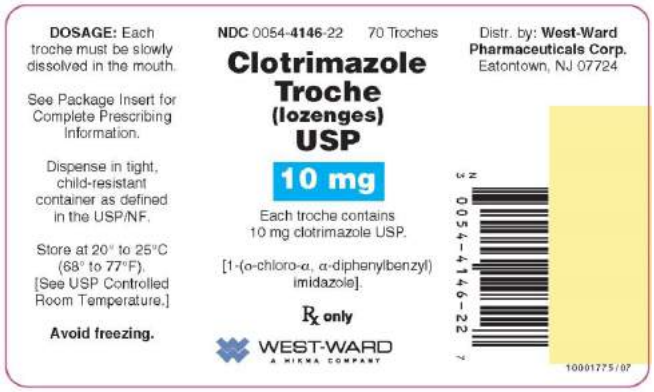
4. Teeth **scaling and prophylaxis** should be provided before radiotherapy is initiated.
5. **Patients** who will be retaining their teeth and undergoing head and neck radiation therapy must be **informed about problems** associated with decreased salivary function, which include xerostomia, the increased risk of oral infection, including radiation caries, and the risk for osteoradionecrosis.

B. During Radiotherapy:

1. **Symptomatic treatment** of mucositis: The treatment of mucositis involve:
   1) Oral mucositis can be reduced by using oral cryotherapy, low-level laser therapy, systemic analgesics, and supplemental zinc.
   2) Eliminate any irritating factor such as a sharp edge; and establish good oral hygiene.
   3) Recommend a salt and sodium bicarbonate mouthwash
   4) Topical anesthetics (viscous lidocaine 0.5%) and/or an antihistamine solution [Tantum rinse], diphenhydramine [Benadryl], promethazine [Phenergan]) to provide pain control
   5) Antimicrobial rinses such as chlorhexidine 0.12% mouth wash.
   6) Prescribe antiinflammatory agents (e.g., topical steroids).
   7) Avoid tobacco, alcohol and irritating foods.
   8) Oral lubricants and lip balms with a water base, beeswax base, or vegetable oil base
   9) Humidified air (humidifiers or vaporizers);
   10) Follow a soft diet; maintain hydration.
   11) Consider systemic antimicrobials, if severe.
2. **Management of xerostomia**:
   Recommend sugarless lemon drops, sorbitol-based chewing gum, buffered solution of glycerine, salivary substitutes and plenty of water and other fluids (sip drinks constantly to keep the oral mucosa moist; such drinks should not be products containing a fermentable carbohydrate or carbonic acid) and avoid the diuretics such as coffee or tea.
3. **Prevention of trismus** by having the patient place tongue blades or Mouth blocks each day to maintain mouth opening.
4. **Diagnosis and treatment of secondary infections** that developed during radiation. Because of the quantitative decrease that occurs in actual salivary flow, and because of compositional alterations in saliva, several organisms (bacterial, fungal, and viral) may opportunistically infect the oral cavity. The organism that most frequently opportunistically infects the oral cavity in individual undergoing cancer therapy (who have hyposalivation and immunosuppression) is Candida albicans. Candidiasis is best managed with the use of topical oral antifungal agents, these include nystatin (oral suspension 100,000 international units [IU]/mL 4 to 5 times daily) and clotrimazole (Mycelex lozenges 10 mg five times a day). Prophylactic use of antifungal agents may be required in patients undergoing chemotherapy who have frequent recurrent infections. Ketoconazole (Nizoral), fluconazole (Diflucan), or itraconazole (Sporanox) may be used if systemic therapy is warranted or if patients develop unusual oral fungal infections Recurrent herpes simplex virus (HSV) eruptions are infrequent during radiation therapy.

5. **Fluoride application** to prevent sensitivity of teeth and to prevent caries.
6. **Instruct** the patient for **oral hygiene measures.**
7. **Dentures should not be worn** until the acute phase of mucositis has resolved. Dentures should be cleaned and soaked with an antimicrobial solution daily for the prevention of infection.





Some of medications used to treat fungal infections in patient on radiotherapy (most frequently Candida albicans).

C. Following Radiotherapy:

1. **Consultation with the physician** to determine whether the patient is cured or in remission or is completing palliative care.
2. **The patient should be examined** every 1 to 3 months during the first 2 years, every 3 to 6 months thereafter and after 5 years examined at least once per year, to check the oral hygiene and treatment of initial caries and management of xerostomia.
3. **Management of xerostomia** as mentioned before. The manifestations of salivary hypofunction in patients having undergone radiation therapy for head and neck cancer include severe salivary hypofunction (unstimulated salivary flow <0.2 mL/min), mucositis, cheilitis, glossitis, fissured tongue, glossodynia, dysgeusia, dysphagia, and a severe form of caries called radiation caries.
4. **Prevention of osteoradionecrosis**:

A. Avoid the extraction of teeth as much as possible

Endodontic therapy is preferred over extraction (assuming the tooth is restorable).

B. If the extraction is unavoidable, then it should be done **with the following precautions**:

I. It is better to use local anesthesia **without adrenaline**.

II. Give the patient a **prophylactic dose** of antibiotic before and after the extraction i.e amoxicillin 2g one hour before extraction, then continue with amoxicillin 500mg 3-times daily for one week.

III. **Atraumatic procedure**.

- Follow atraumatic surgical technique.
- Avoid periosteal elevations in order to maintain a good blood supply to the bone.
- Limit extractions to two teeth per quadrant per appointment.
- Irrigate with saline, obtain primary closure, and eliminate bony edges or spicules.

IV. The use of **hyperbaric oxygen therapy** before and after the tooth extraction. Hyperbaric oxygen therapy is administration of oxygen under pressure to the patient, this process will increase the local tissue oxygenation and vascular ingrowth into the hypoxic tissue, the usual protocol for such treatments is to have 20 hyperbaric oxygen therapy dives before extraction and 10 more dives immediately after extraction. The patient usually undergoes one hyperbaric oxygen therapy each day, therefore it takes 4 weeks before surgery and 2-weeks of treatment after surgery.

5. Maintain **good oral hygiene.**
   - Use oral irrigators.
   - Use antimicrobial rinses (chlorhexidine)
   - Use daily fluoride gels.
   - Eliminate smoking.
   - Attend frequent postoperative recall appointments
6. If the **patient developed osteoradionecrosis**:

Once necrosis occurs, conservative management usually is indicated. Exposed bone should be irrigated with a saline or antibiotic solution, and the Bony sequestra should be removed to allow for epithelialization. If swelling and suppuration are present, broad-spectrum antibiotics are used.

Severe cases benefit from hyperbaric oxygen (60- to 90-minute dives 5 days per week, for a total of 20 to 30 dives). Cases that do not respond to conservative measures may require surgical resection of involved bone.

❖ *PATIENTS ON CHEMOTHERAPY:*

Chemotherapeutic drugs used in treatment of malignancies, based on their ability to destroy or retard the division of rapidly proliferating tumor cells, unfortunately, normal host cells that have a high mitotic index are also adversely affected, especially the epithelium of the G.I.T. (including the oral cavity) and the cells of the bone marrow.

The Effect Of Chemotherapy On Normal Tissues:

1. Oral Mucosa

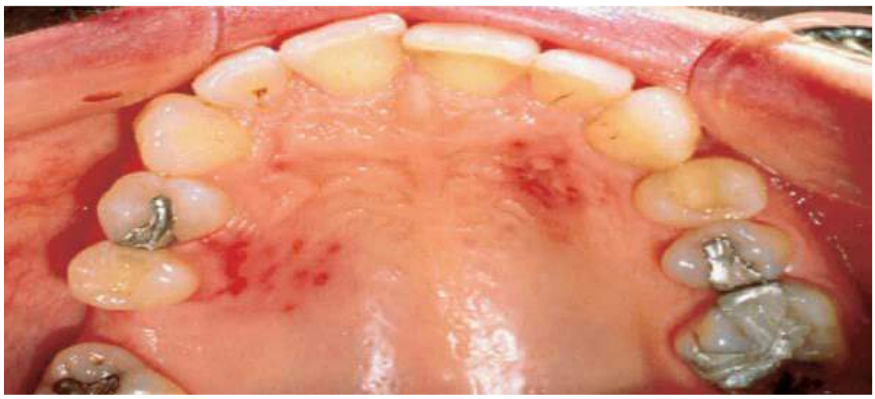
It reduces the turn over rate of oral epithelium, this leads to atrophic and ulcerative mucosal surface.

2. Hematopoietic system

Myelosupression, appear within two weeks and that manifested by:

I. leucopenia and, neutropenia, that leads to the development of an opportunistic infections as viral, bacterial, fungal(candida). Systemic infections are responsible for about 70% of deaths in patients receiving chemotherapy.

II. Thrombocytopenia, so that Gingival bleeding and submucosal hemorrhage as a result of minor trauma (e.g., tongue biting, tooth brushing) can occur when the platelet count drops to below 50,000 cells/mm^3. Palatal petechiae, purpura on the lateral margin of the tongue, and gingival bleeding/oozing are common features. Gingival hemorrhage is aggravated by poor oral hygiene.



Recurrent herpes simplex virus infection presenting as a large ulcer on the palate of a patient undergoing chemotherapy.

DENTAL MANAGEMENT

I. Prior to chemotherapy:

A thorough clinical and radiographic examination, and all sources of oral infection should be eliminated, as follow:

1. Symptomatic nonvital teeth should be endodontically treated at least 1 week before initiation of chemotherapy.
2. Teeth which are indicated for extraction should be extracted

(indications of extraction)

i- Nonrestorable teeth with poor or hopeless prognosis, acute infection, or sever periodontal disease that may predispose the patient to complications (e.g., sepsis) should be extracted.

ii- Partially erupted.or Tooth is associated with an inflammation(e.g., pericoronitis).

iii- Patient has no interest in saving tooth/teeth

3. The guidelines for extraction of teeth:
   • Perform extraction with minimal trauma
   • Ideally one week before initiation of chemotherapy
   • Trim bone at wound margins to eliminate sharp edges
   • Obtain primary closure
   • Avoid intra-alveolar hemostatic packing agents that can serve as a nidus of microbial growth.
4. Tooth scaling and prophylaxis should be provided before chemotherapy is initiated.
5. In children undergoing chemotherapy, mobile primary teeth and those expected to be lost during chemotherapy should be extracted, and gingival opercula should be evaluated for surgical removal to prevent entrapment of food debris and causes an infection.
6. Orthodontic bands should be removed before chemotherapy is began.

II. During the chemotherapy:

1- The dentist should be familiar with the patient's WBC count and platelet status before providing dental care. In general, emergency dental procedures can be performed if the **granulocyte count is greater than 2000/mm3**, and the **platelet count is greater than 50,000/mm3**. and the **patient feels capable of withstanding dental care**.

2- Provide routine care 17- 20 days after chemotherapy or few days before the start of the second chemotherapy cycle. and you have to avoid routine dental care during the chemotherapy .and if the patient needs an emergency treatment, it can be done taking these points in consideration:

a. If urgent care is needed and the platelet count is below 50,000/mm 3, consultation with the patient's oncologist is required. Platelet replacement may be indicated if invasive or traumatic dental procedures are to be performed.

b. If urgent dental care is needed and the granulocyte count is less than 2000 cells/mm 3, consultation with the physician is recommended and antibiotic prophylaxis should be provided. This prophylaxis starting at least 1 hour before any invasive procedure that involves bone, pulp, or periodontium i.e amoxicilline 2g one hour before extraction, then continue with amoxicilline 500mg 3-times daily for at least 3 days.

3- Topical therapy that includes the use of pressure, thrombin, microfibrillar collagen, and splints may be required.

4- Treatment of oral infections:

The organism that most frequently opportunistically infects the oral cavity in individual undergoing cancer therapy (who have hyposalivation and immunosuppression) is Candida albicans. Candidiasis is best managed with the use of topical oral antifungal agents. These include nystatin (oral suspension 100,000 international units [IU]/mL 4 to 5 times daily).

Recurrent herpes simplex virus (HSV) eruptions occur often during chemotherapy if antiviral agents are not prophylactically prescribed.

A daily dose of at least 1 g acyclovir/equivalent is needed to Suppress HSV recurrences.

III. After chemotherapy:

After chemotherapy has been provided, consultation with the physician is recommended to determine whether the patient is cured or not, if cancer therapy has been completed and remission or cure is the outcome, the patient with cancer should be placed on an oral recall program. Usually, the patient is seen once every 1 to 3 months during the first 2 years and at least every 3 to 6 months thereafter. After 5 years, the patient should be examined at least once per year. This recall program is important for the following reasons:

- A patient with cancer tends to develop additional lesions
- Latent metastases may occur

Dental management of Medically Compromised Patients

Allergy

Allergy is an abnormal or hypersensitive response of the immune system to a substance introduced into the body. account for about 6% to 10% of all adverse drug reactions.

Aetiology and pathophysiology

Allergic diseases comprise a series of repeated reactions to a foreign substance, involving (1) immunologic hypersensitivity and elements of the (2) nonspecific and (3) specific (A-humoral, B-cellular) branches of the immune system. The initial function of the **humoral** and **cellular** branches of the immune system → **recognition** of antigens; however, cells **and** chemicals from the *nonspecific* branch of the immune system → act to *eradicate* antigens.

A-Humoral Immune System. B lymphocytes recognize specific foreign chemical (by the help of T lymphocytes and macrophages) → transforms into plasma cell → produces immunoglobulins (antibodies) specific for the antigen involved. Type I, type II, and type III hypersensitivity reactions involve elements of the humoral immune system.

Type I hypersensitivity Is IgE-mediated hypersensitivity. This may be caused by food substances (e.g., shellfish, nuts, eggs, milk), **antibiotics**, and insect bites→release of chemical mediators from mast cells and basophils. Occur (usually) soon after **second contact** with an antigen. Include the following **types;**

Anaphylaxis
Antigen-IgE antibody complexes form on the surface of **mast** cells → **histamine** released →smooth muscle of the **bronchi** contraction + increased vascular permeability →acute respiratory compromise + cardiovascular collapse.

Atopy
Influenced by hereditary factors. Hay fever, asthma, urticaria, and angioedema are **examples** of atopic reactions. ***Lesions*** include **urticaria** (superficial lesion of the skin), and **angioedema** (lesion in the deep dermis or subcutaneous tissues) → diffuse enlargement of the lips, infraorbital tissues, larynx, or tongue.

There are many types of **angioedema**. Three types of interest to dentistry;

#Acquired (allergic-based)

#Drug-induced → due to impaired *bradykinin* degradation → vasodilation, increased tissue permeability and enema. Occur after administration of certain drugs, such as angiotensin converting enzyme inhibitors.

#Hereditary → due to a deficiency or dysfunction of complement C1 inhibitor, which can be triggered by **trauma** → activation of the complement cascade and Hageman factor (factor XII) and overproduction of *bradykinin* → vasodilation, increased tissue permeability and edema.

Type II Hypersensitivity Reactions. (**cytotoxic** reactions) These reactions are IgG-or IgM-mediated. (e.g. transfusion reaction caused by mismatched blood and Rhesus incompatibility).

Type III Hypersensitivity Reactions. (immune complex–mediated) immune complexes→ in blood vessels → vasculitis. Clinical examples include systemic lupus erythematosus and streptococcal glomerulonephritis.

B-Cellular Immune System. (Delayed immune system), T lymphocytes play the central role (by production of various cytokines) →recognize and eradicate antigens (e.g. viruses, tuberculosis, and leprosy) in cells or tissues.

Type IV Hypersensitivity Reactions. Involve the **cellular** immune system. Dendritic cells and Langerhans cells, present the antigen (e.g. metal jewellery, perfumes, rubber products, chemicals such as formaldehyde, and medicines such as topical anaesthetics) to undifferentiated T lymphocytes. These reactions usually are **delayed** (48 to 72 hours after contact with antigen). These include;

1- Infectious-type allergic reactions; e.g. tuberculin skin test → characterized by induration, erythema, swelling, and sometimes ulceration at the site of injection.
2- **Contact** allergy; Low molecular weight substance (Not antigenic) comes in contact with a tissue component (primarily a protein) → antigenic complex → hapten (or one half of an antigen) →sensitization of T lymphocytes. Example; Poison ivy
3- Graft rejection; occurs when organs or tissues from one body are transplanted into another body, when donor and recipient are genetically not identical or the host immune response has not been suppressed enough.
4- Graft-versus-host reaction; Rare and occurs **in** bone marrow transplant **recipients** → due to deficient cellular immune system (e.g. due to irradiation). *Lymphocytes* transferred to the host attempt to *destroy host* tissues.

C-Nonallergic Reactions. Other agents may cause mast cells to release their mediators without inciting a true allergic reaction;

> **e.g.** Chronic urticaria; caused by certain *drugs (e.g. anaesthetics)*, temperature changes, and emotional states (there is no **antigen-antibody** reaction), result from damage to the mast cells →referred to as anaphylactoid or anaphylaxis-like reactions.

Note; Management of anaphylactic and anaphylactoid reactions clinically similar → Both are viewed as true allergic reactions.

MEDICAL MANAGEMENT

A- Atopy
- injections to gradually desensitize.
- Move to an area that does not contain the antigen (asthmatic patients) or treated with systemic steroids
- Hay fever or urticaria are treated with antihistamines.

B- Contact dermatitis
- Variety of treatments; topical steroids, have been used.

C- From a dental standpoint;
- Patient treated for allergies →increased chance of being allergic to another substance.
- Use of steroids as allergy treatment → reaction to stress may be impaired

DENTAL MANAGEMENT

Medical Considerations

- History must be expanded →determine exactly what the offending substance was
- Six common aspects should be considered to *avoid provoking allergic* reactions;

FIRST: Local Anaesthetics.

I- Differentiate signs and symptoms of **true** allergy (D) to those cause by **other** reactions (A, B & C) most often associated with local anaesthetics;

A- Toxic reaction
  a- Causes
    1- intravenous injection of the aesthetic solution.
    2- Excessive amounts of an aesthetic
    3- vasoconstrictor.
  b- Signs and symptoms
    1- Talkativeness
    2- Slurred speech
    3- Dizziness
    4- Nausea
    5- Depression
    6- Euphoria
    7- Excitement
    8- Convulsions

B- Vasoconstrictor reaction; Signs and symptoms: -
    1- tachycardia,
    2- apprehension,
    3- sweating,
    4- hyperactivity.

C- Psychomotor reactions;
  1- Hyperventilation
  2- Vasovagal syncope (bradycardia, pallor, sweating)
  3- Sympathetic stimulation (anxiety, tremor, tachycardia, hypertension)

D- True allergic reactions

I- If the **history** supports a true allergic reaction to a local anaesthetic → Signs and symptoms **(Soft tissue swelling, Urticaria, Swelling, Skin rash, Chest tightness, Dyspnea, shortness of breath, Rhinorrhoea, Conjunctivitis**) these steps should be followed

A- Patients allergic to local anaesthetics and the type caused the allergic reaction can be identified

i. New anaesthetic with a different basic chemical structure (Ester. Amide) can be used

ii. When administering an alternative anaesthetic to a patient with a history of a local anaesthetic allergy, these steps should be followed:

1. Inject slowly, aspirating first to avoid I.V. injection.
2. Place 1 drop of the solution into the tissues.
3. Withdraw the needle, and wait 5 minutes to see what reaction occurs
4. Aspirate before giving the second injection.

B- Patients who have been allergic to local anaesthetics but cannot identify which type caused the allergic reaction

a- Attempt to contact the previous dentist involved, or

b- An antihistamine (e.g., diphenhydramine [Benadryl]; 1% solution of diphenhydramine that contains 1: 100,000 epinephrine) can be used as the local anaesthetic, or

c- The patient may be referred to an allergist for provocative dose testing (PDT)

Notes;

1- The use of diphenhydramine induces anaesthesia of about 30 minutes. No more than 50 mg of diphenhydramine should be given in a single appointment.

2- Skin testing **alone** for local anaesthetic allergy → usually **false**-positive→ PDT better

II- For severe **allergic** reaction occur **in** dental **office** due to injection of local anaesthetics; see "management of **severe** type I hypersensitivity reactions" below

SECOND: Penicillin.

A- Take medical history on all patients, including penicillin previous contact and/or reactions as well as allergic reactions to other agents

Notes;

- Skin testing for allergy to penicillin is reliable
- Sensitizing a patient to penicillin seems highest with Topical application and least with Oral administration

B- If penicillin is needed in dentistry;

1- Patient is allergic to penicillin

i- Treated with an alternative antibiotic.

Examples; Erythromycin or clindamycin for the treatment of oral infection **or** clindamycin for prophylaxis against infective endocarditis.

ii- Drugs that may cross-react, including ampicillin, carbenicillin, and methicillin, should be avoided.

iii- Cephalosporins are often used as alternatives to penicillin, however cephalosporins cross-react in 5% to 10% of penicillin-sensitive patients →skin testing is recommended

2- Patients with a negative history of allergy to penicillin
   a- Can be treated with the drug when indicated (**oral** route).
   b- Patient is observed for 30 minutes after the first dose
   c- Do not use penicillinase-resistant penicillins unless infection is caused by penicillinase-producing staphylococci.
   d- Patient should seek immediate care if any of the signs or symptoms of an allergic reaction occur after leaving dental office.

THIRD; Analgesics.

A- Aspirin
   i- Causes prolonged prothrombin time and to inhibit platelet function →usually of little clinical importance, except in patients with a haemorrhagic disease or a peptic ulcer →avoided.
   ii- Aspirin provokes a severe hypersensitivity reaction (acute bronchospasm, rhinorrhoea, and urticaria) in some patients with asthma→ be cautious
   iii- The dentist should be aware analgesic preparations that include salicylates.
   iv- Most NSAIDs have the potential for cross-sensitivity in patients who exhibit an asthma-like reaction to aspirin.

B- Codeine; Is a narcotic analgesic that commonly is used in dentistry. Emesis, nausea, and constipation may occur with analgesic doses of codeine and should be differentiated form allergic reactions.

FOURTH; Rubber Products.

i- **Anaphylaxis** may occur in the sensitized person after contact has been made with rubber gloves, rubber dam material, blood pressure cuffs, or any other product containing latex.
ii- Latex-allergic persons have IgE antibodies for specific latex proteins.
iii- Latex skin tests are a satisfactory for identifying sensitized individuals.
iv- Nitrile gloves can minimize these adverse reactions.

FIFTH; Dental Materials and Products.

i- Topical aesthetics can cause type I reactions consisting of urticarial swelling.
ii- Mouth rinses and toothpastes containing phenolic compounds, antiseptics, astringents, or flavouring agents have been known to cause type I, type III, and type IV hypersensitivity reactions **involving** the oral mucosa or lips.
iii- Hand soaps used by dental care workers reported as a cause of type IV reactions.
iv- Some dental *agents* can lead to type IV hypersensitivity (contact stomatitis) (described in “Management of Oral Complications and Manifestations”).

SIXTH**;** Hereditary Angioedema.

A condition that can be provoked by dental surgery and trauma and is best managed by

1- Implementation of preventive measures.
2- Androgens such as danazol and stanozolol, which increase hepatic production of C1 inhibitor, help to decrease the number and severity of attacks.
3- Newer agents that include C1 inhibitor concentrate (Cinryze or Berinert) show benefit but are expensive.
4- Use of such preventive agents is important, because hereditary angioedema does not respond well to epinephrine or antihistamines.

Management of Oral Complications and Manifestations

A-Hypersensitivity

I. Type I Hypersensitivity (Oral or Paraoral type I hypersensitivity, Atopic reactions) cause; **Urticarial** swelling (or **angioedema**)

1- Signs and symptoms

a. Reaction occurs soon after contact with antigen.
b. Painless swelling (due to transudate from the surrounding vessels)
c. Itching and burning may occur.
d. May remain for 1 to 3 days.

2. Treatment

a. Reaction not involving tongue, pharynx, or larynx and with no respiratory distress noted; requires 50 mg of diphenhydramine four times a day until swelling diminishes.
b. Reaction involving tongue, pharynx, or larynx with respiratory distress noted requires the following:
    i. 0.5 mL of 1: 1000 epinephrine, IM or SC
    ii. Oxygen
    iii. Once immediate danger is over, 50 mg of diphenhydramine should be given four times a day until swelling diminishes.
c. Further contact with the antigen must be avoided

II. Type III Hypersensitivity.

1. Usually occur within 24 hours after contact with antigen (drug or dental material))
2. Consist of:
    a. Erythema
    b. Rash
    c. Ulceration
3. Treatment requires
    a. Topical steroids or systemic steroids (in severe cases)
    b. Identification and avoidance/elimination of any further contact the antigen

Examples

1- Some cases of aphthous stomatitis

2- Allergic dermatitis to orthodontic appliances (rare); in patient with nickel hypersensitivity and a history of cutaneous/skin piercing.
3- Erythema multiforme
   a- immune complex reaction
   b- Polymorphous eruption of macules, erosions, and characteristic "target" lesions that are symmetrically distributed on the skin and/or mucosa.

   c- Predisposing factor; drug allergy, herpes simplex infection, **Sulfa** antibiotics, Sulfonyl urea hypoglycemic agents (Rx of diabetes).

   d- Treatment; (symptomatic)
      1- bland mouth rinse or syrup of diphenhydramine, and triamcinolone acetonide (Kenalog) in Orabase.
      2- Severe involvement → systemic steroids.
      3- Causative drug should be withdrawn

III. Type IV Hypersensitivity. (Contact stomatitis)
1- **Delayed** allergic reaction → Contacts with allergic materials (dental materials, toothpaste, mouth rinses, lipsticks, face powders etc.) may have occurred **days before** the lesions appeared.
2- Dental materials (*agents*) can be involved in this type of reaction;
   a- Impression materials containing aromatic sulfonate in postmenopausal women
   b- Amalgam restorations → mucosal whitish, reddish, ulcerative, or "lichenoid" lesions, could be due to
      i- Hypersensitivity to the silver in amalgam → if restorations removed →lesions most often cleared.
      ii- Toxic injury to the mucosa
      iii- Hypersensitivity to mercury

   c- Dental composite (rare)
   d- The acrylic monomer in denture construction
   e- Gold and nickel → erythema and ulceration

3- Oral epimucous testing
   The dentist may wish to test agents that are thought to be possible antigens for contact stomatitis. This can be done in two phases;
   a- placing the suspected antigen in contact with the oral mucosa
   b- observing for any reaction over a period of days (Usually, 48 to 72 hours.)

- **Techniques** to conduct epimucous testing
a- Placing the suspected allergen in a rubber suction cup, placing the cup on the buccal mucosa, and observing at intervals for erythema or ulceration under the cup. Or;
b- Placing the suspected allergen in a depression on the palatal aspect of an overlay denture to holds the allergen in contact with the palatal mucosa. Or;
c- incorporating the allergen into Orabase → applied to the mucobuccal fold, and periodically observing for a reaction. Or;

d- Antigen can be incorporated into an oral adhesive spray.

4- **Management** of contact stomatitis
   a- removal of **sources** causing hypersensitivity reactions
   b- assessment for lesion **healing**.
   c- Avoid any **future** contact with the antigen.
   d- If the lesions **persist**, topical **steroids** can be applied.

B-Lichenoid Drug Eruptions.

1- lesions (identical to those of lichen planus) in patients that have taken certain drugs before appearance of the lesions.
2- e.g. of such drugs; levamisole (Levantine) and the quinidine drugs. Other agents; thiazide drugs, gold, mercury, methyldopa, phenothiazines, quinidine, and certain antibiotics.
3- **Biopsy** of the lesion show a microscopic picture similar to that of lichen planus + eosinophils in the subepithelial infiltrate →related to the cellular immune system → considered contact stomatitis
4- Management; drugs withdrawal → Lesions clear within several days (in most patients) or within a few weeks.

MANAGEMENT OF **SEVERE** TYPE I HYPERSENSITIVITY REACTIONS

Occur soon (within minutes) after exposure to antigen (injection, ingestion, or application of a topical aesthetic, medication, drug, local anaesthetic, or dental product). The dentist must take the following actions immediately (**initial procedures)**;

• Place the patient in a head-down or supine position.

• Make certain that the airway is patent.

• Administer oxygen.

• Be prepared to send for help **and** to support respiration and circulation. The vital signs should be noted. The dentist may administer aromatic spirits of ammonia through inhalation, which encourages breathing through reflex stimulation.

• If these initial steps have not solved the emergency problem, and the cause is highly likely to be allergic, an oedematous-type or anaphylactic reaction should be considered.

Angioedema. If an immediate type I hypersensitivity reaction has resulted in oedema of the tongue, pharyngeal tissues, or larynx, the dentist must take additional emergency steps to prevent death from respiratory failure. At this point, if the patient has not responded to the **initial procedures** and is in acute respiratory distress, the following should be done:

• Call for medical help.

• Inject 0.3 to 0.5 mL of 1: 1000 epinephrine by I.M. (into the tongue) or subcutaneous route.

• Supplement with intravenous diphenhydramine 50 to 100 mg if needed.

• Support respiration, if indicated, by mouth-to-mouth breathing or bag and mask; the dentist should make sure the chest moves when either of these methods is used.

• Check the carotid or femoral pulse; if a pulse cannot be detected, closed chest cardiac massage should be initiated.

• Transport to medical facility if needed.

Anaphylaxis. An anaphylactic reaction usually takes place within minutes but may take longer.

- **"Itching" of soft palate**
- **Nausea, vomiting**
- **Substernal pressure**
- **Shortness of breath**
- **Hypotension**
- **Pruritus**
- **Urticaria**
- **Laryngeal oedema**
- **Bronchospasm**
- **Cardiac arrhythmias**

Unlike severe oedematous reaction, in which respiratory distress occurs first, both respiratory and circulatory components of depression occur early in the anaphylactic reaction. Anaphylaxis often is fatal unless, immediate action is taken;

1. Call for medical help.

2. Place patient in the supine position.

3. Check for open airway.

4. Administer oxygen.

5. Check pulse, blood pressure, and respiration.

   a- If any of the vital signs is depressed or absent, inject 0.3 to 0.5 mL 1: 1000 epinephrine into the tongue (I.M).
   b- Provide cardiopulmonary resuscitation if needed.
   c- Repeat intramuscular injection of 0.5 mL 1: 1000 epinephrine if no response (every 5 minutes).

Note:

Intramuscular injection of epinephrine into the thigh has been reported to provide higher plasma concentrations than those administered into the arm.

Principles of Flaps, Suturing and Management of difficult extraction

Oral surgery skills can be learned through the knowledge of the basic principles of surgery, knowledge of the anatomy of the region and good practical training. Whenever surgical intervention is considered, the operator must decide if the procedure is necessary weighing its benefits and its risks, and the patient must be made aware of the other possible non-surgical methods of treatment for the given problem. Also all the short-term and long-term complications of the surgical procedure must be explained to the patient in relation to the known risks.

The main prerequisite for an operator performing surgical procedures is to ensure **Asepsis and Antisepsis,** to prevent pathogenic microbes from entering the body as well as spread of certain infectious diseases from one patient to another. This is accomplished through;

- Sterilization of instruments involving dry heat, moist heat (autoclave) and chemical means of sterilization.
- Preparation of the patient by seating the patient on the dental chair, disinfecting the skin around the mouth and the oral mucosa and covering the patient with sterile drapes.
- Preparation of the operator by disinfecting the hands and wearing the appropriate sterile gown and surgical gloves.

Flap is simply defined as a section of soft tissue that is outlined by surgical incisions, carries its own blood supply, allows surgical access to underlying tissues, can be replaced as required on its original position, maintained with sutures and is expected to heal. Most of the oral surgical procedures require the reflection of a full mucoperiostial flap incorporating mucosa, submucosa and periosteum to gain access to the area that is the object of surgery.

Incision

It is simply defined as a cut or wound made by cutting with a sharp instrument. The basic principles of incisions in oral surgery include:

- ❖ A blade number 15 is suitable for most oral surgical procedures. Sometimes a blade number 12 is used.
- ❖ A new and sterile blade should be used for each patient and it should be replaced with a new one intraoperatively if its cutting edge becomes blunted when necessary.
- ❖ The scalpel blade is mounted on the scalpel-handle with the help of a needle holder, or hemostat, with which it slides into the slotted receiver with the beveled end parallel to that of the handle.
- ❖ The scalpel is grasped in a pen grasp for maximum control and tactile sensitivity.

- ❖ The incision should be made at right angle to the underlying bone to ensure good healing when the tissues are re-apposed.

- ❖ The scalpel should move at uniform speed and with sufficient firmness to cut through not only the mucosal surface but also the periosteum overlying the bone. It should be made, ideally, with a single movement, repeated strokes at the same place should be avoided as they may impair healing.

Flap design

The essential points that should be considered include:

- ➢ Flap design and incision should be carried out in such a way that injury of anatomic structures is avoided, such as: the mental neurovascular bundle, palatal vessels emerging from the greater palatine foramen and incisive foramen, lingual nerve, submandibular duct, facial artery and vein. So thorough knowledge of the anatomy of the orofacial region is essential.>

- ➢ The base of the flap should be wider than its apex (free gingival margin) to ensure adequate blood supply for better healing.

- ➢ The flap should be of adequate width for good visualization and accessibility of the operative field without subjecting the flap to tension and trauma during manipulation.

- ➢ When planning the flap, the care should be given to the fact that the flap should be wider than the anticipated bony defect after completion of the procedure so that the flap margins, when sutured, should rest on intact and healthy bone to prevent wound dehiscence and poor healing.

- ➢ Delicate handling of the flap during the surgical procedure without excessive tension of crushing in order not to compromise the blood supply which leads to delayed healing.

- ➢ Vertical releasing incisions should start at the buccal vestibule and end at the interdental papilla which should either be excluded or included in the flap, the incision should always pass to the interdental papilla and not end at the labial or buccal surface of the tooth to ensure the integrity of the gingiva, but it should not pass through the papilla for accurate replacement of the flap.

Vertical releasing incision are contraindicated in certain sites in the oral cavity:

- ✓ Transverse incisions in the palate: to avoid injury to the greater palatine artery.
- ✓ Lingual surface of the mandible: to avoid injury to the lingual nerve.
- ✓ Canine eminence: because it increases the tension on the suture line which lead to wound dehiscence.
- ✓ In the area of mental foramen, between mandibular first and second premolars: to avoid injury to the mental nerve.

Types of Mucoperiosteal Flaps

❖ Envelope Flaps

This type of flaps is made by a horizontal incision through gingival sulcus for the teeth or through the alveolar mucosa of the edentulous area with no vertical releasing incisions. The envelope flap is used for surgery of incisors, premolars and molars, on the labial or buccal and palatal or lingual surfaces. The **main indications** of this type of flaps include: surgical extraction of impacted mandibular third molars, palatal approach to impacted maxillary canines or removal of mandibular tori.

The **main advantages** of this flap are; easy re-approximation to original position, good blood supply and it can easily modified to two-sided or three-sided flap by adding vertical releasing incisions to either ends of the flap when necessary.

Disadvantages of this flap are the limited accessibility and visualization, difficulty in reflection with greater tension that can result in tearing at the ends of the flap, in addition to defect in attached gingival and the possibility of injury to the greater palatine artery during reflection of palatal flap.

❖ Two-sided Flap (Triangular Flap)

This flap is the made with a horizontal incision along the gingival sulcus or alveolar ridge mucosa and a vertical releasing incision. The vertical incision begins approximately at the vestibular fold and extends to the interdental papilla of the gingiva. This flap is performed labially or buccally on both jaws and is **indicated in** the surgical removal of root tips, impacted teeth, small cysts, and apicectomies.

Advantages are; it ensures an adequate blood supply, satisfactory visualization and accessibility, good re-approximation; it can be easily modified to a three-sided flap, or even lengthening of the horizontal incision.
Disadvantages are; limited access, tension when flap is retracted and it may result in defect of attached gingiva.

❖ Three-sided Flap (Trapezoidal Flap)

This flap consists of a horizontal incision along the gingival or alveolar ridge mucosa and 2 vertical releasing incisions, this flap is **indicated** when an extensive surgical field exposure is required especially when two-sided flap is inadequate.
The main **advantages** include; very good accessibility and visualization of the surgical field with minimal tension on the tissue, and good reapproximation of tissue to the original position.
The **disadvantages** are the possibility of producing an attached gingival defect. This flap cannot be lengthened or modified once reflected.

❖ Semilunar Flap

This flap is the result of a curved incision, which begins just beneath the vestibular fold and has a bow shaped course with the convex part towards the attached gingiva. The lowest point of the incision must be at least 0.5 cm from the gingival margin, so that the blood supply is not compromised. Each end of the incision must extend at least one tooth over on each side of the area of bone removal. The semilunar flap is used in apicoectomies and removal of small cysts and root tips.
Advantages of this flap are small incision, easy reflection, no attached gingival defect especially around prosthetic appliances (crowns and bridges) and easy oral hygiene.

Disadvantages of this flap are limited accessibility and visualization of the surgical field, re-approximation may be difficult due to the absence of reference points, tendency to tear due to excessive tension on reflection and the possibility that the flap may made over defective bone as a result of inadequate planning or underestimation of the size of the bony defect so that the margins of the flap will not rest on intact bone leading to collapse of the flap and wound dehiscence.

❖ Other types of flaps

- A flap with a **Y-shaped incision**. This flap used in surgical procedures of the palate, mainly for removal of exostoses (torus palatinus). The first flap consists of an incision along the midline of the palate with 2 anterolateral incisions made anterior to the canines, additional posterolateral incisions can be added to improve accessibility indicated in large tori, but care should be taken not to sever the greater palatine vessels. The **major disadvantage** of these flaps is that they can easily tear as the mucosa overlying palatine tori can be fairly thin.

- Flaps that are used for closure of oroantral fistula or communication include; **Buccal Advancement Flap** which is in essence a three-sided flap that after reflection the periosteum is transversely incised so that the flap remains pedicled only by the mucosa allowing it to be advanced and sutured to the palatal tissues. The other flap is the **Palatal Transpositional Flap** that incorporates the greater palatine vessel, it is rotated and sutured to the buccal tissues.

Flap reflection

The mucoperiosteal flap is reflected from the underlying bone using periosteal elevators. There are many any types of mucoperiosteal elevators like Howarth, Ash, the no.9 Molt, Seldin, or Freer types. The elevators should be firmly pushed at approximately 30-45° to the surface of the bone such that the periosteum is stripped from it. It is important to try to raise both mucosa and periosteum in one layer and this requires a considerable force to be applied.

Reflection of the flap begins at the papilla; the periosteal elevator is pushed underneath the papilla in the area of the incision and is turned laterally to pry the papilla away from the underlying bone. This technique is used along the entire extent of the free gingival incision. If it is difficult to elevate the tissue at any one spot, the incision is probably incomplete, and that area should be re-incised. A dry, sterile swab can be interposed between the periosteal elevator and the bone.

The elevator may also be used for holding the flap after reflecting, facilitating manipulations during the surgical procedure.

Oftentimes two elevators can be used to advantage one working and the other aiding retraction in the subperiosteal plane. Adequate undermining of the wound margins is required in order to mobilize the flap. Generous reflection is the key to adequate vision, and wide exposure reduces traction trauma to the wound edges.

Suturing

After completion of the surgical procedure, thorough irrigation of the surgical field using sterile normal saline follows. Then the flap is repositioned to its original position and held in place using sutures to protect the underlying tissues from infection and irritating factors and prevent postoperative hemorrhage. Sutures are also used to repair soft tissue lacerations, ligation of vessels and control of bleeding, immobilization of flaps in their new position, and stabilization of drains in place. Suture diameters vary from **0.02 to 0.8 mm.** This corresponds to **10/0 to 5** on the British Pharmacopoeia (BP) system. The finest suture that will hold the wound secure, without it breaking should be chosen. The amount of suture material used should be kept to a minimum, particularly when braided, to reduce bacterial colonization. Suture material can be a nidus for infection, and knots can be the focus of a persistent and chronic inflammatory reaction (suture knot sinus).

Suture Materials

Suture materials are classified as either **absorbable** or **non-absorbable** material depending on whether the body tissues will degrade the suture material and absorb it over time. Absorption takes place either by Hydrolysis or by proteolytic enzymatic degradation depending on the material used. They can also be classified as **monofilament** or **multifilament.**

❖ Absorbable Sutures

They are used in suturing of deep layers of wounds when multilayered suturing is required, they are also used in children, mentally handicapped patients and in patients who cannot return to the clinic to have their sutures removed. They can cause inflammatory tissue reaction that can impede tissue healing. Some of the popular absorbable sutures include:

❖ **Plain Catgut:** it is made from collagen derived from healthy sheep or cattle intestine, its tensile strength is lost within 7-10 days, its absorption is through phagocytosis and enzymatic degradation which occurs within 7-10 days producing high tissue reaction. It is used for suturing subcutaneous tissues that do not require prolonged support. **It is not suitable for suturing in oral surgery.**

❖ **Chromic Catgut:** it is made from collagen derived from healthy sheep or cattle intestine tanned with Chromium salts to facilitate handling and resist tissue degradation. It tensile strength is lost within18-2ldays, its absorption is like that of the plain Catgut but it takes longer time and with moderate tissue reaction. It has the same indication as for the plain Catgut and it is **not suitable in oral surgery.**

❖ **Polyglactin (Vicryl)**

Synthetic suture made of copolymer of lactide and glycolide coated with polyglactin and calcium stearate.

It is **braided multifilament** suture, 60% of its tensile strength remains for 2 weeks, and about 30% for 3 weeks.

Its absorption is through hydrolysis with complete absorption taking place within 60-90 days, it induces mild tissue reaction. This suture is widely used in surgical practice but it is not advised for use where prolonged approximation under tension is required.

❖ **Polydioxanone (PDS)**

Supplied as **monofilament** dyed or undyed, it is made of polyester polymer, 70% of its tensile strength remains at 2 weeks, 50% at 4 weeks and 14% at 8 weeks. Absorption occurs through hydrolysis which is complete in about 180 days, it is used when slight longer wound support is required.

❖

❖ Non-absorbable sutures

These sutures remain in the tissues and are not absorbed, but have to be cut and removed about 7 days after their placement. Commonly used sutures include:

➢ **Silk:** it is made of raw silk from silkworms, and it is supplied as braided or twisted, dyed or undyed, coated with wax or silicon or uncoated. 80%-100% of its tensile strength is lost within 6 months. Fibrous encapsulation occurs in the body within 2-3 weeks, it causes moderate to high tissue reaction. It is used in ligation and suturing when long term tissue support is needed. Silk sutures are the easiest to use and the most economical, and have a satisfactory ability to make a secure knot.

➢ **Nylon**

it is made of polyamide polymer and it is supplied usually as monofilament. It loses 15%-20% of its tensile strength per year. It causes mild tissue reaction and it is used mainly for skin, in plastic surgery, neurosurgery, and ophthalmic surgery. Know it is more widely used in oral surgery.

One of the most commonly used suture for the oral cavity is 3/0 black silk. The size 3/0 has the appropriate amount of strength; the multifilament nature of the silk makes it easy to tie and well tolerated by the patient's soft tissues. The color makes the suture easy to be seen when the patient returns for suture removal. However, because of the multiple filaments, they tend to "wick" oral fluids along the suture to the underlying tissues. This wicking action may carry bacteria along with the saliva. Sutures that are holding mucosa together usually stay no longer than 5 to 7 days, so the wicking action is of little clinical significance.

Needles

Needles are usually made of stainless steel which is strong and flexible material. There are different shapes, sizes and cross sections of needles. Needles of 18-26 mm in length are suitable for use in oral surgery.

There are two basic needle types: Those that have the hole at the suture side of the needle and that need to be threaded with suture are "**eyed.**" Conversely, those that have the suture attached to the needle are "**eyeless**" or "**swaged.**" The advantages of the swaged needles include:

- The eyeless needle is composed of a single use needle and suture. This avoids the loss of sharpness that occurs with reusable needles.
- There is only a single strand of suture that is pulled through the tissues, and the gap that is created by the needle is fully plugged by the suture. This reduces potential leakage through the suture line.
- There is no re-threading of an eyeless needle, and its use is more time efficient.

As compared to a regular circle, needles are either; 1/4 circle, $^1/_2$ circle, ¾ circle, 3/8 circle, or $^1/_8$ circle or they can have different shapes like straight needles, J needles, or compound curve needles.

According to the cross section of the needles, there are:

- ✓ **Needles with round or oval cross section** which are considered atraumatic and are mainly used for suturing thin mucosa. Their disadvantage is that great pressure is required when passing through the tissues, which may make suturing the wound harder. They are used in oral surgery especially in areas of thin mucosa they are also used in suturing of peritoneum, bowel, muscles and fat.
- ✓ **Needles with triangular cross section;** these are either cutting or reverse cutting needles. The difference is that in addition to the two cutting edges of the triangle, cutting needles have a third cutting edge on the inside of the curvature while the reverse cutting needles the third cutting edge is on the outer convex curvature of the needle. These designs allow minimal soft tissue trauma during needle insertion as they cut a path through the soft tissues and do not therefore require excessive force on the part of the operator.

The passage of a needle through tissue should follow its curvature. This minimizes tissue damage and the appropriate size and shape of cutting, or round-bodied atraumatic needle, needs to be chosen for the least traumatic passage through tissue.

Instrument for Suturing includes:

- **Needle Holder**

These instruments come in a variety of sizes and design. In general, they have a locking handles allowing the needle to be locked into the beaks of the instrument. They resemble Hemostats but with few differences:

✓ The beaks of the hemostat are longer and thinner than that of the needle holder.

✓ The internal surface of the short beaks of the needle holder is grooved and crosshatched, permitting a firm and stable grasp of the needle, while the short beaks of the hemostat have parallel grooves which are perpendicular to the long axis of the instrument.

- **Tissue Forceps**

Sometimes known as dissecting forceps, the important requirement is that they hold the soft tissues atraumatically so avoiding crushing and with little chance of slippage. This is achieved by a toothed design in the form of a wedge-shaped projection or tooth on one side, and a receptor on the other, which fit into each other when the handles are locked, although possibly causing tiny puncture points, is ideal for the purposes of suturing and holding soft tissues generally. The use of non-toothed forceps will result in crushing of the tissues as, to prevent tissue slippage from grasp, the instrument must be held too tightly.

- **Suture Scissor**

Principles of suturing

❖ Suturing should be undertaken using a no-touch technique to reduce the risk of a needle-stick injury and the fewer the number of sutures used to produce the desired result, the better. Insertion of too many sutures tears the tissue unnecessarily, and the resulting tangle of suture thread tends to accumulate plaque and promote inflammation.

❖ Before the sutures are inserted the non-flap side of the incision should be undermined to facilitate the insertion of the needle.

❖ When re-approximating the flap, the suture is passed first through the mobile (usually facial) tissue, the needle is re-grasped with the needle holder and is passed through the attached tissue of the lingual papilla. But if the two margins of the wound are close together, the surgeon may be able to insert the needle through both sides of the wound in a single pass. However, for better precision it is better to use two passes in most situations.

- The tissue of the flap should be held firmly by the tissue forceps and the needle passed through the mucoperiosteum about 3 mm from the margin, more if the flap is friable because of chronic infection. The needle is then pushed through the corresponding tissue on the other side of the incision, again about 3-5 mm from the margin. The needle should enter the surface of mucosa at right angle, and the passage of the needle should follow it curvature to prevent tearing of the flap.

- After the needle passes through both wound edges, the suture is pulled, so that the needle-bearing end is longer. Afterwards, the long end of the suture is wrapped around the handle of the needle holder twice. The short end of the suture is grasped by the needle holder and pulled through the loops. The suture is then tightened by way of its two ends, thus creating the first double-wrapped knot. Then a single-wrap knot is created, in the counterclockwise direction, which is named a safety knot.
- Where possible, the knots should be drawn to lie to one or other side of the line of incision. Over-tightening of the suture, manifested by blanching of tissue, must also be avoided, it runs the risk of tissue necrosis and wound dehiscence. Overlapping of wound edges when positioning the knot should also be avoided.

- Sutures placed intraorally are normally removed 5-7 days postoperatively. In the removal of sutures, normal dental tweezers should grasp the free ends of the thread and the suture should be cut by sharp scissors. The suture should then be pulled though in its entirety. The suture is better cut just as it enters the tissue to avoid pulling a contaminated suture through the tissue.

Suturing Techniques

■ Simple Interrupted Suture:

This is the simplest and most frequently used type, and may be used in all surgical procedures of the mouth. The needle enters from the margin of the flap (mobile tissue) and exits at the same distance on the opposite side. The two ends of the suture are then tied in a knot. The advantage of the interrupted suture is that it is simple to execute and when sutures are placed in a row, inadvertent loosening of one or even losing one will not influence the rest.

■ Continuous Suture:

This is usually used for the suturing of wounds that are long, e.g., for re-contouring of the alveolar ridge in the maxilla and mandible. This technique for the **continuous simple (or nonlocking) suture** is as follows: after passing the needle through both flap margins, an initial knot is made just as in the interrupted suture but only the free end of the suture is cut off. The needle-bearing suture is then used to create successive continuous sutures at the wound margins. The last suture is not tightened, but the loop created actually serves as the free end of the suture that is used to tie the knot.

The **continuous locking suture** is a variation of the continuous simple suture. This type of suture is created exactly as described above, except that the needle passes through every loop before passing through the tissues, which secures the suture after tightening. Suturing continues with the creation of such loops, which make up parts of a chain along the incision. These loops are positioned on the buccal side of the wound, after being tightened.

The **advantage** of the continuous suture is that it is quicker and requires fewer knots, so that the wound margins are not tightened too much, thus avoiding the risk of ischemia of the area. Its **disadvantage** is that if the suture is inadvertently cut or loosened, the entire suture becomes loose.

■ **Mattress Suture:**

This is a special type of suture and is described as horizontal and vertical. It is indicated in cases where strong and secure re-approximation of wound margins is required. The main indication for use of vertical mattress sutures is to evert the skin edges, the technique permits greater closure strength and better distribution of wound tension. The horizontal suture also allows eversion of the wound edges and is used in cases which require limiting or closure of soft tissues over osseous cavities, e.g., post-extraction tooth sockets. In the mattress suture the needle passes through the wound margins at a right angle, and the needle always enters and exits the tissues on the same side.

■ **Figure of Eight suturing**

Figure-of-eight sutur occasionally placed over top of socket to aid in hemostasis, it is usually performed to help in maintaining a piece of oxidized cellulose in tooth socket after tooth extraction.

Management of difficult extraction

Difficulties may be encountered during extraction of teeth for a variety of reasons and any oral surgeon should never ignore any warning of the possibility that such difficulties may be encountered during any proposed tooth extraction especially when the patient indicates that such difficulties have been experienced in previous occasions. These difficulties can be diagnosed through clinical examination and a good radiograph that shows the root morphology of the tooth and the surrounding and supporting structures which should be taken before extraction whenever possible.

The main indications for surgical extraction of teeth are:

- ✓ **Retained roots and root tips**.
- ✓ Teeth with **root morphology that is unfavorable** for simple tooth extraction, such as teeth with large bulbous roots due to hypercementosis or those with dilacerated roots or root tips.
- ✓ Teeth that have crowns with **extensive caries**, especially root caries, or that have large amalgam restorations.

- ✓ Teeth with **ankylosed roots**.
- ✓ Teeth that are **fused with adjacent** teeth or roots.
- ✓ Maxillary posterior teeth that are **closely associated with maxillary sinus** due to pneumatization of the sinus into the alveolar process or when there is an increased risk of fracture of maxillary tuberosity during simple extraction.
- ✓ Fully or partially **impacted teeth**.
- ✓ Teeth or retained roots with **periapical lesions** whose removal in entirety is not possible through curettage alone after simple extraction.

Retained roots and root tips that are deeply buried in the alveolus and are asymptomatic are not indicated for surgical extraction especially in older individuals with poor general health, or when there is a risk of serious local complications or damage to vital structures like the inferior alveolar nerve, lingual nerve or maxillary sinus.

Steps of surgical extraction

Surgical extraction of teeth is preceded by proper preparation and disinfection of the patient, and administration of local anesthesia then the surgical procedure proceeds as follows:

1. Creation of a **full mucoperiosteal flap**, this could be envelope, two-sided or three-sided flap based on the anticipated difficulty and the need for better accessibility.
2. For **single rooted teeth** and after reflecting the flap the operator may attempt to re-seat the forceps under direct visualization for better mechanical advantage. The other options include; grasping a bit of buccal bone under the buccal beak of the forceps so that a small piece of buccal bone is removed with the tooth, application of an elevator, or removal of sufficient amount of bone to facilitate the application of the forceps or the elevator. In some cases, a purchase point is made in the root where the elevator is applied and the root extracted.
3. **Bone removal** using surgical hand piece and round burs to expose an adequate part of the tooth or root. Whenever possible the oral surgeon should be conservative by removing bone to allow the creation of a point of application for the elevator for luxation or sometimes removing only a small window of bone overlying the broken apex of roots to allow their retrieval through the socket.
4. In **multi-rooted teeth** sectioning of the crown of the tooth and/or sectioning the roots so that they can be extracted as single rooted teeth.
6. After the tooth and all the root fragments have been removed, the flap is repositioned and the surgical area is palpated for **sharp bony edges**. If any sharp edges are present, they are smoothed with a bone file or a handpiece and bur. The wound is thoroughly irrigated and debrided of loose fragments of tooth, bone, calculus, and other debris.
7. The **flap is repositioned again and sutured** in the usual fashion.

Indications for leaving root fragments

When a root tip has fractured and approaches of removal have been unsuccessful, and when the open surgical approach may be excessively traumatic, the surgeon may consider leaving the root in place. As with any surgical approach, the surgeon must balance the benefits against the risks of surgery. In some situations, the risks of removing a small root tip may outweigh the benefits.

The conditions that must exist for a tooth root to be left in the alveolar process are:

- The root fragment must be **small**, usually no more than 4 to 5 mm in length.
- The **root must be deeply embedded** in bone and not superficial, to prevent subsequent bone resorption from exposing the tooth root and interfering with any prosthesis that will be constructed over the edentulous area.
- The tooth involved must **not be infected**, and there must be no radiolucency around the root apex. This lessens the likelihood that subsequent infections will result from leaving the root in position.
- The root should **not be mobile.**
- If the surgeon elects to leave a root tip in place the **patient must be informed** that, in the surgeon's judgment, leaving the root in its position will do less harm than surgery. In addition, radiographic documentation of the presence and position of the root tip must be obtained and retained in the patient's record, the patient should be recalled for follow-ups to track the fate of this root.
- The **patient should be instructed** to contact the surgeon immediately should any problems develop in the area of the retained root.

Multiple Extractions

If multiple adjacent teeth are t o b e extracted at a single session the surgeon should determine if there is need for interim partial immediate dentures, any type of soft tissue surgery, such as tuberosity reduction or the removal of undercuts or tori in critical areas. If dental implants are tobe placed at a later time, it may also be desirable to limit bone trimming and socket compression. In some situations, dental implants may be placed at the same time as the teeth are removed, which would require the preparation of a surgical guide stent to assist in aligning the implants appropriately.

Extraction sequencing

Maxillary teeth should usually be removed first for several reasons:

- ❖ Infiltration anesthetic has a more rapid onset and also disappears more rapidly. This means that the surgeon can begin the surgical procedure sooner after the injections have been given.
- ❖ Surgery should not be delayed because profound anesthesia is lost more quickly in the maxilla.
- ❖ During the extraction process, debris such as portions of amalgams, fractured crowns, and bone chips may fall into the empty sockets of the lower teeth if the lower surgery is performed first.
- ❖ Maxillary teeth are removed with a major component of buccal force. Little or no vertical traction force is used in removal of these teeth, as is commonly required with mandibular teeth.

A single minor disadvantage for extracting maxillary teeth first is that if hemorrhage is not controlled in the maxilla before mandibular teeth are extracted, the hemorrhage may interfere with visualization during mandibular surgery but this is usually not a major problem because hemostasis should be achieved in one area before the surgeon turns attention to another area of surgery, and the surgical assistant should be able to keep the surgical field free from blood with adequate suction.

Posterior teeth are extracted first, this allows for the more effective use of dental elevators and forceps to extract the teeth.

After extraction the buccolingual plates are pressed with firm pressure and the soft tissues are repositioned, sharp spicules of bone should be removed and smoothed with bone nibbler (Rongeur) and bone file, the area should be thoroughly irrigated with normal saline and the papillae in position.

Dental Implantology

5th year **Dr. Auday M. Al-Anee**

Basic Implant Surgical Procedures

Surgical procedures always start with detailed surgical preparation. Preparation for implant surgery requires a thorough review of the patient's chart, including medical and dental histories, operatory notes, radiographs, anticipated implant sizes and locations, surgical guides, surgical sequencing and strategy, possible complications, patient management, anesthesia, operating time, instrumentation, postoperative management, and restorative plan. Preoperative antibiotic prophylaxis is sometimes recommended. An oral dose of 2 g amoxicillin 1 hour preoperatively or, in patients unable to take oral medications, cefazolin 1 g or ampicillin 2 g intramuscularly or intravenously 1 hour before the dental procedure are effective. Alternative medications include 600 mg of clindamycin orally or intravenously. No postoperative antibiotic administration is necessary.

Once the patient has been draped in a sterile fashion and the surgical team has been gloved and gowned, the patient is anesthetized. In many cases, the implants can be placed using local anesthetic block or infiltration techniques. However, in more complex and lengthy procedures, some type of sedation or general anesthesia may be preferred. Local anesthetics containing vasoconstrictors are usually used for hemostasis. Additional long-acting anesthetics for postoperative pain control may be warranted. It is imperative to have good access to the operative site via effective retraction of cheeks and the tongue. A mouth prop is invaluable.

The surgical site should be kept aseptic, and the patient should be appropriately prepared and draped for an intraoral surgical procedure. Pre-rinsing with chlorhexidine gluconate for 1 to 2 minutes immediately before the procedure will aid in reducing the bacterial load present around the surgical site. Every effort should be made to maintain a sterile surgical field and to avoid contamination of the implant surface. Implant sites should be prepared using gentle, atraumatic surgical techniques with an effort to avoid overheating the bone.

Successful osseointegration occurs predictably for submerged and non-submerged dental implants when proven clinical guidelines are followed. Well-controlled studies of patients with good plaque control and appropriate occlusal forces have demonstrated that root form, endosseous dental implants show little change in bone height around the implant over years of function. After initial bone remodeling in

the first year (1 to 1.5 mm of resorption described as “normal remodeling around an externally hexed implant”), the bone level around healthy functioning implants remains stable for many years afterward. The average annual crestal bone loss after the first year in function is expected to be 0.1 mm or less. Hence, implants offer a predictable solution for tooth replacement.

Regardless of the surgical approach, the implant must be placed in healthy bone with good primary stability to achieve osseointegration, and an atraumatic technique must be followed to avoid damage to bone. Drilling of the bone without adequate cooling generates excessive heat, which injures bone and increases the risk of failure. The anatomic features of bone quality (dense compact versus loose trabecular) at the recipient site influences the interface between bone and implant. Compact bone offers a much greater surface area for bone-to-implant contact than cancellous bone. Areas of the jaw exhibiting thin layers of cortical bone and large cancellous spaces, such as the posterior maxilla, have lower success rates than areas of dense bone. The best results are achieved when the bone-to-implant contact is intimate at the time of implant placement.

One-Stage versus Two-Stage Implant Placement Surgery

Currently, most threaded endosseous implants can be placed using either a one-stage (nonsubmerged) or a two-stage (submerged) protocol. In the one-stage approach, the implant or the abutment emerges through the mucoperiosteum/ gingival tissue at the time of implant placement, whereas in the two-stage approach, the top of the implant and cover screw are completely covered with the flap closure. Implants are allowed to heal, without loading or micromovement, for a period of time to allow for osseointegration. In two-stage implant surgery, the implant must be surgically exposed following a healing period. Some implants, referred to as “tissue level,” are specifically designed with the coronal portion of the implant positioned above the crest of bone and extending through the gingival tissues at the time of placement in a one-stage protocol. Other implant systems, referred to as “bone level,” are designed to be placed at the level of bone and require a healing abutment to be attached to the implant at the time of placement to be used in a one-stage approach.

A one-stage surgical approach simplifies the procedure because a second-stage exposure surgery is not necessary. The two-stage, submerged approach is advantageous for situations that require simultaneous bone augmentation procedures at the time of implant placement because membranes can be submerged, which will minimize postoperative exposure. Mucogingival tissues can be augmented if desired at the second-stage surgery in a two-stage protocol or as part of the one-stage protocol.

Implant Site Exposure

Exposure of the implant site can be accomplished in several ways, including flapless surgery or with tissue elevation that may include sulcular, midcrestal, and vertical releasing incisions. Flapless surgery may be indicated when there is adequate keratinized tissue over an ideal ridge form. This creates the least soft tissue trauma and may provide the best postoperative esthetics in patients with excellent presurgical anatomy and papilla shape. In flapless surgery, the implant and the healing or provisional restoration are placed in a single stage.

When a flap is required, the incision should be designed to allow convenient retraction of soft tissue for unimpeded access for implant placement. This is usually necessary when better access and visualization of the underlying bone is necessary and when additional procedures such as bone or soft tissue grafting are done at the time of implant placement.

• Midcrestal incision: The incision should be made through the keratinized tissue, being sure to place the blade up against the mesial-distal surfaces of the teeth adjacent to the edentulous space. In areas with a narrow zone of keratinized tissue, the incision can be made slightly to the palatal or buccal aspect to allow for keratinized tissue transfer to the buccal or facial aspect and better soft tissue closure. If sulcular incisions are necessary, great care is taken to follow the contour of the sulcus so as not to damage the soft tissue architecture.

• Vertical releasing incision: Using a sharp no. 15 blade, a curvilinear, beveled (~45 degrees), papilla sparring incision should be made to reduce or eliminate incision scarring. It must be ensured that the vertical releasing incision is extended apically enough to allow complete release of the flap.

Implant Placement

Flap Reflection

• Reflection at the papilla is initiated with a periosteal or elevator, using gentle, well-directed, controlled pressure. The periosteal elevator's edge can be used in a "light painting stroke" to cleanly release the subperiosteal fibers. At this point, the flap is developed from the papilla up along the vertical release.

• The dissection is then directed along the sulcular tissue to the point where it meets the crestal portion of the incision. The index finger of the opposing hand supporting the facial aspect of the ridge allows greater control and protection of the flap during reflection.

• The reflection is continued by the elevation sulcularly to the distal extent of the incision.

• Once the buccal flap is reflected, the palatal or lingual flap can be reflected enough to visualize the width of the ridge. Any soft tissue tags should be carefully removed.

• When the buccal flap has been reflected completely, a retractor can be positioned against the bone inside the flap. This allows good visualization of the operative site while protecting the integrity of the flap. It is extremely important to avoid inadvertent trauma to the flap with the tip if the retractors.

Preparing the Osteotomy

The surgeon must confirm that the handpiece and motor are functioning properly: the speed setting on the motor should be checked; it must be confirmed that the drill is spinning in the forward mode. The speed should be set to the appropriate speed as recommended by the manufacturer of the implant system being used.

• All drills, including osteotomy drills, should be copiously irrigated internally, externally, or both when preparing the bone.

• The depth indicator markings on the precision and pilot drills should always be reviewed.

• The entry point and its ideal angulation should be determined with the precision drill. The proper angulation should be verified from different vantage points. A surgical guide is usually used to facilitate orientation.

• Drilling is done with the precision drill at full speed to a depth of 1 to 2 mm short of the depth of the intended implant (e.g., 8 mm deep for a 10-mm implant).

• The area is irrigated and the 2-mm pilot drill positioned in the exact same location after verifying the correct angulation. Once position and angulation are confirmed, the 2-mm pilot drill is run at full speed to the intended depth of the implant (e.g., 10 mm deep for a 10-mm implant).

• The area is rinsed, and the guide pin that corresponds to the intended final size of the planned implant is placed. Use of the guide pin allows the surgeon to evaluate the position, spacing, and angulation of the developing osteotomy. It also helps evaluate where the pin lines up against the opposing dentition.

• The surgeon then determines the location on the twist drill that corresponds to the intended platform position of the implant to the ridge. Typically, the top of the platform would be even with the mesial and distal bone height.

• The tip of the narrowest twist drill is placed into the pilot hole, and the correct position and angulation of the drill are verified. Once confirmed, the drill is run at full speed in a gentle pumping motion. It may be necessary to remove the drill and clean the accumulated bone off the drill. The osteotomy is rinsed, and the drill is then repositioned and the angulation confirmed. The drill is again run at full speed and taken to the final depth of the intended implant. The site is sequentially prepared in this manner.

• The osteotomy is rinsed, and the appropriate guide pin is placed to reevaluate position and alignment.

• The tip of this final twist drill is placed into the opening of the osteotomy; then its position and angulation are verified. Great care is taken to achieve perfect position and angulation, as this is the drill that finalizes the osteotomy.

• Once the drill is properly positioned, it is run at full speed in a gentle pumping motion to the final depth of the intended implant. The osteotomy is then inspected with a thin instrument for possible bone perforation (e.g., sinus communication or buccal wall perforation).

• Immediately after completing the osteotomy, the speed of the motor is changed to the desired and/or recommended torque, measured in newton centimeters (Ncm—typically around 30 Ncm) for the insertion of the implant. If the speed is not changed and the implant is put in at the original setting of 800 to 1500 rpm, the osteotomy could easily be damaged, the implant seated too deep, or primary stability lost.

Inserting Implant

• The implant is opened and placed on the driver that has been inserted into the handpiece. The handpiece must be held such that the tip of the implant is pointing up. This will lessen the likelihood of the implant falling off the driver.

• The tip of the implant is inserted into the osteotomy, and the position and angulation are verified again. The implant is driven into position by keeping light pressure in an apical direction until the implant is almost completely seated or until the motor torques out (approximately 1 to 2 mm short of complete seating).

• Using the hand torque wrench, the surgeon continues to seat the implant, using the torque lever of the wrench to quantify the amount of torque present. If the torque exceeds the lever, the implant is hand torqued to its final position by using the handle of the torque wrench.

• The seating of the implant is finalized by verifying that the platform is even with the mesial and distal heights of bone and that any orientation marker is pointed in the correct position.

• The area is irrigated thoroughly.

• It should be determined if there will be a single- or two-stage healing period. This is determined by the torque value measured on the surgical motor or the hand torque wrench. An implant with a torque value of 35 Ncm or greater is considered

to have good primary stability, and single-stage healing is possible. If so, an appropriate-sized healing abutment is placed. If a two-stage process is required, then an appropriate-sized cover screw is placed.

• The abutment should protrude 1 to 2 mm through the tissue. A tapered abutment rather than a parallel abutment must be determined. The intended tissue emergence of the planned restoration helps determine whether the healing abutment is tapered or parallel.

• The healing abutment is placed onto the insertion wrench, again by holding the screw pointing up. The abutment is screwed into the implant and tightened with finger pressure, making sure that no tissue is caught under the abutment.

Suturing Flap

- The flap is sutured using some type of resorbable suture (chromic gut or Vicryl) or nonresorbable suture (proline).
- The anterior papilla is secured first. The buccal aspect of the papilla is entered with the suture needle, which is passed through the embrasure to engage the palatal tissue. The needle is then positioned lower on the palatal tissue and penetrated and brought through the embrasure to the buccal and the papilla engaged apically to the first entry point.
- The vertical release is then sutured, followed by the mesial and distal sides of the abutment. These are simple interrupted sutures tied in the same fashion as the first suture described.

Postoperative Management

A radiograph should be taken postoperatively to evaluate the position of the implant in relation to adjacent structures such as the sinus and the inferior alveolar canal and relative to teeth and other implants. This radiograph also serves to verify the complete seating of the cover screw or healing abutment.

Patients should be given analgesics. Mild to moderate strength analgesics are usually sufficient. Antibiotics are often given prophylactically before surgery but are usually not required in the postoperative period. Patients may also be instructed

to use 0.12% chlorhexidine gluconate rinses for 2 weeks after surgery to help keep bacterial populations at a minimum during healing. The patient is evaluated weekly until soft tissue wound healing is complete (approximately 2 to 3 weeks). If the patient wears a tissue-borne denture over the area of implant placement, the denture can be relined with a soft liner after 1 week. Interim partial dentures or orthodontic retainers with an attached pontic may be worn immediately but must be contoured to avoid soft tissue loading over the implant site.

Uncovering

The healing time or the length of time necessary to achieve osseointegration varies from site to site and from patient to patient. Insertion torque values, quality of bone, bone grafts, patient health, location, number of implants, and soft tissue health all have an impact on healing time. Typical healing times are 4 to 6 months. In single-stage surgery, no surgical uncovering is necessary. The implant stays exposed via the healing abutment after surgery and throughout the healing phase. After an appropriate integration time, restoration of the implant can proceed.

In a two-stage system, the implant must be surgically uncovered and a healing abutment placed. The goals of surgical uncovering are to attach the healing abutment to the implant, preserve keratinized tissue, and modify the form or thickness of tissue. A soft tissue healing period after uncovering must be allowed before restoration of the implant can take place, typically 2 to 4 weeks.

The simplest method of surgical uncovering is the “tissue punch”. This method of uncovering utilizes a soft tissue punch equal to or slightly larger than the diameter of the implant placed. The implant is palpated through the tissue to determine its location. The tissue punch is placed directly over the implant circumference and twisted through the soft tissue thickness, taking care not to damage the bone at the level of the implant platform. The punch is then removed, along with a precisely determined piece of tissue that was lying directly above the implant, easily exposing the implant cover screw. The cover screw is then removed, and an appropriate sized and appropriate-shaped healing abutment is placed. The advantage to this technique is that it is less traumatic, no periosteum needs to be reflected, and only a short soft tissue healing time is required. This technique does, however, require an adequate zone of keratinized tissue so that the implant can be

accurately located. Disadvantages to this technique include sacrifice of a portion of the keratinized tissue, inability to visualize the bone surrounding the implant, and the inability to directly visualize the precise abutment–implant interface.

If the implants cannot be accurately located, if the clinician needs to visualize underlying bone, or if a slight keratinized tissue transfer is indicated, then a crestal incision with the creation of a slight soft tissue flap is required to uncover the implants. If an adequate zone of keratinized tissue is present, the soft tissue flap can be contoured with a scalpel, scissors, or a punch to conform to the shape of the healing abutment. This allows for a nicely shaped and contoured soft tissue cuff around the healing abutment and eventually the final implant restoration. Obvious advantages to this technique include easy access, minimal invasiveness, and ability to directly visualize the bone surrounding the implant and to precisely fit the healing abutment to the implant platform. The disadvantage to reflecting a flap during uncovering is the possibility of bone loss due to stripping the periosteum from bone during the uncovering. Advanced techniques for cases with an inadequate zone of attached tissue include tissue transfer procedures, tissue grafting, and split-thickness apically repositioned flaps.

Implant Stability

Initial implant stability is one of the most important predictors of long-term implant success. This depends on the depth and density of bone, implant size, and precision of the surgical technique. A good sense of implant stability can be obtained during the seating process and by verifying adequate torque resistance capability of the seated implant.

Radiofrequency analysis has been used to measure and verify implant stability. This technology involves attaching a transducer to an implant and applying a steady-state resonance frequency to the implant. The advantage of this technology is that it is not dependent on measuring implant movement in just one direction but rather by evaluating the complete bone-implant interface.

Complications

Implant placement surgery can be performed with great accuracy and with little complication if the case has been diagnosed, planned, and surgically performed well. However, as with any surgical or clinical procedures, complications are possible and include the following:

• Complications that can occur with any surgical procedure, including pain, bleeding, swelling, or infection.

• A positioning error resulting in implants placed at a compromised angulation or position. The implant may be placed too close to an adjacent tooth root or too far to the mesial, distal, or buccal aspect, thus compromising bony support. The implant can be placed too far into bone, making prosthetic access difficult. If the implant is not placed deep enough into bone, leaving threads of the implant body above the osseous crest, there will be compromise to bony support, soft tissue health, hygiene, and esthetics.

• Surgical technique complications such as a tear of the soft tissue flap, poor closure of the incision, or excessive soft tissue trauma from retraction may result in tissue dehiscence, infection, and eventual loss of the implant. Poor attention to detail in preparation of the osteotomy such as overdrilling the diameter of the osteotomy could result in poor prognosis for integration.

• Invasion of critical anatomic structures can create more serious complications. If the implant invades or impinges on the canal of the IAN, this may result in paresthesia (altered sensation that the patient does not find painful, e.g., numbness, tingling), or dysethesia (altered sensation that the patient finds painful or uncomfortable). If the implant invades the maxillary sinus or the nasal cavity, this may result in an infection. Bone structure compromise can present as overthinning of the buccal or facial plate or dehiscence or fenestration of overlying tissue. Bone perforation can occur at the inferior border of the mandible because of inaccurate drilling depth or on the lingual aspect of the posterior mandible because of the lingual undercut from poor positioning or angulation of the implant drills.

• Mechanical complications can present as an implant platform fracture because of excessive insertion torque. If the osteotomy is improperly prepared in dense bone,

it is possible to get the implant "stuck" in bone, short of complete seating, making it extremely difficult to retrieve the implant.

• Incision line opening can occur from inadequate suturing or not having tension-free closure.

• Esthetic complications can occur from poor implant positioning or angulation, making proper prosthetic restoration unrealistic.

Implant Components

❖ Implant Body or Fixture

The implant body, or fixture, is the implant component placed within bone during the first stage of surgery. Most contemporary implant fixtures are referred to as root form implants, taking the form of a cylinder or a tapered cylinder, and are made of titanium or titanium alloy. Most current implant fixtures have an external threaded design, although historically, there have been smooth-surfaced implants that were pressed into position. A wide variety of external thread designs and different surface textures and coatings that attempt to maximize implant stability and the process of osseointegration have been offered by manufacturers. Most implant fixtures incorporate an antirotational design feature at the interface of the adjoining prosthetic components. This antirotational feature may be located internally or externally to the implant platform.

❖ Cover or Healing Screw

After placement of the implant fixture in a two-stage surgical approach, prior to suturing, the implant fixture is sealed at its platform with a low profile, intra-implant cover screw. It is important that the surgeon be sure that the cover screw is fully seated on the implant platform prior to suturing the flap to prevent bone from growing between the screw and the implant. In the second-stage uncovering procedure, the cover screw is removed and replaced with a healing abutment.

❖ Healing or Interim Abutment

Healing abutments are dome-shaped intra-implant screws that provide permucosal access to the implant platform. Healing abutments are placed at the completion of the implant placement surgery in a one-stage surgical approach or after uncovering in a two-stage surgical approach. Healing abutments are made of titanium or titanium alloy. The abutments can be parallel walled or tapered and range in height from 2 to 10 mm. The height of the abutment used is determined by the thickness of tissue present. The healing abutment should project 1 to 2 mm superior to the height of the gingival tissue. A tapered healing abutment is used to help shape soft tissue to a more appropriate emergence for the planned restoration (e.g., a crown). A parallel-walled abutment would be used where the tapered emergence is not necessary (e.g., a retentive bar for an overdenture). It is important to allow for sufficient healing of soft tissue after placing the healing abutment prior to making any impressions for the final prosthetics.

- Impression Coping

Impression copings facilitate transfer of the intraoral location of the implant to the same position on the laboratory cast. Impression copings can be either screwed into the implant body or screwed or snapped onto an implant abutment.

Typically, the impression transfer can be either closed-tray transfer or open-tray transfer. The closed-tray technique captures the index of the impression coping, and after the impression is removed from the mouth, the impression coping is unscrewed from the implant and placed along with an implant analog back into the impression. An open-tray transfer uses a specific impression coping that is designed to emerge through the impression tray. When the impression is ready to be removed from the mouth, the impression coping is unscrewed and pulled out in the impression. The open-tray method is considered the more accurate transfer method and is indicated when large-span frameworks or bar structures are planned or when the implants are too divergent to easily remove the impression tray in the closed-tray technique. A heavier-bodied polyvinyl siloxane or polyether impression material is recommended. Prior to making the transfer impression, it is imperative that the clinician take a radiograph to confirm that the impression coping is accurately seated on the implant platform. If the impression coping is not properly seated, the accuracy of the transferred location of the implant will be incorrect. On

completion of the transfer impression, an implant analog is screwed onto the impression coping to allow the fabrication of a laboratory cast.

- Implant Analog or Replica

Implant analogues are manufactured to replicate exactly the top of the implant fixture (fixture analog) or abutment (abutment analog) in the laboratory cast. Both are screwed directly into the impression coping. The impression coping or analog component is then placed back into the impression (closed-tray transfer) or is maintained in the impression (open-tray transfer), and the impression is ready to be poured. It is tremendously beneficial to create a soft tissue moulage in the impression prior to pouring. The soft tissue moulage is an elastomeric product that simulates the soft tissue portion on the dental cast. This allows the laboratory technician to have an accurate and flexible representation of soft tissue. The laboratory technician then has a working model that can be used to fabricate either the abutment or the framework for the intended prosthetic design.

- Implant Abutment

The abutment is the portion of the implant that supports or retains a prosthesis or implant superstructure. A superstructure is defined as a metal or zirconia framework that attaches to either the implant platform or the implant abutment(s) and provides retention for a removable prosthesis (e.g., a cast or milled bar retaining an overdenture with attachments) or the framework for a fixed prosthesis. Abutments are described by the method in which the prosthesis or superstructure is retained to the abutment. Abutments can be divided into three main categories: (1) screw retained, (2) cement retained, and (3) prefabricated attachment abutments. A screw-retained abutment uses a screw to retain the prosthesis or superstructure, whereas a cement-retained abutment uses cement to retain the prosthesis or superstructure. A prefabricated attachment abutment (e.g., locator or O-ring attachments) helps retain a removable prosthesis.

- Prosthesis Retaining Screw

Prosthesis retaining screws are intended to attach prosthetic abutments, screw-retained crowns, or frameworks to the implant fixture or implant abutment. The screws are generally made of titanium, titanium alloy, or gold alloy and are sized specific to the type, size, and design of the implant or abutment system. The screws typically have a hex or square design to accept a specific size and shape of wrench or driver. Most prosthesis screws are tightened to specific tolerance by a torque wrench or handpiece. The torque value is measured in newton centimeters and typically ranges from 10 to 40 Ncm.

Defining implant outcomes

Some implant outcomes are reported as the presence or absence of the implant at the time of the last examination, regardless of whether the implant was functional, suffered from bone loss, or had other problems. This type of assessment is a measure of **implant survival** and should not be confused with implant success. In contrast to such an overly simplified assessment, some investigators report implant outcomes using specific criteria to determine implant success.

Implant success is defined by specific criteria used to evaluate the condition and function of the implant. Criteria for implant success have been proposed in the literature but have not been used consistently. The problem is that a universally accepted definition of implant success has not been established. In the classic definition, Albrektsson and colleagues defined success as an implant with no pain, no mobility, no radiolucent peri-implant areas, and less than 0.2 mm of bone loss annually after the first year of loading.

Implants that are osseointegrated but not functional are referred to as **sleepers** and should not be considered successful merely because they are present and osseointegrated.

Aesthetic Results and Patient Satisfaction

The ultimate goal of treatment is to achieve natural-appearing, optimally functioning, implant-supported tooth replacements. Proper tooth dimensions and contours, and ideal soft tissue support are key factors for successful aesthetic outcomes. If crown form, dimension, and shape and gingival harmony around the implants are not ideal, the patient may consider the implant restoration

unacceptable, because the result does not represent a natural dental profile. For some patients, such as those with severe alveolar deficiency, an ideal aesthetic outcome may be impossible because reconstructive surgical procedures are complex, require extensive time, and remain unpredictable. For others, a less-than ideal aesthetic outcome may be acceptable.

Aesthetic problems and dissatisfaction happen when results do not match a patient's expectations. Satisfaction with the aesthetic outcome of implant prosthesis varies among patients. The risk of failure is greater among those with high aesthetic demands and risk factors such as a high smile line, thin periodontal soft tissues, or compromised bone support.

Good Luck

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Dental Implantology

5th year **Dr. Auday M. Al-Anee**

Implant Treatment: Basic Concepts and Techniques

Dental implants have given the profession and the patient an extremely predictable and effective means of tooth replacement. The partially edentulous patient can now undergo replacement of a single tooth or several missing teeth with implant retained crowns and enjoy the function and esthetics they had with their natural teeth. The completely edentulous patient no longer has to live with compromised function and the reduced confidence that traditional full denture wearers have historically experienced. Dental implants can offer the edentulous patient comfort, function, and confidence with either fixed prosthetics or implant-retained removable prosthetic options.

The history of modern implant dentistry began with the introduction of titanium implants. In the 1950s, Per-Ingvar Brånemark, a Swedish professor of anatomy, had a serendipitous finding while studying blood circulation in bone that became a historical breakthrough in medicine. He coined the phenomenon osseointegration and developed an implant system with a specific protocol to achieve it predictably. The first patient was successfully treated in 1965.

Implant Geometry (Macrodesign)

Numerous implant systems with various geometric (macrodesign) designs have been developed and used before the current implant systems in use today. Previous implant designs included blade vents (narrow, flat shape; tapped into bony trough prepared with rotary burs), press-fit cylindrical (bullet shape; pressed or tapped into prepared hole), subperiosteal (custom-made framework; adapted to the surface of jawbone), and transmandibular (long rods or posts; placed through the anterior mandible).Some of these implant systems were initially stable and appeared to be successful over short-term periods (e.g., 5 years) but failed to remain stable, became symptomatic or loose, and failed over longer periods. Lacking predictability, these implant systems are no longer used. Since the time of the Brånemark studies, millions of patients have been treated worldwide using variations of these techniques with implants of different geometries and surface characteristics. Similar research including

that of André Schroeder in Switzerland in the mid-1970s contributed to the success of endosseous dental implants.

The serendipitous finding of Brånemark was that when a hole is prepared into bone without overheating or otherwise traumatizing the tissues, an inserted biocompatible implantable device would predictably achieve an intimate bone apposition, as long as micromovements at the interface were prevented during the early healing period.

Currently, most endosseous implants have a cylindrical or tapered, screwshaped/ threaded design. The disastrous results with other implant configurations were largely responsible for the evolution toward the current popular designs.

The most common implant design being used today is the screw-shaped or threaded cylindrical implant. A threaded implant design is preferred because it engages bone well and is able to achieve good primary stabilization. Even systems that started with cylindrical press-fit (nonthreaded) designs progressively evolved to a threaded geometry. The (longitudinal) shape of implants may be parallel or tapered. Although a majority of all implants have been parallel walled, the use of a tapered implant design has been advocated because it requires less space in the apical region (i.e., better for placement between roots or in narrow anatomic areas with labial concavities). Tapered implants have also been advocated for use in extraction sockets.

Implant Surface Characteristics (Microdesign)

Implant surface characteristics (microtopography) have been shown to positively influence the healing process. Accordingly, modification of implant surface characteristics has been a major area of research interest and development. Modifications in surface energy, chemical composition, and surface topography are known to influence cellular activity and tissue responses, leading to enhanced osteogenesis. At the molecular level, modified implant surfaces increase adsorption of serum proteins, mineral ions, and cytokines, which subsequently promote cellular migration and attachment. Implant surface characteristics can also aid in the retention of a fibrin clot, thus providing a migratory pathway for the differentiating osteogenic cells to reach the implant surface. Today, implants are treated

with a variety of technologies to modify surface characteristics (microscale or nanoscale) to enhance bone formation.

Additive Processes

The additive process modifies the microstructure/macrostructure and chemical nature of the implant surface by adding materials or chemicals to the existing surface. Several methods are used to add materials or chemicals to the implant surface, such as inorganic mineral coatings, plasma spraying, biocoating with growth factors, fluoride, and particulates or cements containing calcium phosphates, sulfates, or carbonates. The addition of materials, such as hydroxyapatite, to the implant surface has been shown to enhance or accelerate the initial bone cells, adaptation or proliferation. In general, additive surface modifications tend to increase the surface texture greater than subtractive surface modifications, resulting in topographically “rougher” implant. Surface roughness can also be increased by oxidizing or adding an oxide layer.

Subtractive Processes

The subtractive process modifies the microstructure and chemical nature of the implant surface by removing or altering the existing surface. The roughness of implant surface can be modified by machining, acid etching, blasting, or a combination of these processes to enhance the amount or speed of osseointegration. Implant surfaces that are modified at the microscopic level with techniques such as acid etching are thought to promote favorable cellular responses and increased bone formation in close proximity to the surface.

Implant Surface Chemical Composition

There have been unsuccessful trials with oral implants made of carbon or hydroxyapatite. The lack of resistance, because of material properties, to occlusal forces led to frequent fractures. The so-called noble metals or alloys, however, do not resist corrosion and have thus been abandoned. Today, the majority of oral implants are made of commercially pure (CP) titanium or titanium alloys. Titanium is a reactive metal that oxidizes within nanoseconds when exposed to air. Because of this passive oxide layer, the titanium then becomes resistant to corrosion in its CP form. Some alloys, such as titanium-aluminum 6%, vanadium 4% (Ti6Al4V), are known to

provoke bone resorption as the result of leakage of some toxic components. The oxide layer of CP titanium reaches 10 nm of thickness. It grows over the years when facing a bioliquid. It consists mainly of titanium dioxide (TiO2).

Hard Tissue Interface

The primary goal in implant placement is to achieve and maintain an intimate bone-to-implant connection. This concept is known as osseointegration. Histologically defined, osseointegration is the direct structural and functional connection between organized, living bone and the surface of a load-bearing implant without intervening soft tissue between the implant and bone. Osseointegration clinically is defined as the asymptomatic rigid fixation of an alloplastic material (the implant) in bone with the ability to withstand occlusal forces.

The osseointegration process observed after implant insertion can be compared with bone fracture healing. Implant site osteotomy preparation (bone wounding) initiates a sequence of events, including an inflammatory reaction, bone resorption, release of growth factors, and attraction by chemotaxis of osteoprogenitor cells to the site. Differentiation of osteoprogenitor cells into osteoblasts leads to bone formation at the implant surface. Extracellular matrix proteins, such as osteocalcin, modulate apatite crystal growth. Specific conditions, optimal for bone formation, must be maintained at the healing site to achieve osseointegration.

For osseointegration to occur in a predictable fashion, several important factors are required:

1. A biocompatible material (the implant)

2. Atraumatic surgery to minimize tissue damage

3. Implant placement in intimate contact with bone

4. Immobility of the implant, relative to bone, during the healing phase

Titanium is the material of choice for dental implants. Titanium is biologically inert and therefore does not elicit a foreign body rejection reaction from host tissue. For the implant to have intimate contact with

bone, the implant site must be prepared with a precise technique. All implant systems have specially designed drills that are used in a specific sequence to remove bone as atraumatically as possible. The drill sizes are matched to the size and shape of the implant being placed, creating the precision necessary for developing initial bony contact and stability.

Atraumatic surgical technique in an aseptic environment is critical to minimize mechanical and thermal injuries to bone. This involves using sharp, precision osteotomy drills run at slow speed with high torque while maintaining gentle, intermittent pressure and providing copious irrigation. Irrigation can be accomplished either externally or internally using special handpieces and burrs with internal ports. The goal is to maintain bone temperatures below 47°C during implant site preparation. Any variance causing temperatures to exceed 47°C is likely to cause bone necrosis and failure of osseointegration.

Initial stability of the implant must be achieved and maintained for formation of bone at the implant surface. Stability at the time of placement is predicated on the volume and quality of bone that intimately contacts the implant as well as the length and diameter of the implant.

During the time required for osseointegration to occur, it is imperative that immobility of the implant be maintained. A mild inflammatory response enhances the bone healing, but moderate inflammation or movement above a certain threshold is detrimental. When micromovements at the interface exceed 150 μm, the movement will impair differentiation of osteoblasts and fibrous scar tissue will form between the bone and implant surface. Therefore it is important to avoid excessive forces, such as occlusal loading, during the early healing period.

New bone formation follows a specific sequence of events. Woven bone is quickly formed in the gap between the implant and the bone; it grows fast, up to 100 μm per day, and in all directions. Characterized by a random orientation of its collagen fibrils, high cellularity, and limited degree of mineralization, the biomechanical capacity of woven bone is poor. Thus any occlusal load should be well controlled or avoided in the early phase of healing.

After several months, woven bone is progressively replaced by lamellar bone with organized, parallel layers of collagen fibrils and dense

mineralization. Contrary to the fast-growing woven bone, lamellar bone formation occurs at a slow pace (only a few microns per day).

Clinically, both primary stability and secondary stability of an implant are critical to success. Primary stability, achieved at the time of surgical placement, depends on the implant geometry (macrodesign), as well as the quality and quantity of bone available for implant anchorage at a specific site. Studies using resonance frequency analysis (RFA) have reported decreased implant stability in the early weeks of post-insertion healing. Secondary stability, achieved over time with healing, depends on the implant surface (microdesign), as well as the quality and quantity of adjacent bone, which will determine the percentage of contacts between the implant and bone. For example, areas such as the anterior mandible have dense cortical bone and provide rigid primary stabilization and good support throughout the healing process. Conversely, areas such as the posterior maxilla have thin cortical bone, and large marrow spaces provide less primary stability. For this reason, the posterior maxilla has been associated with lower success rates compared with other sites with greater bone density and support.

Once osseointegration is achieved, implants can resist and function under the forces of occlusion for many years.

Soft Tissue–Implant Interface

Historically, most basic science and clinical efforts were spent on studying the bone-implant interface of osseointegration. Considerably less attention was given to overlying soft tissues. In contemporary implant dentistry, however, this subject is being researched with great zeal. Driven primarily by the need for satisfactory esthetics as well as maintenance of a soft tissue seal or barrier against bacterial invasion, soft tissue has become a major focus of interest.

It is critical to understand both the striking similarities and the obvious differences between the peri-implant soft tissue and periodontal soft tissue. Peri-implant and periodontal soft tissues do share a number of similarities and only subtle differences. Each emerges from alveolar bone through soft tissue. Soft tissue consists of connective tissue covered by epithelium, which is continuous with an epithelium-lined gingival sulcus, the apical-most portion being lined with junctional epithelium forming an attachment.

From that point down to the level of alveolar bone, both types of soft tissue possess a zone of dense connective tissue. This zone of supracrestal connective tissue is responsible for maintaining a stable interface between soft tissue and the implant and acts as a seal or barrier to the oral environment. It is the orientation of the connective tissue fibers adjacent to an implant that differ from a natural tooth. This zone of connective tissue has been measured to be 1 to 2 mm in height. Clinically this becomes important when examining the health of peri-implant soft tissue. Probing depths in a healthy implant would be approximately 1 to 2 mm less than the total measured dimension from the crest of the sulcus to the alveolar bone crest. The other obvious difference between teeth and implants is that teeth have a periodontal ligament with connective tissue fibers that suspend teeth in alveolar bone. The implant, however, is in direct contact with bone without any intervening soft tissue. This difference has a dramatic impact on the biomechanics, proprioception, and prosthetic consideration for implants versus natural teeth. Because an implant, unlike a tooth, does not have cementum, most connective tissue fibers run in a direction more or less parallel to the implant surface.

Questions emerged decades ago, as it did for the natural dentition, about the need for keratinized tissue to surround implants. Keratinized mucosa tends to be more firmly anchored by collagen fibers to the underlying periosteum than non-keratinized mucosa, which has more elastic fibers and tends to be movable relative to the underlying bone. In clinical studies evaluating intraoral implants, with or without peri-implant keratinized mucosa, no clinically significant difference in implant success was reported. However, when there is a lack of keratinized tissue, patients tend to complain about pain and discomfort while performing oral hygiene procedures or other functions in the area. The symptoms are alleviated by increasing the amount of keratinized (firmly bound) tissue around the implant(s) via soft tissue grafting.

Biomechanical Considerations

Once the implant is properly placed, the long-term success is heavily dependent on restorative biomechanical factors— that is, how the stresses imposed on the functioning implant or prosthetic unit or units will be controlled or distributed. The axiom is simple: The load-bearing capacity of the integrated implant has to be greater than the anticipated load during

function. If applied loads are greater than the load-bearing capacity, it is likely to lead to mechanical failure, biologic failure, or both. Mechanical failure may present simply as porcelain fracture or as a loosened or fractured prosthetic screw (the screw that attaches the abutment or framework to the implant). The most devastating mechanical failure occurs when the force is destructive enough to actually fracture the implant fixture. A biologic failure can occur when the functional load exceeds the load-bearing capacity of the implant-bone interface. This initially presents clinically as bone loss around the platform of the implant. If the loss is severe enough and the provocation is long enough, the bone loss may progress around the entire implant and result in complete failure of the implant. The clinician must remember that an implant-retained restoration lacks the “shock absorbing” periodontal ligament that a natural tooth-retained restoration possesses. The periodontal ligament allows slight physiologic movement of teeth, and in the absence of microbe-induced inflammation, natural teeth can move and adapt to the forces without pathologic bone loss. This, however, is not possible with an osseointegrated implant.

The load-bearing capacity of implants is qualified by several factors, including the number and size of the implants, the arrangement and angulation of the implants, and the volume and quality of the bone-implant interface. The same factors that maximize initial implant stability in hard tissue continue to be important. Thick cortical bone and dense trabecular bone surrounding a long, wide-diameter implant that is positioned to be in line with the functional load, would offer the greatest load-bearing capacity and the best prognosis for long-term success. Conversely, a short, narrow-diameter implant placed in an area of thin cortical bone and less dense trabecular bone and in an off-axis angulation would have far less load-bearing capacity and a poorer prognosis for success. The angulation of the implants as it relates to the occlusal plane and the direction of the occlusal forces is an important determinant in optimizing the translation of the forces to the implants and the surrounding bone. Loads directed through the long axis of the implants are tolerated very well. Slight off-axis loads are usually not clinically detrimental, but loads applied at angles greater than 20 degrees or more can result in load magnification and initiate bone loss at the implant-bone interface. Again, if excessive loads persist, bone loss will continue and will likely lead to implant failure.

The number of implants placed in multi-tooth edentulous spans affects the load-bearing capacity of the implanted prosthesis. If there is a three-tooth edentulous span, the fixed prosthetic options would be to place three implants with three splinted crowns, three implants with three single-unit crowns, two implants as terminal abutments for a three-unit fixed partial denture, or two adjacent implants with a fixed partial denture with a cantilevered pontic. The load-bearing capacity decreases with each successive option.

Straight-line or linear arrangement of multiple implants should be avoided as this provides the least biomechanical advantage and is the least resistant to torqueing forces caused by off-center occlusal and lateral loads. Implants should be placed in a more curvilinear or staggered fashion.

Connecting a single integrated implant to one natural tooth with a fixed partial denture will effectively create an excessively loaded cantilever situation. Because of the immobility of the implant compared with the mobility of the natural tooth, when the loads are applied to the fixed partial denture, the tooth can move within the limits of its periodontal ligament. This can create stresses at the implant abutment junction up to two times the applied load on the prosthesis. Additional problems with a tooth to implant-supported, fixed partial dentures include breakdown of osseointegration, cement failure on the natural abutment, screw or abutment loosening, and possible failure of the implanted prosthetic components.

Detrimental forces can be applied iatrogenically by placing non-passive, ill-fitting frameworks on implants. When the screws are tightened in an attempt to seat the ill-fitting framework, compressive forces are placed on the implant-bone interface. This excessive force can lead to bone loss and potential implant failure.

Preoperative Assessment and Treatment Planning

The ultimate goal of dental implant therapy is to satisfy the patient’s desire to replace one or more missing teeth in an esthetic, functional manner with long-term success. To achieve this goal, clinicians must accurately and comprehensively assess the dentoalveolar condition as well as the overall physical and mental well-being of the patient.

Chief Complaint

What is the problem or concern in the patient's own words? What is the patient's goal of treatment? How realistic are the patient's expectations? The patient's chief concern, desires for treatment, and vision of the successful outcome must be taken into consideration.

The patient will measure implant success according to his or her personal criteria. The overall comfort and function of the implant restoration are often the most important factors, but satisfaction with the appearance of the final restoration will also influence the patient's perception of success. Furthermore, patient satisfaction may be influenced simply by the impact that the treatment has on the patient's perceived quality of life. Patients will evaluate for themselves whether the treatment helped them to eat better, look better, or feel better about themselves.

The clinician could consider an implant and the retained prosthesis a success using standard criteria of symptom-free implant function, implant stability, and lack of peri-implant infection or bone loss. At the same time, however, the patient who does not like the aesthetic result or does not think the condition has improved could consider the treatment a failure. Therefore it is critical to inquire, as specifically as possible, about the patient's expectations before initiating implant therapy and to appreciate the patient's desires and values. With this goal in mind, it is often helpful and advisable to invite patients to bring their spouses or family members to the consultation and treatment-planning visits to add an independent “trusted” observer to the discussion of treatment options. Ultimately, it is the clinician's responsibility to determine if the patient has realistic expectations for the outcome of therapy and to educate the patient about realistic outcomes for each treatment option.

Medical History and Medical Risk Assessment

A thorough medical history is required and must be documented for every dental patient. As with any patient planning a surgical procedure, the patient must be assessed preoperatively to evaluate his or her ability to tolerate the proposed procedure, heal, and to have a favorable prognosis.

There are only a few absolute medical contraindications to implant therapy. Absolute contraindications to implant placement based on surgical and

anesthetic risks are limited primarily to patients who are acutely ill and those with uncontrolled metabolic disease. Often these contraindications are limited in duration; once the illness resolves or the metabolic disease is controlled, the patient may become a good candidate for implant therapy. Relative contraindications are concerned with medical conditions that affect bone metabolism or the patient's ability to heal. These include conditions such as diabetes, osteoporosis, immune compromise (e.g., human immunodeficiency virus infection, acquired immunodeficiency syndrome), medications (e.g., bisphosphonates—oral and intravenous), and medical treatments such as chemotherapy and irradiation (e.g., of the head and neck).

Some psychological or mental conditions could be considered absolute or relative contraindications, depending on their severity. Patients with psychiatric syndromes (e.g., schizophrenia, paranoia) or mental instabilities (e.g., neurosis, somatic symptom disorder), those who have mental impairment or are uncooperative, or those who have irrational fears, phobias, or unrealistic expectations may be poor candidates for implant treatment. Certain habits or behavioral considerations such as smoking, tobacco use, substance abuse (e.g., drugs and alcohol), and parafunctional habits (bruxing and clinching) must be scrutinized as potential contraindications as well. Smoking, in particular, has been documented as a significant risk factor resulting in decreased long-term stability and retention of implants.

Contraindications

Absolute contraindications to implant placement

- Acute illness
- Magnitude of defect/anomaly
- Uncontrolled metabolic disease
- Bone and/or soft tissue pathology/infection

Relative contraindications

- Diabetes
- Osteoporosis
- Parafunctional habits
- HIV
- AIDS
- Bisphosphonate usage—oral and intravenous
- Chemotherapy
- Irradiation of head and neck
- Behavioral, neurologic, psychosocial, psychiatric disorders

Dental History

A review of a patient's past dental experiences can be a valuable part of the overall evaluation. Does the patient report a history of recurrent or frequent abscesses, which may indicate a susceptibility to infections or diabetes? Does the patient have many restorations? How compliant has the patient been with previous dental recommendations? What are the patient's current oral hygiene practices?

The individual's previous experiences with surgery and prosthetics should be discussed. If a patient reports numerous problems and difficulties with past dental care, including a history of dissatisfaction with past treatment, the patient may have similar difficulties with implant therapy. It is essential to identify past problems and to elucidate any contributing factors. The clinician must also assess the patient's dental knowledge and understanding of the proposed treatment, as well as the patient's attitude and motivation toward implants.

Intraoral Examination

The oral examination is performed to assess the current health and condition of existing teeth, as well as to evaluate the condition of the oral hard and soft tissues. It is imperative that no pathologic conditions are present in any of the hard or soft tissues in the maxillofacial region. All oral lesions, especially infections, should be diagnosed and appropriately treated before implant therapy.

Additional criteria to consider include the patient's habits, level of oral hygiene, overall dental and periodontal health, occlusion, jaw relationship, temporomandibular joint condition, and ability to open wide.

After a thorough intraoral examination, the clinician can evaluate potential implant sites. All sites should be clinically evaluated to measure the available space in the bone for the placement of implants and in the dental space for prosthetic tooth replacement. The mesial-distal and buccal-lingual dimensions of edentulous spaces can be approximated with a periodontal probe or other measuring instrument. The orientation or tilt of adjacent teeth and their roots should be noted as well. There may be enough space. Conversely, there may be adequate space between roots, but the coronal aspects of the teeth may be too close for emergence and

restoration of the implant. If either of these conditions is discovered, orthodontic tooth movement may be indicated. Ultimately, edentulous areas need to be precisely measured using diagnostic study models and imaging techniques to determine whether space is available and whether adequate bone volume exists to replace missing teeth with implants and implant restorations.

How Much Space Is Required for Placement of One or More Implants?

Alveolar Bone

Assuming an implant is 4 mm in diameter and 10 mm long, the minimal width of the jawbone needs to be 6 to 7 mm, and the minimal height should be 10 mm (minimum of 12 mm in the posterior mandible, where an additional margin of safety is required over the mandibular nerve). This dimension is desired to maintain at least 1 to 1.5 mm of bone around all surfaces of the implant after preparation and placement.

Interdental Space

Edentulous spaces need to be measured to determine whether enough space exists for the placement and restoration with one or more implant crowns. The minimal mesial-distal space for an implant placed between two teeth is 7 mm. The minimal mesial-distal space required for the placement of two standard-diameter implants (4-mm diameter) between teeth is 14 mm. The required minimal dimensions for wide-diameter or narrow-diameter implants will increase or decrease incrementally according to the size of the implant. For example, the minimal space needed for the placement of an implant 6 mm in diameter is 9 mm (7 mm + 2 mm).

Whenever the available space between teeth is greater than 7 mm and less than 14 mm, only one implant, such as placement of a wide-diameter implant, should be considered. Two narrow diameter implants could be positioned in a space that is 12 mm. However, the smaller implant may be more vulnerable to implant fracture.

Interocclusal Space

The restoration consists of the abutment, the abutment screw, and the crown (it may also include a screw to secure the crown to the abutment if it is not cemented). This restorative “stack” is the total of all the components

used to attach the crown to the implant. The dimensions of the restorative stack vary slightly depending on the type of abutment and the implant-restorative interface (i.e., internal or external connection). The minimum amount of interocclusal space required for the restorative “stack” on an external hex-type implant is 7 mm.

Diagnostic Casts and Photographs

Mounted study models as well as intraoral and extraoral photographs complete the records collection process. Study models and photographs are often overlooked in preoperative history taking, but both contribute significantly to the assessment and treatment planning phases of implant dentistry.

Study models mounted on a semi-adjustable articulator using a face-bow transfer give the clinician a three-dimensional working representation of the patient and provide much information required for surgical and prosthetic treatment planning.

Elements that can be evaluated from accurately mounted models include the following:

1. Occlusal relationships
2. Arch relationships
3. Inter-arch space
4. Arch form, anatomy, and symmetry
5. Preexisting occlusal scheme
6. Curve of Wilson and curve of Spee
7. Number and position of the existing natural teeth
8. Tooth morphology
9. Wear facets
10. Edentulous ridge relationships to adjacent teeth and opposing arches
11. Measurements for planning future implant locations

12. Visualizing force vectors, both present and planned

Medicolegally, the mounted study models are preserved as an exact reference of the preoperative condition.

Intraoral photographs are equally important. They allow visual evaluation of the patient's soft tissue (e.g., quantity, quality, location, texture, color, symmetry). Extraoral photographs provide views of the patient from many different esthetic perspectives. Elements that are easily assessed are as follows:

1. Facial form

2. Facial symmetry

3. Patient's degree of expression and animation

4. Patient's appearance (e.g., facial features, facial hair, complexion, eye color)

5. Smile line

6. Incisal edge or tooth display

7. Buccal corridor display

8. Potential esthetic demand

Hard Tissue Evaluation

The amount of available bone is the next criterion to evaluate. Wide variations in jaw anatomy are encountered, and it is therefore important to analyze the anatomy of the dentoalveolar region of interest both clinically and radiographically.

A visual examination can immediately identify deficient areas, whereas other areas that appear to have good ridge width will require further evaluation. Clinical examination of the jawbone consists of palpation to feel for anatomic defects and variations in the jaw anatomy, such as concavities and undercuts. If desired, it is possible with local anesthesia to probe through the soft tissue (intraoral bone mapping) to assess the thickness of the soft tissues and measure the bone dimensions at the proposed surgical site.

The spatial relationship of the bone must be evaluated in a three dimensional view because the implant must be placed in the appropriate position relative to the prosthesis. It is possible that an adequate dimension of bone is available in the anticipated implant site, but that the bone and thus the implant placement might be located too lingual or too buccal for the desired prosthetic tooth replacement. Bone augmentation procedures may be necessary to facilitate the placement of an implant in an acceptable prosthetic position despite the availability of an adequate quantity of bone (i.e., the bone is in the wrong location).

Soft Tissue Evaluation

Evaluation of the quality, quantity, and location of soft tissue present in the anticipated implant site helps to anticipate the type of tissue that will surround the implant(s) after treatment is completed (keratinized vs. nonkeratinized mucosa). For some cases, clinical evaluation may reveal a need for soft tissue augmentation. Areas with minimal or no keratinized mucosa may be augmented with gingival or connective tissue grafts. Other soft tissue concerns, such as frenum attachments that pull on the gingival margin, should be thoroughly evaluated as well.

Debate continues about whether it is necessary to have a zone of keratinized tissue surrounding implants. Despite strong opinions and beliefs about the need for keratinized mucosa around implants versus this mucosa being unnecessary, neither argument has been proved.

Some studies have concluded that, in the presence of good oral hygiene, a lack of keratinized tissue does not impair the health or function of implants. Others strongly believe that keratinized mucosa has better functional and aesthetic results for implant restorations. Keratinized mucosa is typically thicker and denser than alveolar mucosa (nonkeratinized). It forms a strong seal around the implant with a cuff of circular (parallel) fibers around the implant, abutment, or restoration that is resistant to retracting with mastication forces and oral hygiene procedures.

Radiographic Examination

Several radiographic imaging options are available for diagnosis and for planning of dental implantation. Options range from standard intraoral projections (e.g., periapical, occlusal) and extraoral projections (e.g.,

panoramic, cephalometric), to more complex cross-sectional imaging (e.g., computed tomography [CT], conebeam computed tomography [CBCT]).

Multiple factors, however, influence the selection of radiographic techniques for any particular case. Such factors as cost, availability, radiation exposure, and the type of case must be weighed against the accuracy of identifying vital anatomic structures within a given bone volume and being able to perform the surgical placement without injury to these structures. Areas of study radiographically include the following:

1. Location of vital structures

• Mandibular canal

• Anterior loop of the mandibular canal

• Anterior extension of the mandibular canal

• Mental foramen

• Maxillary sinus (floor, septations, and anterior wall)

• Nasal cavity

• Incisive foramen

2. Bone height

3. Root proximity and angulation of existing teeth

4. Evaluation of cortical bone

5. Bone density and trabeculation

6. Pathology (e.g., abscess, cyst, tumor)

7. Existence of anatomic variants (e.g., incomplete healing of extraction site)

8. Cross-sectional topography and angulation (best determined by using CT and CBCT)

9. Sinus health (best evaluated by using CT and CBCT)

10. Skeletal classification (best evaluated with the use of lateral cephalometric images)

Radiographic images allow for quantifying dimensions or for taking measurements. Traditional radiographs must be calibrated for potential magnification. Magnification on a traditional panoramic image can be as much as 25%. One way to determine magnification is to place a metal sphere near the plane of occlusion when taking the radiograph. By comparing the radiographic size with the actual size of the sphere, the magnification can be determined. Digitally acquired periapical, panoramic, lateral cephalometric images and CT and CBCT scans have bundled software applications that allow for very accurate measurement.

Critical measurements specific to implant placement include the following:

• At least 1 mm inferior to the floor of the maxillary and nasal sinuses

• Incisive canal (maxillary midline implant placement) to be avoided

• 5 mm anterior to the mental foramen

• 2 mm superior to the mandibular canal

• 3 mm from adjacent implants

• 1.5 mm from roots of adjacent teeth

CT and CBCT image data files can be reformatted and viewed on personal computers using simulation software. This allows the diagnosis and treatment planning processes to be more accurate with regard to measurements and dimensions. Critical anatomic structures can be visualized in all three coordinate axes so that their superoinferior, anteroposterior, and buccolingual locations can be identified.

Key Fact

The American Academy of Oral and Maxillofacial Radiology recommends panoramic radiography as the initial evaluation of the dental implant patient, supplemented with periapical radiographs as needed.

Surgical Treatment Planning Considerations

Surgical treatment planning takes the diagnostic data that have been gathered and combines them with the surgeon's clinical judgment to determine the potential surgical options. The surgeon must be mindful of the proposed prosthetic goals, typically driven by the number of implants required in suggested locations for a specific prosthetic design. Because implant dentistry is often a team endeavor, it is advantageous for the surgeon to have a reasonable understanding of the prosthetics and for the restoring dentist to have an understanding of the surgical aspects of implant placement.

After evaluating all of the previously described information, the surgeon must determine the prognosis of implant placement based on specific limitations as a result of anatomic variations, bone quality, and bone quantity in different areas of the jaw. The anterior mandible is usually tall enough and wide enough to accommodate implant placement. Bone quality is usually excellent, typically the densest of any area in the two arches. Primary surgical concerns in this area include proper angulation of the implants and avoiding the mental foramen and mandibular canal. Implants should be placed at least 5 mm anterior to the most anterior portion of the mental foramen, avoiding the anterior loop of the mandibular canal.

The posterior mandible limits the length of the implants based on the position of the mandibular canal that traverses the body of the mandible in this region. Ideally, the tip of the implant should be at least 2 mm from the inferior alveolar nerve (IAN). It is important to consider the buccolingual position of the nerve as well. The width of the posterior mandible must also be considered. If the nerve is located very near the buccal cortex, a longer implant could be placed, with the implant extending lingual to the IAN, even though the implant extends vertically past the nerve. CT or CBCT can be helpful in making this determination. The mandibular canal also precludes any posterior implants from engaging the inferior cortical plate, which could lessen the initial primary stability of the implant. The attachment of the mylohyoid muscle helps maintain the bony width along the superior aspect of the ridge, although this can often be deceiving because a deep lingual depression, "the lingual undercut," usually is present immediately below this attachment. This is a critical area to be examined and palpated during the clinical examination.

In planning the implant placement, if primary stability is questionable, increased time for osseointegration may be considered. The clinician may also want to consider “over-engineering” the case by using more implants (e.g., three implants replacing three teeth, vs. two implants replacing three teeth).

The posterior maxilla poses two specific concerns related to implant placement. The first is the quality of bone in this area. As previously discussed, bone quality in the posterior maxilla is typically the poorest of any area, limited by thin cortical bone at the ridge crest and the least dense trabecular bone. This often results in less implant stability at the time of placement. For this reason, more time (6 months or longer) may be required for osseointegration to occur in this region. The second concern is the proximity of the maxillary sinus to the edentulous ridge. Often, as a result of bone resorption and increased pneumatization of the sinus, a limited height of bone remains for implant placement. If an adequate height of bone is present, the implant should be placed, leaving 1 mm of bone between the sinus and the implant. If there is inadequate bone height, then either a “sinus bump” or “sinus lift” procedure would be necessary to augment the height of bone.

The anterior maxilla, even though it is the most surgically assessable area, may be one of the most difficult regions for implant placement. This area, even when healthy teeth are present, usually has a thin buccal plate. After tooth loss, the resorption of the ridge follows a pattern of moving apically and palatally, only exacerbating an already tenuous anatomy. The residual ridge anatomy results in a ridge that is narrow and angulated such that ideal implant positioning may be impossible and the esthetic outcome may be compromised. The nasal cavity and the incisive canal are vital structures that also define the anatomic limitations of anterior implant placement. Implants should be placed 1 mm short of the nasal floor and should not be placed in the maxillary midline.

Final Treatment Planning

The final stage of treatment planning involves consolidating all of the clinical and radiographic information in combination with surgical options and limitations to produce the best final result of the prosthetic treatment. The positioning and angulation of implant placement is critical to the

biomechanical stability and esthetics required for long-term success. To facilitate ideal implant placement, surgical guides are frequently utilized. The surgical guide template is a critical factor for implants placed in an esthetically important area because even slight variations of angulation can have large effects on the appearance of the final restoration. The construction of the surgical guide template is nearly indispensable in patients for whom it is necessary to optimize implant placement to ensure correct emergence profiles in the anterior esthetic zone. The four objectives of using a surgical template for the partially edentulous patient are as follows: (1) delineating the embrasure, (2) locating the implant within the tooth contour, (3) aligning the implants with the long axis of the completed restoration, and (4) identifying the level of cementoenamel junction or tooth emergence from soft tissue. This template can be constructed by using a diagnostic wax-up over the preoperative cast to construct a clear resin template with a guide hole. This provides the surgeon ease of access to bone and uninterrupted visual confirmation of frontal and sagittal positions and angulation. Although underlying bone may dictate some minor variation, the surgeon must stay as close as possible to the template during implant placement. With the aid of computer technology, accurate “virtual” treatment planning can be accomplished. CBCT data are used to produce a three-dimensional reconstruction, which offers the ability to view anatomic structures in cross-section. The ideal prosthetic position can be simulated and the position and angulation of the implant determined. A computer-generated splint can then be constructed with guide sleeves matched to implant drill sizes. This allows precise placement of the implant at the time of surgery. The ultimate result should allow the surgeon to place the implant optimally in bone while maintaining the angulation that provides the best foundation for the final restoration.

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Principles of Surgical Management of Impacted Third Molar

Part (I)

1 Lower third molar

1.1 Causes of impaction

The third molars or the wisdom teeth normally erupt last, between 18 and 25 years of age. Since they erupt at about the time when the youth goes off into the world to become 'wise' the name 'wisdom teeth' was used to describe them

1.1.1 Theories of impaction

1. Discrepancy between the arch length and the tooth size.
2. Differential growth of the mesial and distal roots.
3. Evolution theory.
4. Lack of development of jaw bones due to consumption of more refined food which causes lack of functional stimulation to the growth of jaw bone.

1.1.2 Local causes of impaction

Like Irregularity in the position of adjacent tooth. density of investing bone, presence of inflammation, under developed jaws, retention or premature loss of primary tooth.

1.1.3 Systemic causes of impaction

A. Prenatal causes: Hereditary and miscegenation

B. Postnatal causes: Rickets, anaemia, congenital syphilis, tuberculosis, malnutrition.

C. Rare conditions: Cleidocranial dysostosis, oxycephaly, achondroplasia, and cleft palate.

1.2 Causes of impacted teeth removal

1. **Pericoronitis and pericoronal abscess:** Most common cause for extraction of mandibular third molars (25 to 30%). Its risk increase with vertical orientation and higher the eruption → distoangular and vertical impaction. If improperly treated→ sub masseteric abscess.
2. **Dental Caries**
3. **Periodontal diseases:** There is high prevalence of increased periodontal probing depths (PD) in **asymptomatic** third molar region.

4. **Orthodontic reasons:** like crowding of incisors or for non-extraction orthodontics, (molar distalization instead of premolars extraction in Class II malocclusion)
5. **To facilitate orthognathic surgery:** e.g. sagittal split osteotomy
6. **Odontogenic cysts and tumours**
7. **Management of unexplained pain:** Possibility of the following must be eliminated
   - Temporomandibular joint dysfunction.
   - Muscle spasm
   - Clenching habit
8. **Resorption of root of adjacent tooth**
9. **Teeth under dental prosthesis:** Teeth that are more superficial
10. **Prevention of jaw fracture:** For those engaged in contact games.
11. **Deep fascial space infection:** In pericoronitis associated with impacted
12. **Impacted teeth as potential source of infection;** in partially erupted third molar tooth. Third molar should be removed in following patient to eliminate further complication in their existing conditions
    a. Prior to administration of radiotherapy
    b. Cardiac patients with valvular disease or those who have undergone valve replacement
    c. Organ transplantation
    d. Insertion of alloplastic implants
13. **For autogenous transplantation to a first molar socket**

1.3 Classification of Impacted Mandibular Third Molar

It is a tool for predicting the difficulty of removal based on the analysis of the periapical x-ray or OPG. The most widely used;

1. Angulation (Winter, 1926) of the impacted tooth: Vertical, Mesioangular, Horizontal, Distoangular, Buccoangular, Linguoangular, Inverted, Unusual.
2. Relationship of the impacted tooth to the anterior border of the ramus (Pell and Gregory, 1942) → space available between ramus and the distal side of the second → space available for tooth eruption;
   - Class I: Sufficient space available to erupt.
   - Class II: Space is less than the mesio-distal width of the crown of 3rd molar
   - Class III: All or most of 3rd molar located within the ramus (difficult to remove)

3. Depth of impaction and the type of tissue overlying the tooth (Pell and Gregory Classification based on relationship to occlusal plane): i.e. soft tissue, partial bony, or complete bony impaction;
    - Position A: The highest portion of the tooth is on a level with or above the occlusal line.
    - Position B: The highest portion of the tooth is below the occlusal line but above the cervical line of second molar.
    - Position C: The highest portion of the tooth is below the cervical line of second molar.

    A mesioangular impaction with a class I ramus relationship and position A depth would be the easiest type → distoangular impaction with a class III ramus relationship and position C depth would involve a difficult surgical procedure.

4. Type of tissue overlying the tooth: i.e. soft tissue, partial bony, or complete bony impaction.
5. State of Eruption; 1. Erupted 2. Partially erupted 3. Unerupted.
6. Number of roots 1. Fused roots (Single) 2. Two roots 3. Multiple roots

Note; Disadvantage of classification: no accepted classification for impacted tooth

- In edentulous jaw
- Associated with infection (bony/soft tissue) or pathological lesions
- Associated with local complicating factors or systemic condition

1.4 Clinical Examination

This include (1) History taking (2) Extra oral examination, and (3) Intraoral examination

1.4.1 History Taking;

1.4.1.1 Complaints of the patient:

1.4.1.2 Medical and dental history:

1.4.2 Extraoral Examination

The face and neck are examined for signs of swelling or redness of the cheek suggestive of infection. The lower lip is tested for anaesthesia or paraesthesia. The regional lymph nodes are palpated for enlargement and tenderness.

1.4.3 Intraoral Examination

1. Mouth opening
2. General examination of oral cavity: Oral mucosa, teeth, oral hygiene.
3. Third molar area: State of eruption of tooth, tissue overlying the tooth (bone/soft tissue only), signs of pericoronitis.
4. Condition of impacted tooth: Carious or with fillings, internal resorption, angulation of tooth, locking of crown of third molar beneath second molar.
5. Condition of second molar and first molar: Crown condition, periodontal condition
6. Amount of space available between the distal surface of second molar and the ascending ramus: If the distance is small, the tooth is less accessible.
7. Pathological complications due to skeletal diseases should be noted

1.5 Radiographic examination

Should provide additional information to those found during clinical examination about;

- third molar
- related teeth
- related anatomical features
- surrounding bone.

The following intra oral and extra oral radiographs are required:

1. **Periapical radiograph:** are more discriminating than OPG and may be more helpful in detecting caries, bone height at the level of second molar and root contour. Periapical radiographs should
2. **Occlusal X-ray**: This will help to show
   - Bucco-lingual relationship indicated by a periapical X-ray
   - Exact position of the crown of the tooth
   - Shape of laterally deviated roots.
3. **Lateral oblique view of mandible:** useful in the following situations:
   - Periapical film could not be taken due to retching, trismus, etc.
   - To provide additional information like vertical height of mandible, amount of bone beneath deeply buried impacted tooth in a thin mandible, existence of pathology in the area. Its use should be considered in the absence of OPG.

4. **Orthopantomogram (OPG):** All the information available from a lateral oblique view can be had from OPG with less distortion (considered the gold standard for surveying the maxilla and mandible for pathological conditions in the lateral plane).
5. **Cone beam computed tomography (CBCT):** Gives accurate three-dimensional imaging of the relationship between the roots of the third molar and the inferior alveolar nerve (IAN). When the OPG suggests a close relationship between the roots of the lower third molar and IAN, CBCT scanning should be advised.

1.5.1 Interpretation of Periapical X-ray

The following factors are considered while interpreting the radiograph:

a. **Access:** By noting the inclination of the radiopaque line cast by the external oblique ridge→ If this line is vertical the access is poor and if horizontal, access is good
b. **Position and depth of impacted tooth:** This is determined by a method described by George Winter; three imaginary lines are drawn described as 'white', 'amber' and 'red' lines.

- The first line or 'white' line is drawn along the occlusal surface of the erupted mandibular molars and extended posteriorly over the third molar region. From this the axial inclination (vertical. mesioangular, distoangular)
- The second imaginary line or 'amber' line is drawn from the surface of the bone lying distal to the third molar to the crest of the interdental septum between the first and second molar → this line indicates the margin of the alveolar bone covering the tooth.
- The third line or 'red' line is used to measure the depth of impaction in bone. Itis a perpendicular dropped from the 'amber' line to an imaginary 'point of application' of an elevator. With the exception of disto-angular impaction, the cementoenamel junction on the mesial surface of the impacted tooth is used for this purpose. It has been noted that for every 1 mm increase in the length of 'red' line, extraction becomes about three times more difficult.
- As a general rule, any tooth with a 'red' line 5 mm or more is better removed under general anaesthesia. If the 'red' line is 9 mm or more, the inferior surface of the crown of the impacted third molar will be either at the level or below the apex of the second molar. When assessing the depth of disto-angular impactions, the perpendicular 'red' line should be dropped to the cemento-enamel junction on the distal side of the impacted tooth and not on the mesial side as in other angulations.

c. Root pattern of impacted tooth:

- The number, shape and curvature of roots
- The presence of hypercementosis or ankylosis
- Root appears blunt and short when the apical portion of root takes a sharp bend in the direction of X-ray beam.
- Root morphology influences the degree of difficulty
  - i- Limited root development leads to a "rolling" tooth → difficult to remove → sectioned in multiple planes → removal.
  - ii- A tooth with one-third to two-thirds root development is easier to remove.
  - iii- Third molars with conical and fused roots are easier to remove than those with widely separated roots.
  - iv- Roots with severe curvature are more difficult to remove.
  - v- Roots that curve in the same direction as the pathway of removal break less often
  - vi- Roots with a mesiodistal diameter that is greater than the tooth diameter at the cervical line must be sectioned longitudinally before removal.
  - vii- The presence of multiple roots may not be visible in radiographs (overlapping) → change angulation of central ray

d. Shape of crown: Teeth with large crowns and prominent cusps → difficult to remove.

e. Texture of the investing bone: If cancellous spaces are large and the bone structure is fine, the bone is generally elastic. The denser the bone→ more time required for its removal with a bur.

f. Inferior alveolar canal: It can be seen to be crossing the roots of the third molar due to either superimposition or grooving/ perforation of the root. This can be distinguished through

1. A band of reduced radio-opacity crossing the roots and coinciding with the outline of the inferior alveolar canal indicates that the tooth root is grooved by the inferior alveolar canal.
2. The compact bone forming the roof and floor of the canal is represented on the radiograph by parallel lines of radio-opacity. Break in the continuity of one or both of these lines is seen when the root is grooved by the inferior alveolar canal.
3. In cases where the radiolucent band crosses the apex of the root and if only the upper white line is broken, a notching of the root is present
4. Characteristic narrowing of the radiolucent band with loss of white lines is suggestive of perforation of the root by the inferior alveolar canal.

The following signs suggests increased risk of nerve injury during third molar surgery

- Diversion of the inferior dental canal (IDC)
- Darkening of the root where crossed by the canal
- Interruption of the white lines of the canal.

Notes

- If the grooving is on the lingual side of the tooth, generous amount of bone is removed on the buccal side and the tooth delivered through the resultant defect.
- If apical notching present by IDC; tooth division is done to avoid nerve damage.
- If root is perforated by the neurovascular bundle →removal of buccal plate of bone →root is then carefully sectioned using a bur at the level of the neurovascular bundle → root fragments removed.

g. Position, root pattern and nature of crown of second molar:

- The closer the third molar is to the second molar → more difficult the surgery.
- Distal tilt of the long axis of the second molar → more difficult the surgery.
- If the second molar has a single conical root it can be easily displaced by an elevator applied to the mesial surface of third molar.
- Large restorations, crown and root canal therapy in second molar teeth → risk to damage to it.

1.6 Armamentarium

1. **Local anaesthesia:** Local anaesthetic containing vasoconstrictor (e.g. lignocaine 2% with adrenalin) ensures adequate analgesia as well as reduces bleeding.
2. **Instruments to incise mucoperiosteum:** Usually No.15 scalpel blade on a No.3 Bard Parker handle
3. **Instruments to reflect mucoperiosteum:** Mucoperiosteum is reflected using periosteal elevator. Howarth periosteal elevator is an ideal instrument for reflecting the mucoperiosteum. This instrument can also be used as a retractor of the mucoperiosteum.
4. **Instruments to retract mucoperiosteal flap:** Numerous instruments are available for this purpose. Austin's retractor for retracting the flap, Kilner retractor for holding the lip, Lack's tongue depressor for retracting the tongue and Rowe's lingual retractor. Other

retractors now available are Minnesota retractor, Cawood-Minnesota. A flat bladed retractor to hold the mucoperiosteal flap when bur is used to remove bone.

5. **Bone cutting/tooth division instruments:** Chisel and bur are used for the removal of bone. Chisel has the advantage of rapidity, no production of heat and no generation of bone dust. However, use of chisel and mallet is an unpleasant experience for the patient → under L.A. avoided → use bur. Other situations for bur usage over chisel are;

- Mandible is thin and atrophic or when the bone is brittle or sclerosed
- Access is limited for the use of chisel (e.g. deeply impacted tooth)

Use of chisel and mallet: The chisel should be 5 to 6 mm width with tungsten carbide tip with sharp edge and the shaft should be sufficiently long enough (nearly17 cm) → adequate visibility of operative site. Give short, sharp, light taps with the mallet by wrist movement and not heavy blows. A chisel has a bevelled and a flat surface → with the bevel superiorly, a deeper cut will result when used with mallet. The chisel should be held at right angles to bone surface with adequate support given to the mandible by the assistant →avoid damage to the temporomandibular joint.

Use of micromotor and bur: micromotor, straight hand piece and bur. Air driven handpiece has the disadvantage of

- causing surgical emphysema
- driving the tooth and bone particles into the soft tissues →can result in postoperative infection.

Tungsten carbide fissure bur now commonly used for bone cutting ('guttering') and sectioning of tooth compared to the round bur (rose head bur). It is preferable to use a straight handpiece since, it is easier to control during use as well as effortless to clean.

6. **Instruments for irrigation and suction:** continuous irrigation used to avoid overheating of bone (can result in necrosis of viable bone cells and subsequent osteomyelitis) and generation of bone dust during the use of bur. A practical method is to use a saline filled syringe with its needle directed towards the revolving bur. An alternative method is to use a system with inbuilt saline pump and irrigation facility connected to the handpiece.

It is mandatory to use a suction apparatus for effective drainage of the irrigant and blood as well as to clear the surgical site off the debris, pieces of crown or root or solid debris

as this can block the suction tip as well as confuse the operator as to where it has disappeared.

7. **Instruments for removal of tooth and debridement of surgical site:** Once, adequate amount of bone has been removed to relieve the tooth of its obstruction, only slight force with an elevator is usually sufficient to deliver it;
    - Dental extraction forceps in general are not advisable → can result in fracture of mandible.
    - Cross bar elevators like Winter's cross bar elevator also generate tremendous force → should be avoided. Moreover, their beaks can cause perforation of thin lingual plate pushing the fractured root piece into the lingual pouch
    - Elevators like straight elevator, Coupland elevator and Creyer elevator may be used with caution.
    - Instruments that can be safely used are elevators with small mechanical advantage like Warwick James elevator.

    After the tooth has been removed → debridement of the wound → uneventful healing;
    - Curved mosquito → remove follicular remnants and bone pieces.
    - Angulated curette → clean the socket off the debris.
    - Bone file → Smoothening rough edges of bone
    - The socket and the soft tissue flap are once again thoroughly irrigated with saline taken in a syringe to wash off the debris.
8. **Instruments for closure of mucoperiosteal flap:**
    - Medium sized triangular cutting needle with 11/16 circle
    - Needle holder
    - Toothed dissecting forceps
    - Suture material (3-0 size) → black silk, linen, catgut or vicryl
9. **Other equipment:** like
    - Operating loupe → better visualization of the surgical site.
    - Mouth prop →reduces the fatigue of the jaw.

Principles of Surgical Management of Impacted Third Molar

Part (II)

1.7 Surgical procedure

1.7.1 Patient positioning

Generally, for operative procedures in mandible the occlusal plane of lower teeth should be parallel to the floor and for the maxillary teeth the occlusal plane of the upper teeth at 45° angles to the floor. The instruments are arranged in a rational order of their intended use over the instrument tray of the dental chair or more preferably on a separate instrument trolley.

1.7.2 Incision and Designing the Flap

- As a general rule, the deeper the third molar → more extensive the bone removal required and the necessity for tooth sectioning.
- The most commonly used flap is the envelope flap, which extends from just posterior to the position of the impacted tooth anteriorly to the level of the first molar. The posterior end of the incision is directed buccally along the external oblique ridge.
- If greater access is required, a release incision is given on the anterior aspect of the incision, creating a triangular flap (started from a point approximately 6 mm down in the buccal sulcus and then extended obliquely upwards to the gingival margin to a point at the junction of the posterior and middle thirds of the second molar).
- The blunt end of the periosteal elevator is passed beneath the mucoperiosteum to reflect the soft tissue.
- Reflection of lingual mucoperiosteum is kept to the minimum to avoid injury to lingual nerve.

1.7.3 Bone Removal

The amount of bone removal varies with the depth of impaction→ remove sufficient amount of bone to free the tooth from obstruction and to provide a point of application for the elevator.

A. A common technique 'lingual split bone technique' introduced by Ward (1956) → a section of bone lingual to the wisdom tooth is fractured off to facilitate the removal of the impacted tooth.
B. Buccal approach; should be kept to minimum to avoid weakening of the mandible.

1. The bone on the buccal and the distal aspect of the impacted tooth is removed down to the level of the cervical line (Further bone removal if required is done in a manner not detrimental to the strength of mandible) → achieved by drilling a deep vertical **gutter** alongside the buccal aspect and if required on the distal aspect of the tooth.
2. Guttering method will ensure that the height of the buccal plate is maintained without weakening the mandible and adequate space is created for tooth delivery.
3. As the bur reaches the apex of the tooth, the inferior alveolar canal may be opened → brisk haemorrhage from inferior alveolar vessels → controlled with pressure pack or bone wax.
4. Drilling in the region of the mesial surface of impacted tooth should be kept to the minimum → avoid damage to the distal aspect of second molar.
5. Removing bone on the distolingual aspect → care protect the lingual.
6. Moderate force alone is sufficient to displace the tooth.
7. If the tooth is still resistant → plan for further bone removal or tooth sectioning.
8. At the time of elevating the tooth, the index finger of the operator's left hand should rest on the occlusal surface of the wisdom tooth to judge its movement and the other fingers support the mandible.
9. In order to apply the elevator, a point of application (purchase point) is required →either in the bone **or** a bur *cut* is made on the tooth → deep enough and *placed* in substantial portion of tooth structure → elevation of rather than fracture.

1.7.4 Sectioning and Tooth Delivery

Tooth sectioning is performed either with a bur or a chisel and it helps to;

- reduce operating time
- Avoid the need to remove additional amount of bone.

A- First section is generally done at the neck of the tooth using bur →facilitate the removal of the crown followed by the roots in one piece.

B- Divergent roots → roots have to be divided and removed separately.

C- The following key points should be observed while performing tooth sectioning:

- If sectioning tooth in a buccal to the lingual direction → only three quarters the way is cut using bur. The reminder is then split with a straight elevator → prevents injury to the lingual cortical plates and lingual nerve

- The line of sectioning of crown/tooth should be perpendicular. If NOT → sectioned segment will be wider at the bottom → elevation will be difficult
- If sectioning in the superior to inferior direction → Entry of bur is limited to three fourth of the width of the tooth and the rest is separated with elevator → if NOT → possibility of damaging the contents of the canal

1.7.5 Modifications for Removal of Impacted Tooth

- Mesioangular impaction; Buccal gutter is extended mesially to reach the mesial surface of impacted tooth beneath the cementoenamel junction → tip of the elevator can engage beneath the cervical cementum on the mesial aspect →the tooth turns distally → mesial angulation of the tooth into a vertical position → deliver the tooth. If removal is difficult → remove necessary distal bone and/ or sectioning the distal half of the crown to just below the cervical line.
- Mesioangular tooth is 'locked' beneath the distal convexity of the crown of the second molar → section impacted tooth's crown at the cervical region → remove coronal portion by applying force below its inferior surface →The roots are removed by engaging the bifurcation.
- The horizontal impaction; Bone is removed superiorly to expose the whole width of the crown and the upper third of the root → tooth sectioned at the cervical region → crown is removed → The root is then brought forwards into the space previously occupied by the crown → removed either in a single piece or after sectioning. If impacted tooth is not locked beneath the crown of the second molar → turn the tooth into a vertical position by application of force in the mesial aspect.
- Deep vertical impaction; one of the more difficult ones to remove. Bone is removed first from the occlusal, buccal, and distal aspect. The distal half of the crown is then sectioned and removed, and the tooth is elevated by applying a small straight elevator at the mesial aspect of the cervical line.
- Less deep vertical impactions, mesial application of force with an elevator can dislodge the tooth
- Vertical impaction with widely divergent roots; crown is divided first followed → sectioning of the roots and its subsequent removal.
- A deep, vertically impacted third molar below the cervical line of the second molar and fully covered with bone → tooth should be exposed and a buccal and distal trough

(guttering) created → sectioning of crown in a horizontal fashion → roots can be elevated in one piece or sectioned and removed (distal preceding that of the mesial).

- The distoangular impaction; Most difficult tooth to remove → because the pathway of delivery is into the vertical ramus (more distal bone must be removed) → create an adequate buccal and distal trough (guttering) around the full crown of the tooth to a depth below the cervical line → create a point of application of elevator on the buccal aspect of the tooth → using the buccal cortical plate as the fulcrum → elevate the tooth out of the socket upwards and ***distally*** → some movement → distal portion of the crown sectioned in a horizontal fashion and removed (section the tooth segments further as needed rather than to remove more bone) → The roots delivered together or sectioned and delivered independently with a Cryer's elevator.

1.7.6 Other Methods for Removal of Impacted Lower Third Molar

- Sagittal split ramus osteotomy: In this technique two cortical cuts are done: horizontal cut through the medial aspect of the ramus to area posterior and above the lingula, and a vertical cut through the anterior border of ramus and extends down to the inferior border of mandible. An osteotome is used to complete the split along the horizontal and then the vertical cuts. the result is longitudinal split of the mandible in the cut area. The technique is used in patients with;

    - history of recurrent infection with/ or without trismus associated with a deeply impacted lower right third molar tooth, and/or
    - impacted lower third molar intimately involved with the inferior alveolar nerve.

- Buccal corticotomy: For deeply impacted mandibular teeth → a rectangular **window** is made over the deeply impacted tooth using a narrow fissure bur →mesial and distal cuts almost reaching the inferior border of the mandible → **window** removed with osteotome → impacted molar divided and removed → The bony (window) replaced.
- Lingual Split technique: Involves the use of a chisel and mallet to remove or displace the lingual plate of bone adjacent to lower third molar. A small amount of buccal bone is often removed to facilitate exposure of the crown and provide a point of application for an elevator. Sometimes tooth division is required for its removal (operated under general anaesthesia, young patients → elastic bone)

- Partial Odontectomy: (coronectomy, deliberate root retention) procedure devised to protect the IAN. Radiographic features suggesting an intimate relationship:
    - Darkening of the root and interruption of the white line of the canal
    - Narrowing of the canal
    - Deflection of the roots

  Adequate amount of root must be removed below the crest of the lingual and buccal plates of bone → bone forms over the retained roots. Contraindications to Partial Odontectomy

    - Active infection around the tooth
    - Mobile teeth - any retained mobile root → nidus for infection.
    - Horizontally impacted tooth along the course of the nerve→ sectioning the tooth will damage the IAN.
- Orthodontic extraction: for the safe extraction of impacted third molars with a high risk of neurological complication due to the close proximity to mandibular canal;
    - Phase 0 Assessment of surgical risks
    - Phase 1 Creation of orthodontic anchorage: stainless steel lingual arch and buccal stainless-steel sectional wire tied from second molar to the first bicuspid.
    - Phase 2 Surgical exposure of the third molar crown
    - Phase 3 Orthodontic extrusion (3^{rd} molar); setting 3^{rd} molar apart from IAN
    - Phase 4 Clinical and radiographic assessment of the extrusion level
    - Phase 5 Third molar extraction →if adequate bone separating the root from INA

2 Upper third molars

2.1 Classification of impacted maxillary third molars:

- State of Eruption; 1. Fully erupted 2. Partially erupted 3. Unerupted
- Angulation of the Tooth; 1. Vertical, 2. Mesioangular, 3. Distoangular, 4. Laterally displaced with the crown facing the cheek, horizontal, inverted and transverse positions, 5. Aberrant position
- Pell and Gregory Classification;
    - Position A; 3rd molar occlusal surface is at the same level of that of 2^{nd} molar.

- Position B; 3rd molar occlusal surface is between occlusal plane and cervical line 2nd molar.
- Position C; 3rd molar occlusal surface is at or above cervical line of 2nd molar

- Relationship of Impacted Maxillary Third Molar to the Maxillary Sinus
  - Sinus approximation (SA): No bone or a thin partition of bone between third molar and maxillary sinus.
  - No sinus approximation (NSA): 2 mm or more bone between third molar and maxillary sinus.
- Nature of Roots; 1. Fused (conical) 2. Multiple—Favourable/Unfavourable

2.2 Indications for the Removal of Maxillary Third Molar

1. Extensive dental caries which is beyond restoration
2. Recurrent pericoronitis
3. Buccally or distally erupting tooth → cheek biting
4. Tooth involved in pathological process
5. Over erupted and non-functional upper third molar
6. Buccally erupting upper 3rd molar impinging on the coronoid process → pain during movement.
7. Interference with placement of prosthesis

2.3 Local Contraindications for Removal

1. Symptom-less upper third molar completely embedded in bone.
2. Third molar high in alveolus → displacing into antrum or infratemporal fossa
3. Deeply impacted tooth →removal can damage the adjacent second molar

2.4 Radiographic Examination

1. Periapical X-ray
2. OPG
3. Occlusal X-ray
4. True lateral view
5. PNS (paranasal sinus) view of maxilla → if associated pathology
6. CT scan—especially if associated pathology

2.5 Determining the Degree of Difficulty of Removal

1. Angulation: same angulations in mandibular third molar cause opposite degree of difficulty for maxillary third molar extraction.
2. Position in buccoangular direction: directed towards the buccal aspect → easy. Positioned towards the palatal aspect → difficult to remove.
3. Type of overlying tissues; only soft tissue covering is easier to remove
4. Proximity to maxillary sinus
5. Proximity to maxillary tuberosity; tuberosity can be fractured. Factors contributing to this hazard are:
    - Dense and non-elastic bone as in old age
    - Multirooted tooth with large bulbous roots
    - Large maxillary sinus (that include roots of third molar)
    - Use of excessive force to elevate the tooth
    - Mesioangular impactions
6. Other factors influencing the degree of surgical difficulty:
    - Tooth with roots which are thin →difficulty increased
    - Hypercementosis →difficulty increased
    - Wide periodontal space →difficulty decreased.
    - Tooth with a wide follicular space →difficulty decreased
    - Bone is more elastic as in young patients →difficulty decreased
    - Close relationship to second molar →difficulty increased
    - Fusion of third molar with roots of second molar→difficulty increased
    - Presence of large restoration on second molar →difficulty increased
    - Difficult access due to small oral aperture or trismus→difficulty increased

2.6 Steps in the operative procedure for removal of unerupted third molar

One of the difficulties that will be encountered during its surgical removal is the limited access due to the presence of the coronoid process. This can be overcome by opening the mouth only partially.

1. Incision: It starts from the mesial aspect of first molar and extends posteriorly beyond the distobuccal aspect of second molar and then continued into the tuberosity. If greater access is required (deep impaction) → release incision in the mesial aspect of second molar to raise a triangular flap.

2. Using a Howarth's periosteal elevator, the mucoperiosteum is reflected.
3. Bone removal is restricted to the occlusal and the buccal aspect of the tooth down to the cervical line (using chisel or bur). Additional bone is removed on the mesial aspect of the tooth above the height of contour of the crown → for the insertion of an elevator and to act as a purchase point.
4. Maxillary third molars rarely need sectioning (overlying bone is usually thin and elastic). If bone is thick, sclerotic and less elastic→bone removal rather than tooth sectioning.
5. Delivery of the tooth: using small straight elevators. The following points should be borne in mind while elevating the tooth
    - Due to the proximity of the maxillary sinus and infratemporal fossa → no upward pressure exerted during bone removal and delivery
    - This can be avoided by creating sufficient room between crown and surrounding bone
    - Moderate pressure is exerted downward and outward with the forefinger placed posterior to maxillary tuberosity to detect tuberosity fracture if it occurs.
    - Minnesota retractor or periosteal elevator can be placed distal to impacted maxillary third molar during final elevation →avoid displacement under the flap and into the infratemporal fossa.
6. Debridement and Closure: A single suture is all that is needed to secure the wound. The suture is passed from the palatal side of the interdental papilla between the first and second molars into the anterior end of the buccal flap.

2.7 Complications During Surgery of Impacted Maxillary Third Molar

1. Displacement of tooth into maxillary sinus:
    - partially erupted and has conical roots
    - excessive force is exerted for elevating a buried wisdom tooth
    - Retrieval can be accomplished via a Caldwell-Luc approach
2. Dislodgement into soft tissues and into the infratemporal fossa may occur:
    - Buccal flap is not adequate
    - Decreased visibility during surgical extraction
    - Incorrect extraction technique
    - Distolingual angulation of tooth
    - Third molar crown above the level of the adjacent molar root apices.

Such displaced tooth should be removed as early as possible to avoid development of infection. Tooth retrieval can be done with haemostat or Allis' forceps. Surgical access is gained through an incision along the crest of the alveolus. If the tooth could not be removed after a single effort → patient informed → antibiotic is administered to prevent infection→ tooth removed four to six weeks later by an oral and maxillofacial surgeon.

3 Summary of Instructions to Patient Following Surgical Removal of Impacted Tooth

1. Remove the gauze pack after 30 minutes to one hour
2. Apply ice (ice cubes taken in a polythene bag) on the face for the first 24 hours.
3. For the first day take cold liquids or semisolids.
4. Avoid warm saline gargle in the first 24 hours.
5. There may be mild to moderate swelling on the side of the face for three to four days.
6. Mild bleeding/oozing of blood can be there from the surgical site for one to two days. In the event of excessive bleeding bite on a fresh piece of sterile gauze and inform the doctor.
7. In the first few days, difficulty may be experienced in opening the mouth. To avoid this, from the next day of surgery onwards try to open the mouth forcefully.
8. From the next day onwards after surgery or once the oozing of blood has completely stopped, warm saline mouth-baths can be used at fourth hourly intervals. Avoid application of dry heat on the face.
9. Tooth brushing have to be done from the next day on wards.
10. Take the drugs prescribed by the doctor at regular intervals.
11. Avoid alcohol, smoking, physical exercise and long journey for the next few days.
12. Report for review to the doctor as suggested for suture removal.

4 Drug therapy

4.1 Use of antibiotic

Postoperative oral prophylactic antibiotic treatment after the removal of lower third molars have **not** shown to contribute to a better wound healing, less pain, or increased mouth opening and could not prevent inflammatory problems after surgery. And therefore, is **not** recommended for **routine** use. The following principles should be considered before prescribing antibiotics:

1. The surgical procedure should harbour a significant risk for infection, for example:
    - Long procedure (> 30 minutes) or difficult surgery involving significant tissue trauma.
    - Where there is existing infection in and around the surgical site.
2. Administration of the antibiotic must be immediately prior to or within 3 hours after the start of surgery:
    - The ability of systemic antibiotics to prevent the development of a primary bacterial lesion is confined to the first 3 hours after inoculation of the wound.
    - Commencing prophylactic antibiotic cover the day before surgery only leads to the development of resistant organisms.
    - Continuing antibiotics for days after surgery has not been shown to decrease the incidence of wound infection.
3. Prophylactic antibiotics should be given at twice the usual dose over the shortest effective time so as to minimize the potential side-effects of long-term use (e.g. diarrhoea) and to prevent the growth of resistant strains of bacteria.
4. Examples of antibiotic prophylactic regimens:
    - Amoxicillin 3 gm orally, 45 minutes before surgery under local anaesthesia.
    - Clindamycin 600 mg orally, 30 minutes before surgery under local anaesthesia for patients allergic to penicillin.
    - Benzyl Penicillin 600 mg IV/IM on induction for procedures under general anaesthesia.
    - Erythromycin lactobionate 500 mg IV on induction for surgery under general anaesthesia for patients allergic to penicillin.

    The above dose may be followed with an additional oral dose 6 hours after the initial dose

4.2 Use of Anti-inflammatory Drugs and Steroids

Perioperative corticosteroids have been used to minimize swelling, trismus and pain in oral and maxillofacial surgery patients. However, the method of usage is extremely variable. The most widely used steroids are dexamethasone and methylprednisolone. Common dosages of dexamethasone are 4 to 12 mg given IV at the time of surgery. Additional oral dosages of 4 to 8 mg. twice a day for the day of surgery and 2 days afterwards leads to the maximum relief of swelling, trismus and pain. Methylprednisolone is most commonly given IV 125 mg at the time

of surgery followed by significantly lower doses, usually 40 mg 3 or 4 times daily taken orally for the day of surgery and for 2 days after surgery.

4.3 Use of Non-steroidal Anti-inflammatory Drugs (NSAIDs)

they are frequently used after surgical procedures in order to reduce the soft tissue oedema and pain by suppressing inflammation

4.4 Use of Analgesics

- It has been reported that soluble aspirin 900 mg provides significant and more rapid analgesia than paracetamol 1,000 mg in the early postoperative period
- Patients should be encouraged to take analgesics either before the onset or at the time of onset of pain or discomfort rather than waiting till the pain becomes unbearable.
- Long-acting local anaesthetic solutions may be of value in some situations where extreme pain is likely to be a feature in the immediate post-operative period.
- It has been shown that administering a dose of analgesic preoperatively markedly reduces postoperative pain.

5 Complications of Impaction Surgery

5.1 Complications during the Surgical procedure

1. Complications during incision; Excessive bleeding may occur in the following situations:
   a. Pre-existing local inflammation.
   b. Bleeding from retromolar vessels
   c. Bleeding from facial vessels
   d. Damage to lingual nerve

2. Complications during bone removal
   a. Use of bur
      - Accidental burns
      - Laceration of soft tissues
      - Injury to inferior alveolar bundle
      - Injury to adjacent tooth
      - Injury to lingual nerve
      - Necrosis of bone
      - Emphysema

b. Use of chisel:
    - Splintering of bone
    - Fracture of mandible
    - Displacement of tooth into lingual pouch
    - Injury to lingual nerve
    - Injury to second molar tooth and soft tissues

3. Complications during sectioning of tooth
    - Incorrect line of sectioning of crown
    - Injury to mandibular canal: During

4. Complications during elevation of tooth
    - Fracture of impacted tooth/ root
    - Breakage of bur
    - Injury to second molar
    - Fracture of mandible
    - Dislodgement of tooth/crown into the lingual pouch or lateral pharyngeal space
    - Injury to mandibular canal

5.2 Post-Surgical Sequelae and Complications

1. Haemorrhage
2. Oedema
3. Trismus
4. Pain
5. Infection
6. Alveolar osteitis (Dry socket)
7. Nerve Injury
8. Surgical Emphysema
9. Hematoma
10. Pain during swallowing
11. Pyrexia
12. Osteomyelitis
13. Temporomandibular joint (TMJ) complications
14. Fracture of instruments: Especially that of sharp Ones
15. Periodontal pocket formation distal to second molar
16. Aspiration /Swallowing of tooth

ORAL SURGERY

LECTURE

ا.م.د.احمد فاضل ابراهيم القيسي

Odontogenic and Fascial Spaces Infections

Infection: is the invasion and the multiplication of pathogenic microorganisms into a bodily part or tissue, which may produce subsequent tissue injury and progress to overt disease through a variety of cellular or toxic mechanisms.

The pathogenic organisms can be Bacteria, Viruses, Fungi or Protozoan.

The body's response to infectious agents is an inflammatory response, which is essentially protective, it consists of:

a. Hyperemia caused by vasodilatation of the arterioles and capillaries, and the release of permeability factors to allow for the passage of nutrients and leukocytes.
b. Precipitation of a network of fibrin to wall off the infected region.
c. Phagocytosis of bacteria and other organisms as well as dead cells by macrophages.

- Depending on the duration and severity of the infection, the inflammatory response is divided into:

  Acute inflammation; with rapid progression and typical signs and symptoms.
  Subacute inflammation; considered as transition between acute and chronic inflammation.
  Chronic inflammation; associated with longer duration and slight clinical symptoms. Infections could be local; confined to one body system, or generalized like Septicemia.

- **Signs and Symptoms of infection**

  These include systemic signs and symptoms like; fever, malaise and anorexia. There are also local signs and symptoms, which are present to some degree in all patients:

  a. **Rubor** (redness), it results from vasodilatation.
  b. **Tumor** (swelling), in infection it results from accumulation of tissue fluid or pus.
  c. **Calor** (heat), results from the inflow of warm blood from deeper tissues, increased velocity of blood and increased metabolic rate.
  d. **Dolor** (pain), results from pressure on the nerve endings, also from the action of liberated or activated factors such as Kinins, Histamine and Bradykinin on nerve endings.
  e. **Functio laesa** (loss of function), caused by mechanical factors and reflex inhibition of muscle movement associated with pain.

Odontogenic Infections

They are almost always bacterial in origin, they consist of Periapical, Periodontal or Pericoronal infections

1- **Periapical infections**: These infections arise when bacteria from infected necrotic pulp from a carious tooth or contaminated traumatic exposure of the pulp invade the periapical tissues through the apical foramen, sometimes the bacteria gain access through an accessory canal or through furcation area leading to infection that presents on the lateral surface of the tooth. These infections manifest themselves as a primary acute infection or as an exacerbation of a preexisting chronic periapical infection. The main clinical features include; a non-vital tooth, the tooth is tender to bite and to percussion, the periapical region is tender to pressure, the tooth is slightly raised in the socket due to the acute inflammation and swelling of the periodontal inflammation, the tooth may be mobile. There is an intense throbbing pain, if treatment is delayed bone resorption will allow the pus to drain under the periosteum with diminution of pain intensity. A periapical radiograph may show no significant changes except for widening of the periodontal space especially in acute infections or it may reveal the presence of periapical radiolucency.

2- **Periodontal infections:** it arises from preexisting periodontal pocket, in some cases food impaction or repeated occlusal trauma are the precipitating factors. The teeth involved are not necessarily non vital, an acute infection produces redness and swelling near the gingival margin, the pain is continuous, dull or throbbing but usually less than that experienced in acute periapical infections, pus may discharge from the gingival margin or may produce a sinus. The tooth may be mobile.

3- **Pericoronal infections:** Also called pericoronitis is an infection of the soft tissue covering the crown of a partially erupted tooth, it is almost always associated with partially erupted impacted lower third molars.

   Etiology involves:

   - Food impaction between the crown and the overlying gum (operculum) which is a favorable site for bacterial proliferation and inflammation.
   - Trauma to the overlying gum from the opposing tooth, but it is hard to determine whether the trauma occurs before or after the inflammatory edema of the gum.
   - Virulent microorganism.
   - Lowering of the host resistance.

   Pericoronitis appears to be the result of a combination of four mentioned etiological factors.

   The clinical presentation depends on the severity of infection:

   Chronic pericoronitis is either asymptomatic or there is mild discomfort. In Subacute pericoronitis there is a dull pain which is well localized, the gum pad may be tender and red and there may be expression of a white material from underneath the gum flap, this material is composed of desquamated epithelial cells, food stuff, dead and living bacteria, there may be slight trismus and lymphadenopathy (LAP). In Acute pericoronitis there is severe pain and limitation of mouth opening, intraoral swelling

and there may be extraoral swelling as well as fever, malaise and LAP. Pus may be expressed from underneath the flap, sometimes the pus may track submucosally and is expressed from a sinus in the molar or premolar region, this is termed migratory abscess.

In treating pericoronitis, it is essential to determine if the tooth is likely to achieve full eruption, this is done by full clinical and radiographic examination, consideration should be given to the age of the patient and history of previous attacks.

Treatment of pericoronitis consists of:

- Drainage of abscess if present.
- If there is no abscess, gentle irrigation of the pericoronal space with normal saline, sometimes an antiseptic solution can be instilled in this space.
- If the upper third molar irritates the swollen gum flap, the pressure should be relieved by extraction or grinding the offending cusps.
- Antibiotics are needed for cases of acute infections.
- Surgical extraction of the impacted teeth should be carried out after the inflammation subsides.

Bacteriology

Odontogenic infections are caused mostly by bacteria that live on or in the host, when such bacteria gain access to the deeper tissues, they cause infection. Most odontogenic infections are mixed infections, caused by multiple bacteria (aerobic and anaerobic), with an average of five species involved. The most common aerobic bacteria involved in odontogenic infections are Streptococci and Staphylococci (Aureus and epidermidis), other less common aerobic organisms include Neisseria species, Corynebacterium species and Haemophilus Influenzae. While the most common anaerobic bacteria are anaerobic Streptococci species, Peptostreptococcus, Bacteroids and Fusobacterium species.

Presentation of the odontogenic infection

Odontogenic infections may spread beyond the confines of the dentoalveolar bone into the soft tissue, they may be presented as:

Cellulitis; which results from spreading of infection into the loose connective tissues (C.T.), it presents as a warm, diffuse, erythematous, indurated and painful mucosal or cutaneous swelling. It does not result in the formation of large amount of pus, Streptococci are more often associated with cellulitis, these organisms produce enzymes such as Streptokinase and Hyaluronidase that break down fibrin and C.T. ground substance, facilitating the rapid spread of infection. Antibiotics and removal of the cause of infection are usually sufficient. Incision and drainage are indicated if there is no improvement or if evidence of purulent collection is identified.

Suppurative infections; characterized by abscess formation, which can be defined as a thick-walled pocket of tissue containing pus. Pus consists of necrotic tissue, dead and

living bacteria and dead white cells, it is often associated with Staphylococci and anaerobes such as Bacteroides.
S. aureus produce the enzyme Coagulase that coats the bacteria with fibrin and reduces the ability of the host cells to phagocytize it. The area of infection may or may not be fluctuant, treatment is by incision and drainage with antibiotics, followed by the treatment of the cause of infection.

Routes of Spread of infection: it can occur through the following routes
1. By direct continuity through the tissue.
2. By the lymphatics to the regional lymph nodes which may result in secondary infection. In acute infections the lymph nodes are enlarged, soft and tender, the surrounding skin may be red and edematous. In chronic infections the enlarged nodes are firmer, not tender with no redness or edema of the surrounding skin.
3. By blood stream, this is uncommon, it occurs along the veins, it can result in serious complications like cavernous sinus thrombosis and septicemia.

The factors that influence the spread of odontogenic infections:
1. Virulence of the organism, it is the disease producing ability of the organisms.
2. Status of patient's immune system, several conditions affect the general health of the patient adversely such as diabetes, malnutrition, alcoholism, HIV infections or patients taking immunosuppressive drugs.
3. Anatomical factors influencing the direction of spread of infection such as:
- The site of the source of infection.
- The point at which the pus escapes.
- The natural barriers to the spread of pus such as fascia, muscles or bone.

Physical examination:
Clinical examination: It includes inspection, palpation and percussion and it aims to identify the signs and symptoms of infection like the presence of swelling, redness or draining fistula. Palpation is used to examine the size, note the tenderness, assess local temperature and to determine the presence of fluctuation. Trismus should be noted with measurement of the interincisal distance.
Intraorally, the teeth and gingiva should be examined for the presence of caries, restorations or localized swelling. Percussion determines the areas of tenderness. Pulp testing may be needed to assess the vitality of the teeth. The intraoral examination should include the ducts of the parotid and submandibular salivary glands, tongue, soft palate, tonsillar fossa and oropharynx. Manifestations of serious odontogenic infections may include; airway compromise, septicemia, fever, lethargy, fatigue, dehydration, rapid progression, dysphagia, odynophagia and drooling.
Radiological examination: radiographs to identify the cause of infection, periapical, occlusal and OPG radiographic views are used. Ultrasound, CT scan and MRI can also be used in deep infections.

Laboratory studies: are also indicated to evaluate the immune system, white blood cells (WBC) and differential WBC count.

Infection of the fascial spaces

Fascial spaces are potential spaces, they are surrounded by muscles, loose C.T. and bone, they contain different anatomical structures and they are separated by collection of pus, blood or by surgeon's finger. They are contiguous and infection spreads readily from one space to another. A thorough knowledge of the anatomy of the face and neck is necessary to predict the pathways of spread of infections.

Fascial spaces are sometimes classified as **primary**; which are directly adjacent to the origin of the odontogenic infections, and **secondary**; that become involved following the spread of infection to the primary spaces.

Infection of spaces in relation to the lower jaw

1. **Submental space infection :**

   Anatomic boundaries: this space lies between the Mylohyoid muscle above, skin, subcutaneous tissue, Platysma muscle and deep cervical fascia below, laterally by lower border of the mandible and anterior bellies of Digastric muscle. It contains submental lymph nodes embedded in adipose tissues.

   Source of infection : direct source from infected lower incisors and canines, lower lip, skin overlying the chin or from the tip of the tongue and the anterior part of the floor of the mouth. An indirect source of infection from submandibular spaces. The site of the swelling is mostly extraoral including the chin and submental areas which are firmly swollen.

   The site of incision and drainage: is extraoral horizontal incision through the skin posterior to the crease behind the chin, providing dependent drainage and most esthetically acceptable scar. It may be drained intraorally through the Mentalis muscle via the labial vestibule, but the dependent drainage can not be established

2. **Submandibular space infection**

   Anatomic boundaries it is bounded by Mylohyoid muscle superiorly, anterior and posterior bellies of Digastric muscle inferiorly, hyoid bone posteriorly, Mylohyoid, Hyoglossus and Styloglossus muscles medially, laterally the space is bounded by the skin, superficial fascia, Platysma, deep fascia and the lower border of the mandible. This space contains the submandibular salivary gland and lymph nodes in addition to facial artery and vein, lingual and Hypoglossal nerve as they course deep to the submandibular salivary gland.

   Source of infection from the lower molar teeth especially second and third molars, as the infection perforates the lingual cortex of the mandible below the Mylohyoid muscle attachment. Infection can also spread from the tongue, posterior part of the floor of the mouth, upper posterior teeth, cheek, palate, the maxillary sinus and the submandibular salivary gland. Indirectly the infection may spread from infected sublingual and submental spaces. Submandibular space infections can spread posteriorly to the pharyngeal space. Submandibular space infection presents as a firm or fluctuant erythematous swelling of the submandibular region, the swelling

bulges over and obliterates the inferior border of the mandible, there may be trismus, other signs and symptoms of infection may or may not be present.

Site of incision and drainage it is extraoral incision made parallel and about 2 cm. below the inferior border of the mandible to avoid injury to the marginal mandibular branch of the facial nerve, the incision extends through the skin and subcutaneous tissue only while the space is entered bluntly to avoid structures within the space.

3. Sublingual space infection:

Anatomic boundaries this is a V-shaped space, it is bounded anteriorly and laterally by the mandible, superiorly by sublingual mucosa, inferiorly by the Mylohyoid muscle and medially by Genioglossus, Geniohyoid and Styloglossus muscles.

Source of infection it is usually from the premolar and less commonly from molar teeth when the infection perforates the lingual cortex of the mandible above the attachment of the Mylohyoid muscle. Indirectly the infection may spread from submental and submandibular spaces. Infection from sublingual space may invade the submandibular and pharyngeal spaces. Clinically there is erythematous swelling of the floor of the mouth that may extend through the midline since the barrier between the two sublingual spaces is weak, usually there is elevation of the tongue.

Site of incision and drainage intraorally by an incision through the mucosa only parallel to Wharton's duct and lingual cortex in anteroposterior direction and away from the sublingual fold. This space may be drained extraorally through submandibular and submental incisions through the Mylohyoid muscle if the infection of these latter spaces is also evident.

Ludwig's Angina

It is a massive firm cellulitis, affecting simultaneously the submandibular, submental and sublingual spaces bilaterally. It is a very serious conditions that require prompt treatment, it was described by Wilhelm Friedrich von Ludwig in 1836.

Causes

- Dental infections in 90% of the cases.
- Submandibular salivary gland infections.
- Mandibulr fractures.
- Soft tissue lacerations and wounds of the floor of the mouth. The term angina is related to the sensation of suffocation. If untreated this condition is almost fatal mainly due to posterior extension of the infection into the epiglottis causing epiglottic edema and respiratory obstruction.

Signs and Symptoms:

there is a firm extensive bilateral submandibular swelling, intraorally there is swelling of the floor of the mouth that raises the tongue which may protrude from the mouth in extreme cases. The patient is toxic, feverish and there is dyspnea and difficulty in swallowing.

Treatment:

- securing the airway, endotracheal intubation is very difficult in this situation, tracheostomy may be needed, but it is also difficult to perform due to the massive neck edema.
- General anesthesia should be avoided.
- Early surgical drainage of all the infected spaces bilaterally under local anesthesia, little pus is obtained since the infection is usually cellulitis.
- Intravenous antibiotic, using a combination of Penicillin and Metronidazole. Some authorities advocate high dose of antibiotics without surgery until fluctuation develops.

4. **Buccal space infection**

   Anatomic boundaries bounded by the Buccinator muscle and buccopharyngeal fascia medially, skin of the cheek laterally, labial musculature anteriorly, zygomatic arch superiorly, the inferior border of the mandible inferiorly and the pterygomandibular raphe posteriorly. It contains the buccal pad of fat, facial artery and the parotid duct.

   Source of infection of this space can be related to both jaws. The relationship of the origin of the Buccinator muscle from the alveolar bone and the apices of the upper and lower premolars and molars determines the direction of the spread of infection from these teeth. If the infection exits the alveolar bone above the attachment of the muscle in the upper alveolus or below the attachment in the lower alveolus, the infection spreads to the buccal space. Otherwise the infection spreads intraorally into the vestibule where it can be drained easily. Usually the swelling appears in the cheek, the inferior border of the mandible can still be palpated.

   Site of incision and drainage intraorally by a horizontal incision in the buccal mucosa below the parotid duct, the incision should be through the mucosa only, the space should be entered bluntly using artery or sinus forceps through the Buccinator muscle to avoid damage to the facial artery and nerve. The incision can be placed extra orally if the pus points cutaneously.

5. **Masticator spaces infection** These are well differentiated spaces but they communicate with each other as well as with the buccal, submandibular and pharyngeal spaces.

   They are:

   - Masseteric space.
   - Pterygomandibular space.
   - Temporal space.

Masseteric space infection (also called sub masseteric space)

Anatomic boundaries this space lies between the outer surface of the ascending ramus of the mandible medially, the Masseter muscle laterally and the parotid gland posteriorly.
Source of infection usually from molar teeth especially lower third molars, it can also occur after fracture of the angle of the mandible or it can also spread from buccal space. The swelling is moderate in size over the ascending ramus and the angle of the mandible region. This infection is characterized by a marked trismus. Chronic abscess can run a protracted course and can spread to the muscle itself or it can cause osteomyelitis of the ramus of the mandible.
Site of incision and drainage extra orally below and behind the angle of the mandible, the incision is carried through the skin and the subcutaneous tissue then by blunt dissection through the Platysma muscle and the deep fascia, after incising the attachment of the muscle at the angle the periosteal elevator is inserted beneath the muscle and in close contact with the outer surface of the ramus of the mandible to drain all the pus.
Intraorally drainage can be carried out through an incision along the anterior border of the ramus of the mandible, but in this case the drainage can be insufficient as it is not in a dependent point, also intraoral drainage may prove to be very difficult due to the presence of trismus.

Pterygomandibular space infection
anatomic boundaries it is bounded medially by the Medial Pterygoid muscle, laterally by the medial surface of the ramus of the mandible, Lateral Pterygoid muscle superiorly, parotid gland posteriorly and the pterygomandibular raphe and the Superior Constrictor muscle of the pharynx anteriorly.
Source of infection usually from molar teeth especially lower third molars, it can also result after inferior dental nerve block with contaminated needle or solution. Infection can spread from submandibular, sublingual and infratemporal spaces. Swelling is minimal near the angle of the mandible or sometimes there is no swelling at all, but there is a marked trismus.
Site of incision and drainage extraorally, the same as that described in the masseteric space infections but directed to the inner surface of the ramus. Intraorally can be drained through an incision made just medial to the pterygomandibular raphe and dissecting along the inner surface of the ramus., but the presence of trismus can prevent efficient drainage.

Temporal space infection
Anatomic boundaries the Temporalis muscle divides this space into two spaces:
- Superficial temporal space; between the muscle and temporal fascia.
- Deep temporal space; between the muscle and the temporal bone.

The temporal space is contiguous with the pterygomandibular and masseteric spaces.

Source of infection upper and lower molars, or by extension from the other masticator spaces. The swelling is behind the lateral orbital rim and above the zygomatic arch, it is almost always associated with trismus.
Site of incision and drainage extraoral, through an incision superior and parallel to the zygomatic arch between the lateral orbital rim and the hair line. Intraorally this space can also be drained through an incision along the anterior border of the ascending ramus with the artery forceps directed upwards on the outer aspect of the ramus, but the presence of trismus makes this approach difficult.

Lateral pharyngeal space infection (parapharyngeal space)
Anatomic boundaries this space extends from the base of skull to the hyoid bone, it is conical in shape, the lateral boundaries include the medial surface of the Medial Pterygoid muscle, the medial wall is the Superior Constrictor muscle, Styloglossus muscle, Stylopharyngeus muscle and the Middle Constrictor muscle of the pharynx. Posteriorly by the parotid gland and anterirorly by pterygomandibular raphe. This space can be divided into two compartments; anterior and posterior, the latter contains the carotid sheath.
Source of infection spread of infection from upper and lower molar teeth, most commonly from lower third molar infections by the way of submandibular, sublingual and pterygomandibular spaces. A non-odontogenic infection can spread to this space like tonsillar infections. Infections of this space are serious, the patient exhibits pain, fever, chills, medial bulge of the lateral pharyngeal wall, extraoral swelling below the angle of the mandible and trismus. It may lead to respiratory obstruction, septic thrombosis of the internal jugular vein and carotid artery hemorrhage.
Site of incision and drainage intraoral incision medial to the pterygomandibular raphe with the dissection medial to the Medial Pterygoid muscle. Extraoral incision at the level of the hyoid bone anterior to the Sternocleidomastoid muscle (SCM) and the dissection continued superiorly and medially between the submandibular gland and the posterior belly of Digastric muscle. Through and through drainage can also be applied.

Retropharyngeal space infection
Anatomic boundaries extend from the base of the skull to the upper mediastinum (C6- T1), it is bounded anteriorly by posterior wall of the pharynx and posteriorly by the Alar fascia.
Source of infection upper and lower molar teeth by lateral pharyngeal space by the way of pterygomandibular, submandibular, sublingual spaces. It can also result from nasal and pharyngeal infections. The swelling causes bulge of the posterior pharyngeal wall, there is dysphagia, dyspnea, and fever. Lateral neck radiograph may reveal widening of the retropharyngeal space.

Site of incision and drainage extraorally by an incision anterior to the SCM below the hyoid bone, SCM and the carotid sheath are retracted laterally and blunt dissection is carried out deeply to enter the space. Some authors advocated intraoral drainage by an incision along the posterior pharyngeal wall in extreme Trendlenburg position and suction. Most anesthesiologists prefer tracheostomy to secure the airway.

Peritonsillar abscess (Quinsy)

Anatomical boundaries it is localized between the C.T. bed of the faucial tonsil and the Superior Constrictor muscle of the pharynx.

Source of infection it arises from tonsillitis, but it is occasionally a complication of pericoronitis of the lower third molar. It causes swelling of the anterior pillar of the fauces and a bulge of the soft palate of the affected side which may reach the midline and push the uvula. Also there is acute pain, dysphagia, the voice becomes muffled, odynophagia, drooling and anorexia.

Site of incision and drainage the incision is placed in the point of maximum fluctuation, this can be done under local anesthesia, if general anesthesia is used the anesthetist should be experienced and good suction be available to prevent aspiration and the patient should be in head down position.

Infections of spaces in relation to the upper jaw

1. **Upper lip infection**

   Infections of the upper incisors and canines can spread to the upper lip usually on the oral side of Orbicularis Oris muscle and points in the vestibule. Infection of the upper lip can lead to serious complications like orbital cellulitis or cavernous sinus thrombosis by extension of infection through the superior labial vein to anterior facial vein to ophthalmic vein to cavernous sinus. Incision for drainage is made near the vestibule intraorally.

2. **Canine fossa infections**

   Anatomic boundaries it lies between the canine fossa and the muscles of the facial expression.

   Source of infection mostly is the canine and first premolar but the infection can spread from upper incisor teeth. Infection occurs when it spreads in the area above the origin of the Levator Anguli Oris and is directed toward the medial edge of the Levator Labii Superioris. The swelling is lateral to the nose leading to obliteration of the nasolabial fold and may lead to periorbital cellulitis, there is risk of cavernous sinus thrombosis.

   Site of incision and drainage intraoral horizontal incision in the buccal vestibule.

3. **Buccal space infections**

   Infections spread from infected upper molar teeth where it spreads buccally above the attachment of Buccinator muscle. This space is already discussed

4. **Subperiosteal abscess in the palate**

   This potential space lies between the palatal mucoperiosteum and the underlying bone, the mucoperiosteum is strongly attached in the midline and at the gingival margin, pus may accumulate beneath the mucoperiosteum leading to its separation from the underlying bone.

   Source of infection it may spread from the apex of the lateral incisor which is close to the palatal bone. Also, infection can spread from the palatal root of multirooted upper molars. It can also originate from palatal periodontal pocket. the swelling causes palatal bulge between the gingival margin and the midline, confined to one side.

   Site of incision and drainage anteroposterior incision parallel to greater palatine vessels.

5. **Maxillary antrum Infection**

   Source of infection from upper molars and less frequently premolars may spread to the maxillary antrum, this depends on the size of the maxillary antrum and the length of the root.

   Signs and symptoms: It causes acute sinusitis with facial pain that worsens on bending or leaning forward, the infection may lead a chronic course leading to mucosal thickening and polyps. Occipitomental radiograph shows opaque maxillary sinus or fluid level.

   Site of drainage Pus may drain partially through the sinus osteum, extraction of the causative tooth leads to drainage of pus but it may leave a defect in the floor of the sinus and cause oroantral fistula. If the defect is small and with antibiotic treatment the socket may heal uneventfully, but larger defects may require further management.

6. **Infratemporal space infection**

   Anatomic boundaries this space is bounded laterally by the ramus of the mandible and the Temporalis muscle, medially by lateral pterygoid plate, superiorly by infratemporal surface of the greater wing of the sphenoid. It is traversed by the maxillary artery and contains pterygoid venous plexus. It represents the upper extremity of the pterygomandibular space.

   Source of infection directly from upper molar teeth or through contaminated needle from the pterygomandibular space. Infection may spread to the temporal space. There could be moderate swelling in the temporal region with trismus, usually the patient is toxic with high temperature. These infections are serious since they can spread through the pterygoid venous plexus to the cavernous sinus through emissary vein or it can spread to the middle cranial fossa with headache, photophobia, irritability, vomiting and drowsiness.

   Site of incision and drainage intraorally through an incision buccal to the upper third molar following the medial surface of the coronoid upward and backward, but with the presence of trismus this approach is difficult. Extra orally through an

incision in the upper and posterior edges of the Temporalis muscle within the hair line passing downward, forward and medially. Infection related to the maxillary teeth can spread to the masticator spaces and pharyngeal spaces and these were already discussed.

Cavernous sinus thrombosis

It is a very serious ascending infection, although not a fascial space infection but it can be caused by odontogenic infections especially of upper teeth. It can also result from upper lip, nasal and orbital infections.

Infection can spread to the cavernous sinus through two routes:

- Anterior route; through the valve-less angular vein and inferior ophthalmic vein.
- Posterior route; through the pterygoid venous plexus and transverse facial vein. This infection has a high mortality rate.

Clinical features:

- Marked edema and congestion of the eyelids and conjunctiva which can be bilateral due to the spread of infection to the other side.
- Proptosis (exophthalmos) and ptosis.
- Ophthalmoplegia and dilated pupil.
- Papilloedema with multiple retinal hemorrhage.
- Fever.
- Depressed level of consciousness.

Treatment:

It is an emergency that requires a neurosurgical consultation, the lines of treatment include:

- Antibiotic treatment
- Heparinization to prevent extension of thrombosis.
- Treatment of the odontogenic cause.

Principles of treatment of odontogenic infections

In treating odontogenic infections, the clinicians need to identify the presence of infection through the presence of the local and systemic signs and symptoms of the infection and whether it is cellulitis or abscess, also to determine the state of the host defenses, as these can be depressed by many factors such as; physiologic factors, disease related factors, immune-system related factors and drug suppression related factors.

Treatment of odontogenic infections requires medical, surgical or dental therapy or a combination.

Dental treatment; aims to eliminate the source of infection through endodontic treatment, periodontal treatment or extraction of the offending tooth. These factors should be taken in consideration:

- The extent of infection.
- Patient general health status.
- Degree of trismus.
- Biomechanical necessity of retaining the tooth.

Surgical treatment; aims to drain the accumulated pus to rid the body of the toxic purulent material and to relieve and decompress the tissues allowing better perfusion of blood to the infected area.

Methods of drainage

1. Through the root canal after access opening.
2. Through the socket by extraction of the offending tooth.
3. Through fenestration of alveolar bone using surgical handpiece and bur, made at the level of the root apex, to drain periapical abscess, after reflection of a semilunar mucoperiosteal flap.
4. Through incision and drainage of an abscess.

Incision and drainage

Is one of the oldest surgical procedures, it requires a thorough knowledge of facial and neck anatomy which is necessary particularly in draining deep tissue abscesses, on the other hand drainage of vestibular and dentoalveolar abscess is easily carried out.

Indications of incision and drainage:

1- When there are signs of accumulation of pus .
2- When the involved compartment is inaccessible; like pterygomandibular or pharyngeal spaces ,
3- Where is no improvement with adequate doses of antibiotic
4- Recurrence of pyrexia or a sudden increase in temperature and severe trismus .

5- Serious and rapidly evolving infections of the neck and floor of the mouth, like Ludwig's angina.

Principles of incision and drainage:

1. Incise in healthy skin and mucosa as possible,
2. incisions should be placed in an area of maximum fluctuation in a dependent position to encourage drainage by gravity.
3. Incisions made in areas where the tissue is necrotic or beginning to perforate may leave unaesthetic scars.
4. It should be placed in an esthetically acceptable area, in a natural skin crease or fold.
5. Incision in the skin of face and neck should include only the skin and subcutaneous tissues and the dissection through deeper tissues is continued bluntly using closed sinus or artery forceps which are advanced by controlled pressure to the pus containing tissue space and opened inside the cavity, then the forceps are drawn open then closed again and reinserted. The process is repeated until all the pus is evacuated. This is termed Hilton's method (after john Hilton, an English surgeon 1804-1878).

The forceps should not be closed inside the tissue to avoid damage to vital structures. Intraorally, in vestibular abscess, the pus accumulates under the mucosa with no intervening vital structures, so incision and drainage is made by the scalpel through the abscess cavity, here scalpel blade no. 11 is preferably used. Generally, all portions of the abscess cavity should be explored to ensure evacuation of all compartments, sometimes through and through drainage is necessary.

6. After incision and evacuation of pus a drain is inserted into the abscess cavity and is stabilized with suture. Corrugated drains or Iodoform ribbon gauze can be used for this purpose. Drains should not be left for long periods; they should be removed when the drainage is minimal.
7. Wound margins should be cleaned daily to remove clots and debris.

Note: Needless to say, that a sample of pus should be obtained and sent to the laboratory for culture and sensitivity, sometimes this can be achieved by aspiration with a syringe and needle prior to incision and drainage.

Medical treatment; it consists of supportive care which include hydration, soft diet, analgesics and good oral hygiene, and antibiotic therapy. It is essential to say that medical treatment is not a substitute for surgical treatment if indicated. In treatment of odontogenic infections,antibiotics are indicated therapeutically in the following cases:

1. Acute cellulitis.
2. Acute pericoronitis with elevated temperature and trismus.
3. Deep fascial space infections.
4. Dental infections in the compromised host.

Principles for the use of appropriate antibiotics

Once the decision has been made to use antibiotics as an adjunct to treat an infection, antibiotics should be properly selected, the following guidelines are useful:

1. Identification of the causative organisms, this is determined either in the laboratory where the organism can be isolated from pus, or empirically based on the knowledge of the pathogenesis and the clinical presentation of specific infections.
2. Determination of the antibiotic sensitivity.
3. The use of specific, narrow spectrum antibiotic, for example; if the causative organism is Streptococcus, sensitive to Penicillin, Cephalosporin and Tetracycline, then Penicillin should be selected because it has the narrowest spectrum. The main advantages of using narrow spectrum antibiotic are; less opportunity to develop resistance and minimize the risk of superinfection.
4. The use of least toxic antibiotic.
5. Patient's drug history should be known, especially drug allergy and various drug reactions.
6. It is imperative to know that the combat of the infection is the result of the host defense mechanism and that antibiotics are used as an adjunct to this process. Bacteriostatic antibiotics inhibit the growth and the reproduction of the bacteria usually by inhibiting protein synthesis, whereas bactericidal antibiotics kill the microorganisms by interfering with cell wall synthesis or nucleic acid synthesis. The main advantages of bactericidal antibiotics are:
   - Less reliance on the host resistance.
   - Kill the bacteria.
   - Work faster.
   - Greater flexibility with dosage interval.
7. The use of antibiotic with proven history of success.
8. Cost of the antibiotic should be taken into consideration.
9. Antibiotics should be administered in proper dose and proper dose interval.
10. Proper dose of administration either; oral (most commonly used), intramuscular or intravenous.
11. The antibiotic administration should be continued for an adequate length of time.
12. A combination of antibiotic therapy is indicated in:
    - When it is necessary to increase the antibiotic spectrum.
    - To increase bactericidal effect against specific organisms.
    - To prevent the rapid emergence of resistant bacteria.
    - Severe, rapidly progressing infections.

Most odontogenic infections respond to a combination of Penicillin and Metronidazole. Monitoring the patient in the follow up appointments the clinicians should look for the following:

- Response to treatment.
- Recurrence of infection.
- Presence of allergic reaction.
- Toxicity reaction.
- Secondary infections.

Failure of antibiotic therapy may be attributed to:

- Inadequate surgical treatment.
- Depressed host defense.
- Presence of foreign body.
- Problems associated with the patient and antibiotic, like lack of patient's compliance and inadequate dose...etc.

Sinus formation

When the abscess is not drained properly and neglected for a sufficient period of time, it will burst and drain spontaneously leading to sinus formation in an unfavorable site, the sinus is puckered, thickened and depressed.

If the source of infection is not treated the sinus will become chronic and it will be subject to exacerbations and remissions. During the active phase the sinus will exhibit signs of inflammation and may be tender while in the quiescence phase it heals over.

Sinus excision, after treatment of the source of infection, is carried out through an elliptical incision around the external orifice of the sinus, the tract of the sinus is followed bluntly to its source on the surface of the bone of the mandible. The resulting defect is closed in layers to eliminate dead space, the skin is closed with careful eversion to ensure an acceptable scar.

Necrotizing Fasciitis (flesh-eating bacterial infection)

Necrotizing fasciitis, known colloquially as flesh-eating bacterial infection due to the unique characteristic of the infection, does not obey the typical organization of the fascial planes of the head and neck.

First described by Pearse in 1938, who reported a 49% mortality rate, the progression of cervical necrotizing fasciitis does not follow the normal fascia planes of the head and neck. This is due to the unusually aggressive nature of the disease process. Often patients are immunocompromised, leading to this unusual and complex clinical course and extensive progression of an odontogenic head and neck infection.

Cervical necrotizing fasciitis is often polymicrobial in nature, is strikingly destructive, and is often fatal with a mortality rate of 7% to 20%. When the infection progresses to the thoracic region as a descending necrotizing mediastinitis, the mortality rate rises dramatically.

Management includes:

1- Removal of the offending source of the infection
2- Prompt, very aggressive debridement and removal of all affected soft tissues.
3- Creation of a surgical airway and continued intensive care unit monitoring and management.
4- Broad-spectrum empiric bactericidal intravenous (IV) antibiotics are generally always indicated in these cases because all involved tissues cannot be completely eradicated until specific culture and sensitivity results are available to guide specific antibiotic regimens.
5- Medical optimization of the patient

The disease process is characterized by the rapid spread of the infection on the superficial surface of the anterior (investing) layer of the deep cervical fascia deep to the platysma muscle.

Note: the necrosis of the platysma muscle and overlying skin due to thrombosis of the underlying muscles and soft tissues, as well as the dermal blood supply. The extensive tissue necrosis, including the skin, must be debrided thoroughly to halt the continued spread of the disease process.

بسم الله الرحمن الرحيم

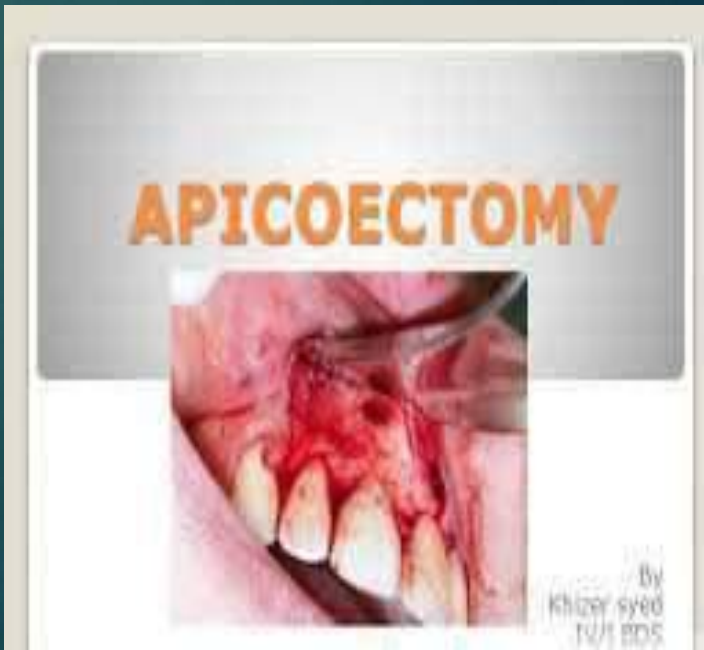
Endodontic surgery (Apicoectomy)

الدكتور: احمد عبدالكريم محمود

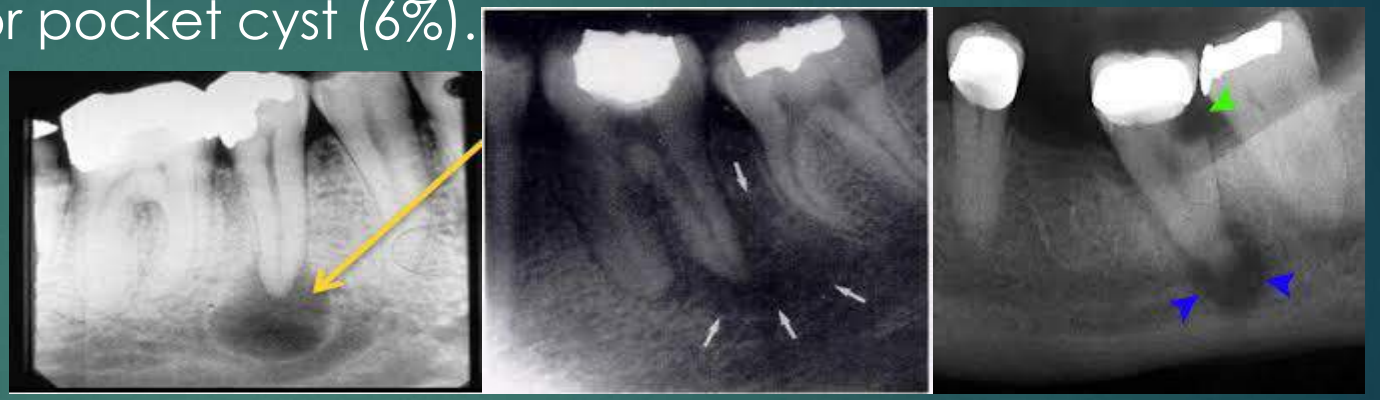
بكالوريوس طب وجراحة الفم والاسنان

دبلوم تجميل الاسنان والوجه

ماجستير جراحة الفم والوجه والفكين

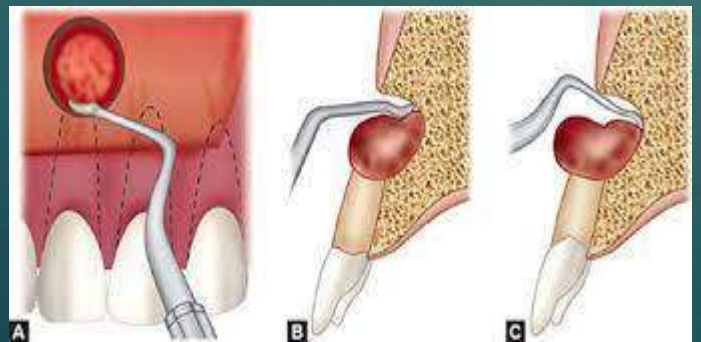


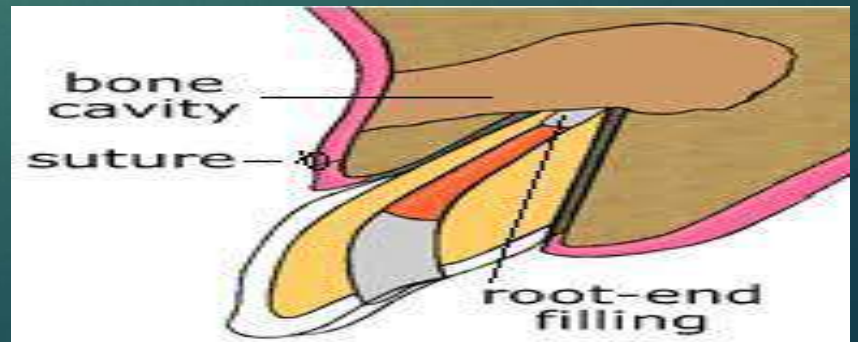
- The infected necrotic pulp tissue will produce periapical irritation through the apical foramen. The body attempts to combat this irritation by an inflammatory response. If the microorganisms are virulence will cause acute infection, while if the M.O. are non virulence or the host has good defense mechanism which will cause chronic infection of the periapical area. Chronic periapical infection may be a subsequent to acute periapical infection and there is acute exacerbation of chronic infection. Chronic periapical lesion reveals as abscess (12%), granuloma (73%), true cyst (9%) or pocket cyst (6%).



- Preoperative assessment:
  - Dental history (C.C., H.P.I.)
  - Medical history
  - Clinical examination (extra-oral & intra-oral examination)
  - Radiographic examination (Periapical x-ray, O.P.G,
- In acute periapical abscess the x-ray film reveals a slight or no evidence of periapical radiolucency, in chronic periapical abscess the x-ray film reveals a break in lamina dura at the apex of the root and there is irregular periapical radiolucency.
- In granuloma and periapical cyst there is also loss of the lamina dura and the apex is surrounded by rounded radiolucency, sometimes there is radio-opaque line encircle the radiolucent area in cystic lesion.

- In some cases there are failures of previous RCT or there are some obstacles to do RCT, so we must do Endodontic surgery which is apicoectomy and periapical curettage.
- Periapical curettage means the enucleation of the entire pathological lesion present in the periapical region using curettes and excavators.
- Apicoectomy means the amputation or resection of 3mm from the root apex. The resections of a portion of the apex are to:
- ✓ Facilitates the complete curettage of the pathological lesion present in the periapical area
- ✓ The removal of the apical accessory canals
- ✓ Enables the operator to check and ensure that the apical end of the root canal is effectively sealed under direct vision
- ✓ Survive a maximum amount of the bony support for the tooth, that not shorten too much from root length.
- For these reasons apicoectomy is still preferred by most oral surgeon at the present time, there are 2 types of apicoectomy: conventional (orthograde) apicoectomy, and retrograde apicoectomy.





- Indications of conventional apicoectomy

1. Severely curved root apex
2. Continuous drainage from a periapical cyst which prevent effective apical seal
3. Perforated root by wrong instrumentation technique.
4. Fracture apical 3rd of the root which when removed will leave a sufficient length of the remaining root to support the tooth
5. Incompletely formed root and not closed apex due to trauma and death of the tooth prior to complete closure of the apical area
6. When the patient cannot come for many visits and single visit RCT may failed.

- Indications of retrograde apicoectomy: the indications are the same as conventional apicoectomy with the addition of:

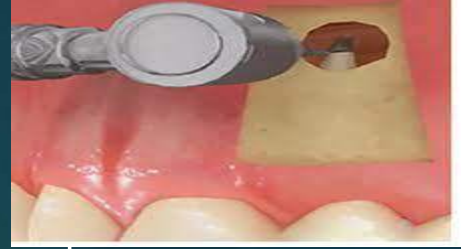
1. The root canal cannot be adequately cleansed and filled via the pulp chamber due to: presence of pulp stone, calcified canal, imperfect obturates root canal by gutta-percha(over or under extension root canal filling), fractured reamer, and the affected tooth is covered by a crown or a bridge.
2. Persistent postoperative discomfort after RCT or there is no obvious healing of periapical granuloma or small cyst fail to regress after RCT.
3. Limitation of mouth opening preventing access through crown of the tooth to do filling and obturation.

Contraindications of apicoectomy

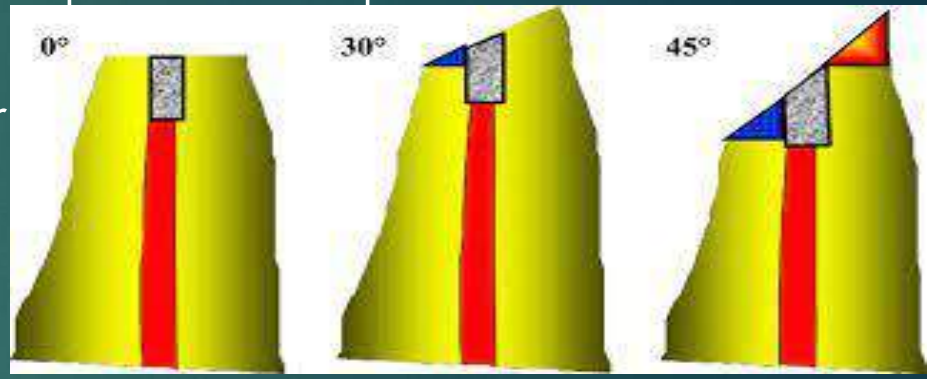
1. Presence of acute infection
2. Tooth mobility greater than 2 mm (grade 2) and more due to periodontitis because there is insufficient bony support after apicoectomy which need resection of the root apex (reduce root length) and need bone removal for good access which leads to more mobility of the tooth postoperatively. However, in some cases we can do fixation of such a tooth to neighboring healthy teeth for 3 weeks, or may add bone graft if the labial surface of the root is exposed and not covered by cortical bone due to periodontal disease
3. Unrestorable badly carious tooth or deeply fractured under gingiva
4. If the surgical operation may cause trauma to the adjacent teeth or vital structures such as nerves, nasal cavity or maxillary sinus.
5. Medically compromised patient

Surgical technique of Orthograde apicoectomy

1. Anesthetize the area for the width of 2 teeth on either side of the affected tooth or teeth
2. Do access opening on the cingulum of the non vital tooth and do reaming and filing of the canal until reaching a sufficient size according to the width and length of the root canal

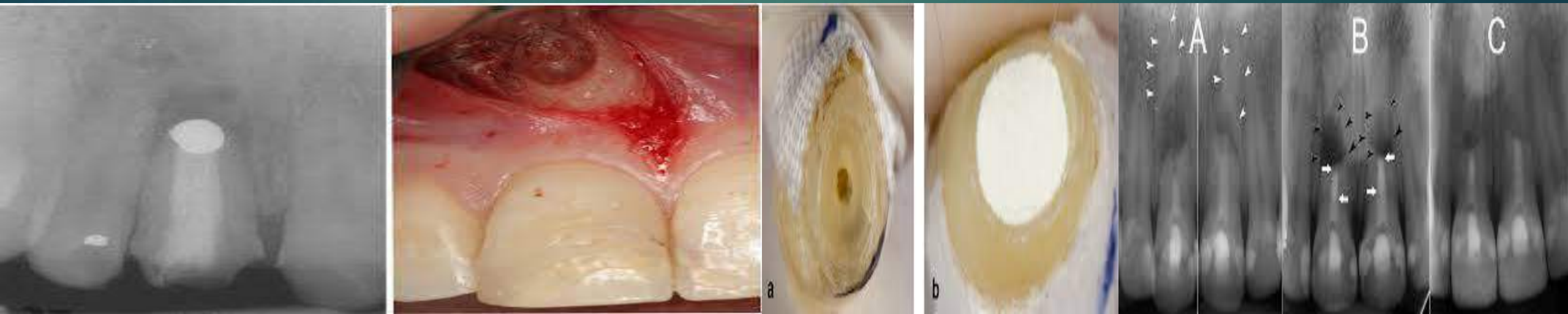


3. Design the mucoperiosteal flap whether semilunar, 2-sided or 3-sided flap
4. The mucoperiosteal flap is reflected carefully well above the apical area of the teeth by the use of mucoperiosteal elevator
5. The labial cortical plate near the apex of the non-vital tooth should be explored, a perforation may be already present in the cortical plate over the periapical area or explore the bony defect with the sharp end of cumine to locate the defect by penetrating the thin layer of cortical bone over it. Sometimes must make a small window with round surgical bur under copious normal saline irrigation over the estimates site of the apex on the labial cortex then the bony cavity is gradually enlarged until the apex is exposed with precaution not to damage the roots apices of the adjacent teeth



6. Enucleate the periapical lesion with curette and excavator
7. The root apex is exposed, resect 3 mm from its top utilizing fissure surgical bur, the cutting of the apex is oblique (bevel angle of 45-60 degrees) toward the labial surface
8. Filing using files and to see the tip of the files out of the root apex by direct inspection, irrigation of the root canal by normal saline through access opening then dry the canal with paper point and finally obturate the root canal with gutta percha and sealer also to see the tip of the gutta percha extend out of the root apex by direct vision, then with a hot ash cut and condense the gutta percha from access opening then cut the extended end of the gutta percha apically, then smooth the bony edge and root end with surgical bur. The success of endodontic surgery depends on the removal of all pathological tissue and complete sealing of the entire root canal.

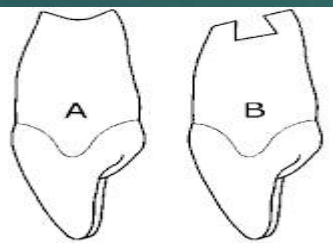
9. The periapical bone cavity should be irrigated with normal saline to ensure the area is clean and there are no bleeding areas
10. The flap is replaced and wound closure by sutures using 3/0 black silk suture, moist gauze compresses applied to the tissues after suturing to remove blood clots between the bone and soft tissues, to assure proper alignment of the flap and to reduce stress on the suture lines.
11. Give the patient postoperative instructions: apply cold pack extra-orally for the first 8 hours, prescribe antibiotic, anti-inflammatory, analgesic and mouth wash, inform the patient that the area may be slightly bruise and swell, instruct the patient to keep good oral hygiene by gentle brushing the surgical area, avoid smoking or eat hard foods, avoid lift his lip to examine the area this can loosen the stitches and disrupt formation of a blood clot that is needed for healing, the patient may complain from numbness in the area for days or weeks after the surgery, the numbness usually goes away with time, all soreness and swelling are usually gone within 14 days, and the sutures removed 7 days after the surgical procedure.



Surgical technique of retrograde apicoectomy

- The surgical steps are the same as in conventional or orthograde apicoectomy with the exception of not making access opening in the tooth crown, and after the curettage and resection of the apex in oblique line labially the following steps are done:

1. Create a class I cavity at least 3 mm depth into root dentine, with walls parallel to and coincident with the anatomic outline of the root canal space using low-speed angle hand piece and a small round bur
2. The bone cavity is packed with gauze or cotton and the prepared apical cavity is inspected to see that it is clean and dry
3. Fill the prepared apical cavity with a zinc-free amalgam using a special amalgam carrier, condenser and burnisher. Any excess amalgam is scraped away and remove the gauze or cotton from the bony cavity then irrigate and sucked out the cavity taking special care not to spread the amalgam into the cancellous bone or under the flap which leads to postoperative amalgam granuloma or tattoo. For those reasons, it was preferred that the prepared apical cavity filled with MTA, Biodentine, or calcium enriched mixture CEM. In comparison to amalgam as root-end filling materials, MTA consistently resulted in regeneration of periapical tissues including PDL and cementum (induce bone, dentin and cementum formation).



- TO PERFORM A BIOPSY OR NOT
- A clinical controversy has ensued over the consideration as to whether all periapical lesions treated surgically should have soft tissue removed and submitted for histologic evaluation.
- Certain guidelines have been set up for not to submit tissue for histopathological examination. These guidelines are:
- 1- Clear evidence of pre-existing endodontic involvement of a tooth (pulpal
- necrosis was present, not just a periapical radiolucency).
- 2- Unilocular radiolucency associated with apical one-third of the tooth.
- 3- Lesion is not in association with an impacted tooth.
- 4- No history of malignancy that could represent spread of a metastasis.
- 5- Patient will return for follow-up examinations and radiographs.
- 6- No tissue recovered at the time of surgery.

- Complications of endodontic surgery:
  - Intraoperative:

1. Bleeding; can controlled by using local application of adrenaline pack, pressure pack, Gelfoam or surgicel
2. Damage to the neighboring root
3. Entry into sinus/ inferior alveolar canal, nasal cavity

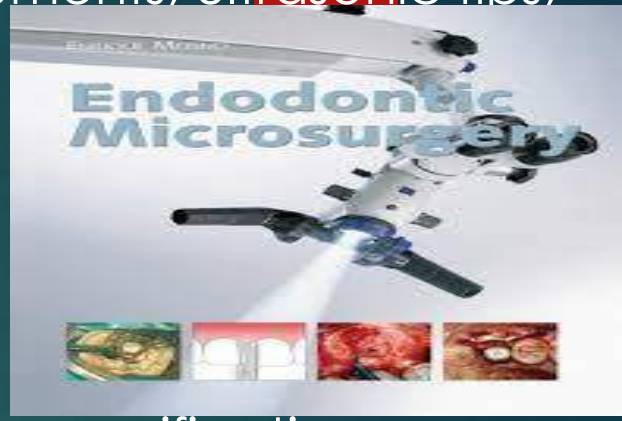
  - Postoperative:

1. Abscess formation
2. Fenestration, sinus tract formation
3. Increased tooth mobility

4. Staining of the mucosa due to amalgam that remained at the surgical field

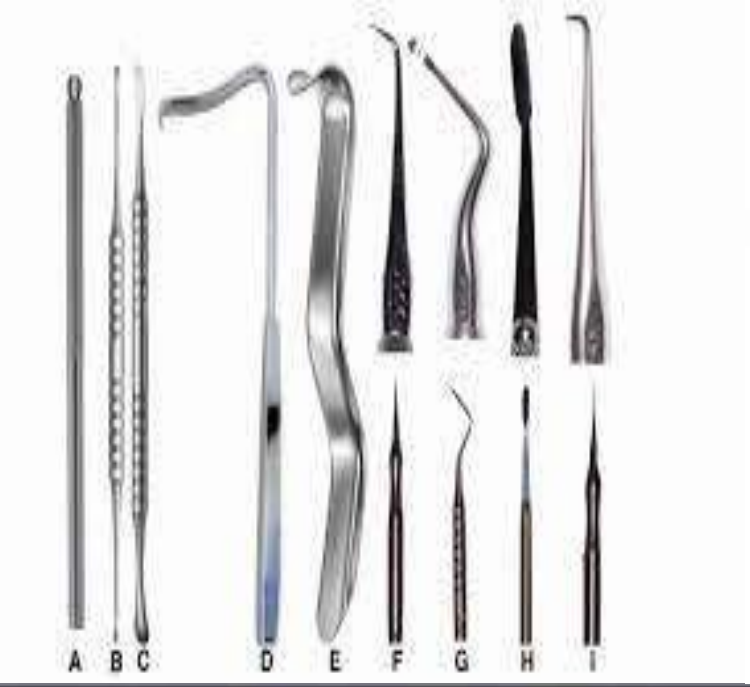
- Follow up for endodontic surgery
  - Healing of the periapical area is checked every 6-12 months radiographically, until ossification of the cavity is ascertained. In order to evaluate the result, a preoperative radiograph is necessary, which will be compared to the postoperative radiograph later.

- Endodontic microsurgery: microsurgical approach produce predictable outcome in the healing of lesions of endodontic origin by using microscope, micro-instruments, ultrasonic tips, and more biologically acceptable root-end filling materials



- Advantages of microsurgery versus traditional surgical technique:

1. The surgical field can be inspected at high magnification so that small but important anatomical details e.g. the lateral canals can managed
2. Removal of pathological tissue is precise and complete
3. Distinction between the bone and root tip can easily be made at high magnification, especially with methylene blue staining
4. At higher magnification the osteotomy can be made small as much as possible and this results in faster healing and less postoperative discomfort
5. Shallower resection angles (bevel angle of 0-10 degrees) that conserve cortical bone and root length
6. Combined with the microscope, the ultrasonic instruments permits conservative and precise root-end fillings
7. Occupational and physical stress is reduced since using microscope requires an upright posture
8. Video recordings of the procedure can be used effectively for education of patients and students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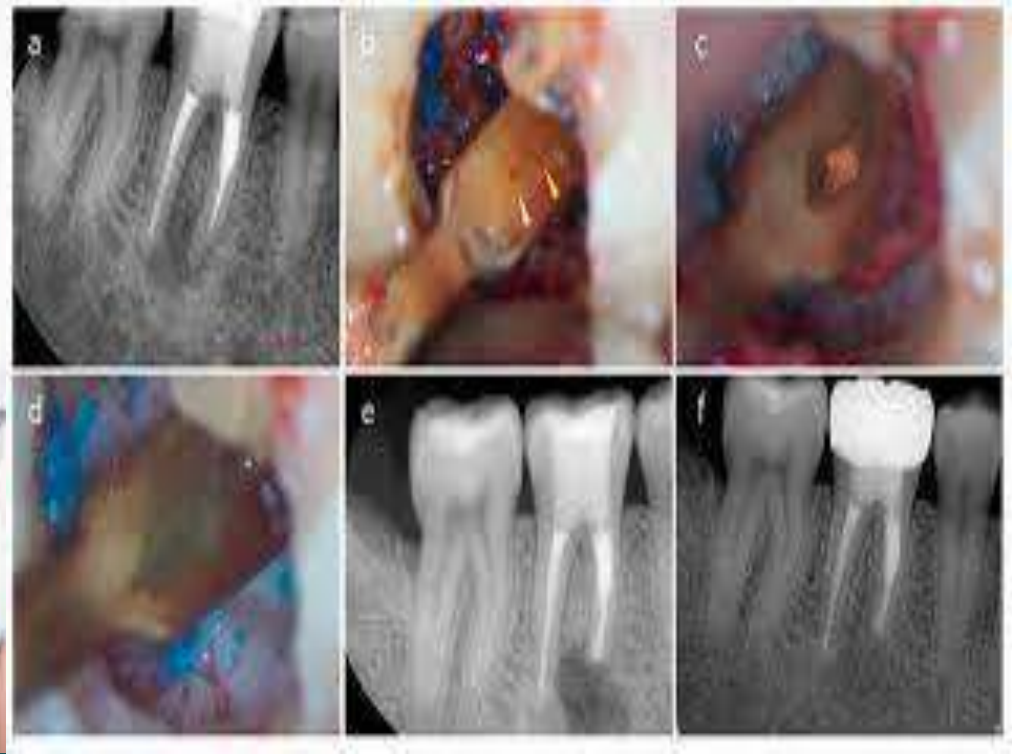



Thanks
a lot

Lec. 2 Biopsy in oral surgery

Dr.Hassanien Aljumaily

The term biopsy indicates removal of tissue from a living body for microscopic diagnostic examination. Biopsy is the most precise and accurate of all diagnostic tissue procedures and should be performed whenever a definitive diagnosis cannot be obtained using less invasive procedures. The primary purpose of biopsy is to determine the diagnosis precisely so that proper treatment can be provided, for many different lesions have similar clinical or radiographic appearances.

Indications for Biopsy:

1. Any persistent pathologic condition that cannot be clinically diagnosed
2. Any lesion that is felt to have malignant or premalignant characteristics
3. Confirmation of clinical diagnostic suspicions
4. Any lesion that does not respond to routine clinical management (i.e., removal of local irritant) over a 10- to 14-day period
5. Any lesion that is the basis of extreme concern to the patient (cancer phobia)

Characteristics of Lesions that Raise Suspicion of Malignancy

- Bleeding: lesion bleeds on gentle manipulation
- Duration: lesion has persisted more than 2 weeks
- Erythroplasia: lesion is totally red or has a speckled red and white appearance
- Fixation: lesion feels attached to adjacent structures
- Growth rate: lesion exhibits rapid growth
- Induration: lesion and surrounding tissue is firm to the touch
- Ulceration: lesion is ulcerated or presents as an ulcer

Prebiopsy Monitoring

Any undiagnosed or suspicious change in oral tissues that cannot be explained by localized trauma (and the source corrected) or other factors should be followed up in 7 to 14 days, with or without local treatment. If the lesion enlarges or expands, develops an altered appearance, or does not respond as expected to local therapy, then biopsy is usually indicated.

Biopsy or Referral

Clinicians vary in their surgical interests, training, and skills. Some dentists may feel comfortable performing many biopsy procedures on their patients, whereas others may refer their patients to other specialists. This is a personal choice and should take several points into consideration:

1. Health of the patient

Patients can be referred to specialists who are trained to deal with patients with special medical needs so that the procedure is carried out as safely as possible.

2. Surgical difficulty

If any of the basic surgical principles (such as access, lighting, anesthesia, tissue stabilization, and instrumentation) pose a problem if the dentist were to treat the patient, then referral should be considered. Similarly, as the size of a lesion increases or its position encroaches on significant anatomic structures, the potential for significant complications (e.g., bleeding and nerve damage) increases. Each dentist should use judgment when deciding whether the biopsy is within the dentist's surgical abilities or whether the patient would be better managed by a more highly trained specialist.

3. Malignant potential

The dentist who suspects that a lesion is malignant has two choices:

(1) Perform a surgical biopsy after completion of comprehensive diagnostic workup or

(2) Refer the patient before biopsy is performed to a specialist who is able to provide definitive treatment if the lesion is shown to be malignant. In such cases, it is better for the referral to specialist to evaluate the lesion before any surgical intervention which can compromised its clinical features and also to evaluate the patient before biopsy which helps to obtain a more accurate diagnosis and simplifies the formulation of a suitable treatment plan.

Post biopsy Monitoring

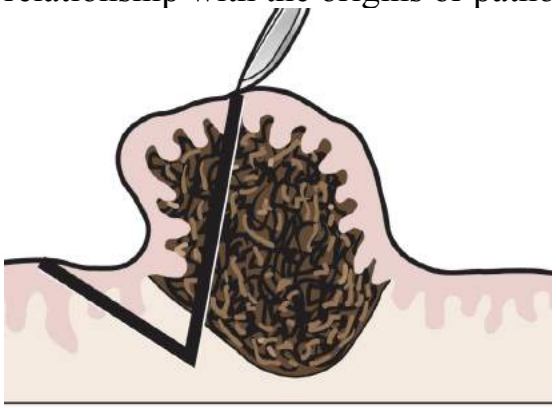
Following an incisional biopsy for diagnosis, a positive pathology report (indicating dysplastic changes or malignancy) generally mandates appropriate surgical excision of the lesion and contiguous tissues as indicated by the histopathologic diagnosis. This might necessitate referral to an oral-maxillofacial surgeon or other head and neck specialist, who is experienced in the management of malignancies.

A negative biopsy report, however, should never be taken at face value but interpreted with clinical and historical findings in mind. If doubt exists, a second biopsy might be indicated. At the very least, plans should be developed for a structured schedule of continued close observation at appropriate intervals. Generally, it is prudent to re-examine the patient within 1 month and then at 3, 6, and 12 months during the first year. Thereafter, if clinical and radiographic findings are unchanged, the interval between follow-up visits can be increased to 6 and then 12 months, as appropriate. Patients should always be counseled to contact the dentist immediately if any clinical changes or new symptoms are noted between visits.

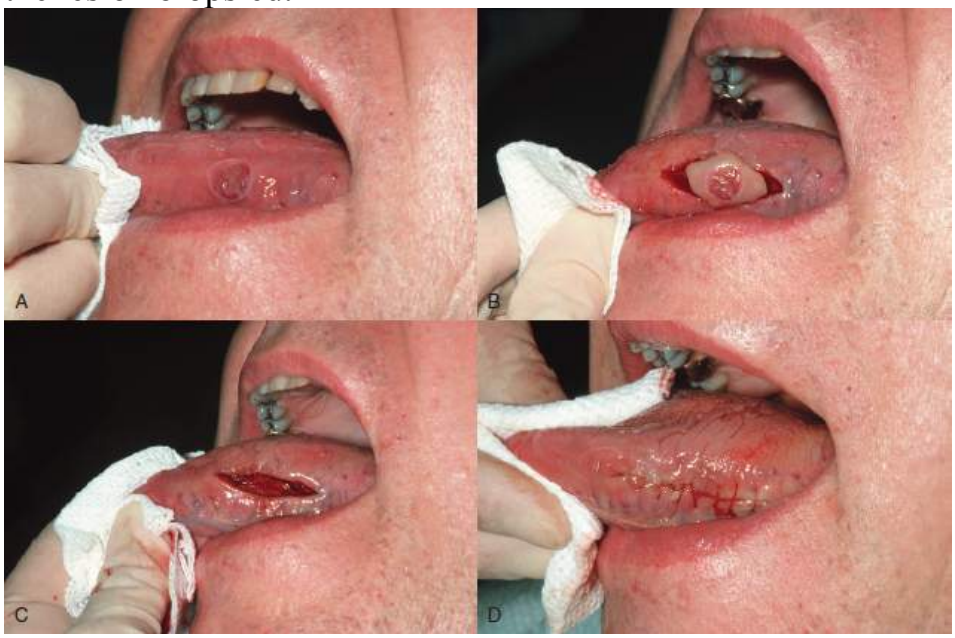
Biopsy types:

1. Surgical (incisional or excisional):

- Incisional: is a biopsy procedure that removes only a small portion of a lesion. Care must be taken to include an adequate depth of tissue as well so that cellular features from the base of the lesion are included. Generally, it is better to take a narrow, deep specimen than a broad, shallow one. Care should be taken not to compromise significant adjacent anatomic structures such as nerves and major blood vessels unless they seem to have a relationship with the origins or pathology of the lesion.



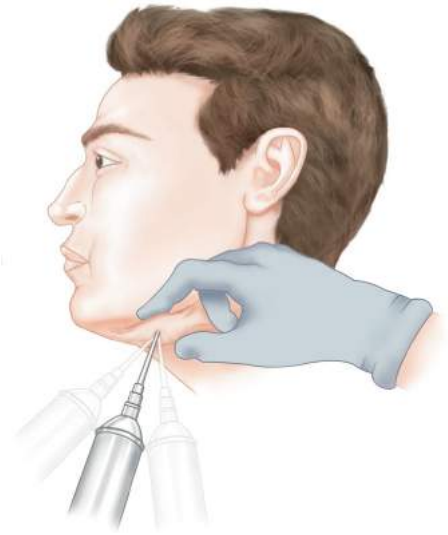
-Excisional: removal of a lesion in its entirety, to include a 2 to 3 mm perimeter of normal tissue around the lesion, *indicated for lesion sized 1 cm or less and it is clinically benign*. The width of the perimeter of normal tissue may vary, depending on the presumptive diagnosis. An additional 2 to 3 mm in tissues may be required for specimens suspected of malignancy, including some pigmented lesions and lesions already diagnosed as having dysplastic or malignant cells. Complete excision often constitutes definitive treatment of the lesion biopsied.



2. Aspiration biopsy: is performed with a needle and syringe by penetrating a suspicious lesion and aspirating its contents.

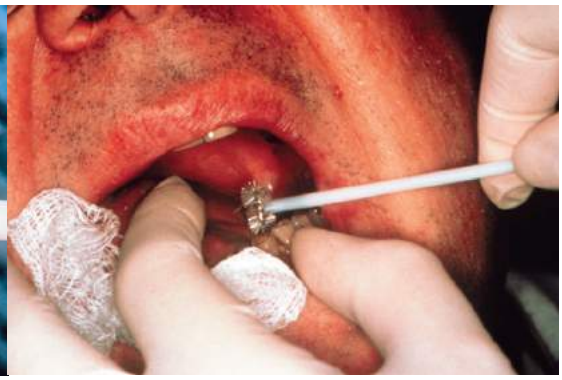
Two main types of aspiration biopsy in clinical practice are:
(1) Biopsy to explore whether a lesion contains a fluid; and
(2) Biopsy to aspirate cells for pathologic diagnosis. This latter is termed fine-needle aspiration (FNA). FNA is used when a soft tissue mass is detected beneath the skin or mucosal surface and the patient wishes to avoid a scar or adjacent anatomic structures pose a risk. FNA is an especially effective diagnostic tool for neck masses, from which it can be difficult to obtain a biopsy surgically. Routine aspiration of intra osseous radiolucent lesions is also performed before entering into the bony defect to rule out the potential of the lesion being vascular in origin and to define whether it is cystic or solid. Aspiration is performed on any fluid-filled lesion, except a mucocele. A 16- to 18-gauge needle connected to an aspirating syringe is used. The needle tip may have to be repositioned repeatedly in an effort to locate a suitable fluid-containing cavitation.
FNAB guided by ultrasound is a proven tool in evaluating the neck to confirm benign lymphadenopathy. Again, this occurred in a specialized center that had a particular interest in the process.

-Inability to aspirate fluid or air suggests that the intraosseous mass is probably a solid tumor.
-If straw-colored fluid is aspirated, the dentist is likely dealing with a cyst
-If pus is aspirated, an inflammatory or infectious process is likely present,
-if air without any fluid is suggestive of a traumatic bone cavity
-If blood is aspirated, several diagnoses must be entertained, the most significant of which would be a pulsatile vascular lesion within the jaw (e.g., hemangioma or arteriovenous malformation). Other vascularized intraosseous lesions, including aneurysmal bone cysts and central giant cell lesions, may passively (i.e., nonpulsating) produce blood on syringe aspiration.



3. Oral Cytology-Based Procedures
Noninvasive, cytology-based screening tests and modalities have many pitfalls and should not be considered substitutes for surgical biopsy. These tests are generally used as screening or follow-up adjuncts to careful clinical examinations. Two main forms of oral cytologic tests are available for use in clinical practice, depending on the method of collection or examination.
1. Exfoliative cytology: for examination of mucosal cells, which was first described and most commonly used diagnostic procedure for the detection of uterine cervical cancer. Although some authors have suggested oral applications, studies have consistently shown

that the results are not as reliable with keratinized oral tissues and often yield an unacceptable incidence of false-negative diagnoses, especially when the examining cytologists lack expertise in examining the unique nature of oral tissues. Furthermore, postoperative discomfort from a properly performed cytologic scraping can be more significant than if a surgical biopsy had been performed.
2. Brush cytology: (often imprecisely referred to as oral brush "biopsy"). This is a more recent development that is marketed heavily to general dentists. A handheld rotary wire brush is used to collect epithelial cells), which are then fixed on a glass slide and submitted for evaluation. Studies have shown this technique to be superior to exfoliative oral cytologic examinations.

4. Core needle biopsy:
It is the procedure to remove a small amount of suspicious tissue with a large core needle. It can remove more tissue than FNB.
5. Punch Biopsy
A small part of the lesion is obtained as specimen using a punch. This technique is of particular use in mucosal lesions from inaccessible regions that cannot be reached by conventional methods. The technique produces some amount of crushing or distortion of the tissues.

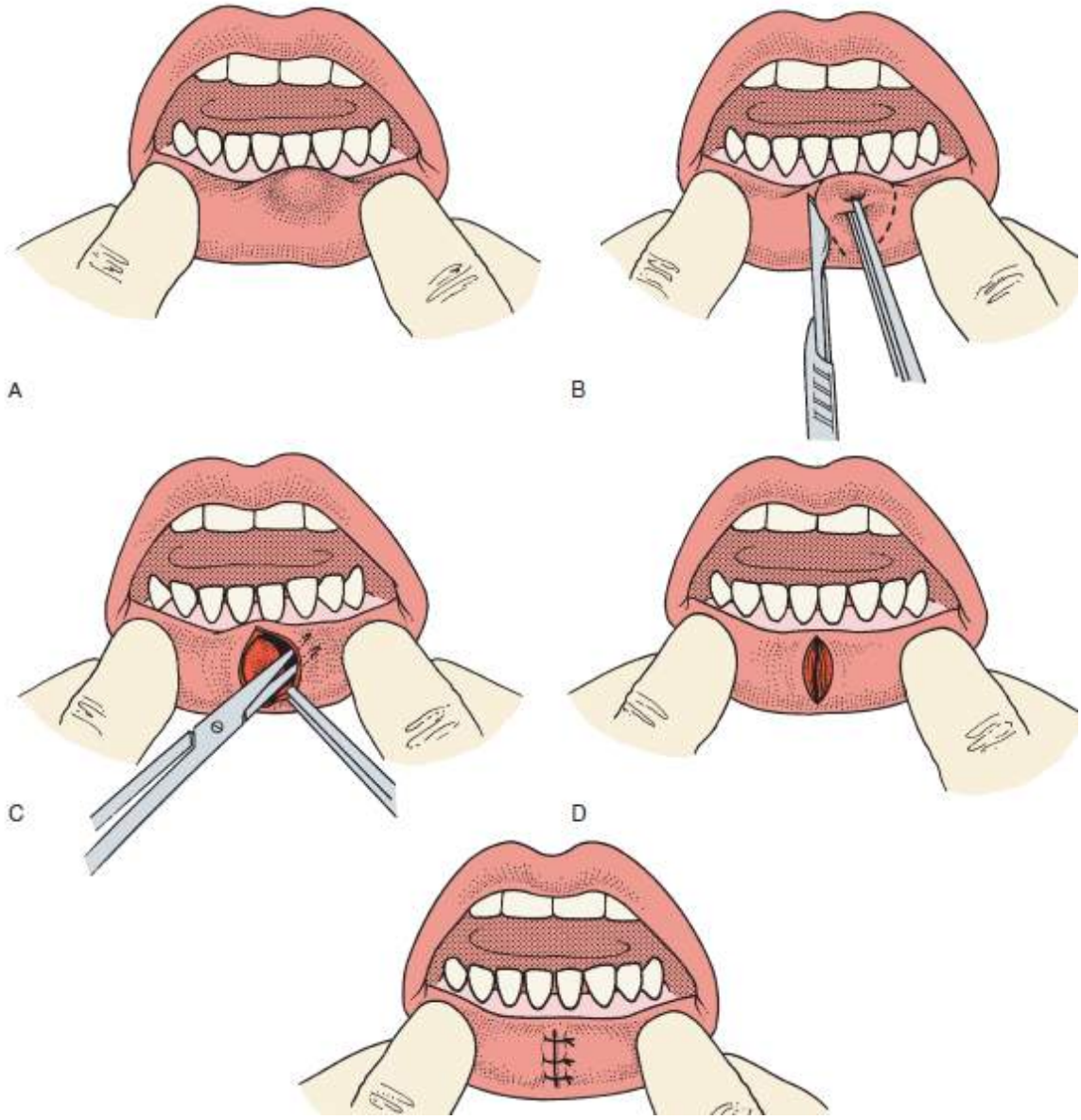
Soft Tissue Biopsy Techniques and Surgical Principles:

Anesthesia

Block local anesthesia techniques are preferred over infiltration, whenever possible, so that the anesthetic solution is not inadvertently incorporated in the surgical specimen. This can cause distortion of the cellular architecture of the specimen and make pathologic diagnosis more difficult, if not impossible. Peripheral infiltration of local anesthetic with a vasoconstrictor is often helpful, and it should be injected at least 1 cm away from the lesion perimeter to prevent tissue architectural distortions. The vasoconstrictor will decrease hemorrhaging in the wound and improve the surgeon's visibility of the site during surgery.

Tissue Stabilization

Oral and perioral soft tissue biopsies frequently involve mobile surfaces and structures (e.g., lips, cheek, soft palate, and tongue). Accurate surgical incisions can be placed with greater ease when the involved tissues are first stabilized. The surgical assistant can grasp the lips on both sides of the biopsy site with his or her fingers, which also retracts and immobilizes the lips. A variety of retractors are available that can perform the same function.



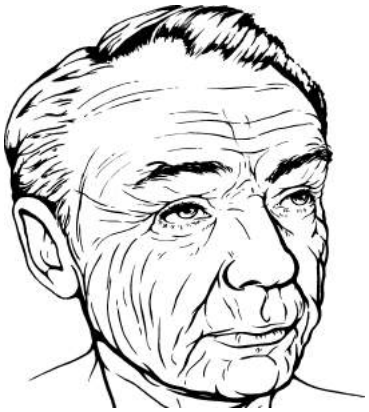
Hemostasis

The assistant can often use gauze sponges to blot the site. Suctioning not only can increase bleeding but also increases the risk of the biopsy tissue sample being accidentally aspirated

into the suction. If suction is needed, it is helpful to place gauze over the end of the suction tip to serve as a filter.

Incisions

A sharp scalpel, usually with a No. 15 blade, should be used to incise the tissues. To the maximal extent possible, incisions should parallel the normal course of nerves and blood vessels, as well as lines of muscular tension (i.e., smile lines and facial creases), to minimize secondary injuries and for esthetic reasons.



Wound Closure

Following removal of the tissue sample, primary closure of the wound is desirable and usually possible. If the wound is deep, incorporating different tissue layers, deep closure should be carried out for each layer, using a resorbable suture material

Handling of Tissues; Specimen Care

Any tissue specimen must be maintained in a condition that is optimal for preserving the histologic and structural architecture of the cells of the lesion. The specimen should be immediately placed in a glass or plastic container that contains a quantity of 10% formalin solution
(4% formaldehyde) that is at least 20 times the volume of the specimen itself

INTRAOSSEOUS (HARD TISSUE) BIOPSY

TECHNIQUES AND PRINCIPLES:

Often, the cause of jaw lesion is odontogenic, and the lesion will resolve once the dental problem is addressed. If the lesion appears to be unrelated to the dentition or does not respond to treatment of the presumed odontogenic problem, then the lesion should be removed for definitive diagnosis

Before performing intraosseous biopsy, the dentist should carefully palpate the area of the jaw and compare it with the contralateral side. Bone that has a normal contour and feels firm and smooth suggests that the lesion has not expanded or eroded the cortical plate of bone. However, a spongy feel when the jaw is compressed with the fingers usually

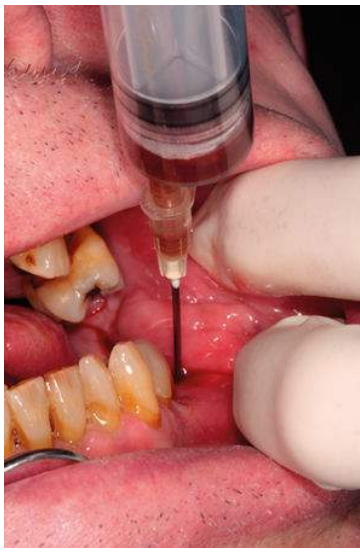
indicates erosion or thinning of the cortical plate, which suggests a more aggressive neoplastic lesion.

Mucoperiosteal Flaps

Because of their proximity to the jaws or their location within the bone, most biopsies require an approach through a mucoperiosteal flap. The flap should rest entirely on sound bone for closure, that is, extend 4 to 5 mm beyond the surgical margins of any bony defects. All mucoperiosteal flaps for biopsies in or on the jaws should be full thickness with the incisions transecting the mucosa, submucosa, and periosteum.

Precautionary Aspiration

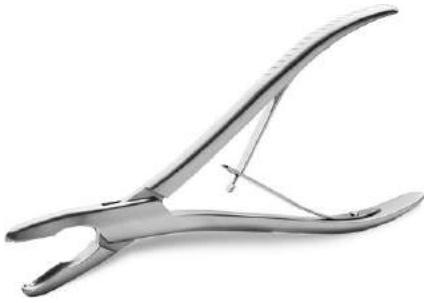
Aspiration of all intraosseous lesions should be performed routinely before opening into the osseous defect to determine whether it contains fluid, including blood., use a 16- or 18-gauge needle connected to a 5- or 10-ml syringe. If the cortical plate cannot be penetrated by pressing the needle firmly through the mucoperiosteum with a twisting movement, a flap is reflected and a large round bur, under constant irrigation, is used cautiously to penetrate the cortical plate.

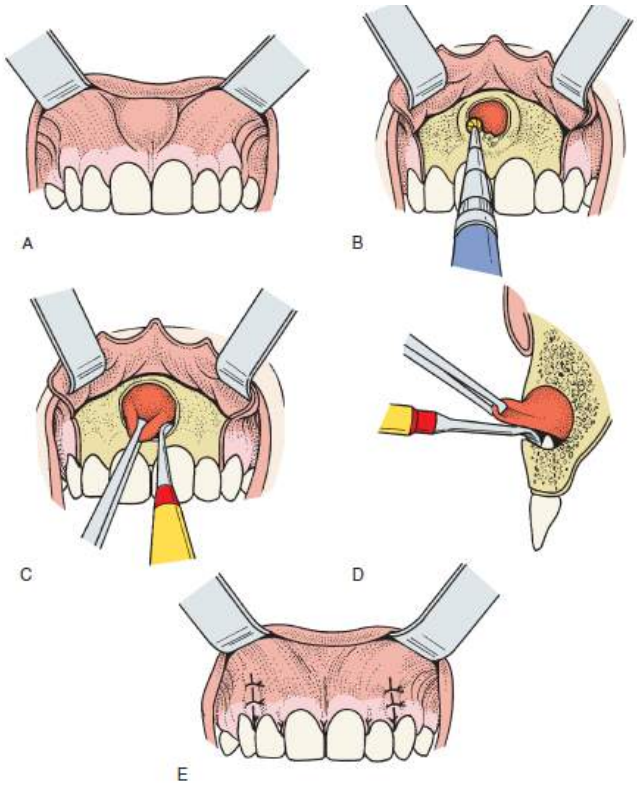


Inability to aspirate fluid or air suggests that the intraosseous mass is probably a solid tumor. If straw-colored fluid is aspirated, the dentist is likely dealing with a cyst, which can then be enucleated. If pus is aspirated, an inflammatory or infectious process is likely present, whereas aspiration of air without any fluid is suggestive of a traumatic bone cavity. If blood is aspirated, several diagnoses must be entertained, the most significant of which would be a pulsatile vascular lesion within the jaw (e.g., hemangioma or arteriovenous malformation). Surgical invasion into such a lesion could produce sudden, life-threatening hemorrhage and should not be attempted by the general dentist. Other vascularized intraosseous lesions, including aneurysmal bone cysts and central giant cell lesions, may passively (i.e., nonpulsating) produce blood on syringe aspiration.

Osseous Window

Intraosseous lesions of the jaws generally require creation of a cortical window for access. If the cortical plate is intact, a round surgical bur under constant fluid irrigation can be used to create an osseous window over the lesion site. The size of the window depends on the size of the lesion and the proximity of significant anatomic structures such as tooth roots and neurovascular structures. Once the window is created, it can be progressively enlarged with a rongeur, as necessary, for access. The removed bone that composed the window should be submitted along with the primary specimen if the lesion is a solid tumor.





Enucleation of a cyst. A, Mild swelling in area of periapical cyst. B, A mucoperiosteal flap is elevated from around the necks of teeth, and a bur is used to remove thinned cortical bone overlying the cyst. Care is taken to prevent rupturing of cystic contents during this and the following steps. C and D, A spoon-type curette is used to strip the cyst from bone. Note that the concave side of the curette is kept in contact with bone. The convex surface is the working end of the instrument. E, Closure

Specimen Management

Once the lesion is completely freed from attachment, it is removed and placed immediately into the formalin preservative. If possible, diagnostic radiographs should be included with the intraosseous specimen.
The pathologist should be provided with as much clinical information as. The pathology report may take 2 weeks or longer, if decalcification of osseous tissues is required before microscopic evaluation.

Postbiopsy Follow-Up
If the lesion is thought to be benign, then routine follow-up is carried out, with periodic radiographs to monitor osseous healing. If an incisional biopsy was performed, once the microscopic diagnosis becomes available, the patient should be re-evaluated and plans should be formulated for any required definitive treatment, referral for additional treatment, or both

ا.م.د. علي الحسيني

Diagnostic imaging in oral and maxillofacial surgery

Technological advances in the imaging science improve the accuracy in diagnosis, reducing radiation risk and progress the differentiation of non-osseous tissues. The application of these technical advances has assisted in the identification of an abnormality in terms of presence, site, size and nature of the lesion which aid for accurate diagnosis, also assisted in preoperative planning of surgical procedure and for evaluation of postoperative results. There are many diagnostic imaging modalities which are widely used throughout the world, from this clinician must select the right choice.

The imaging modalities classified into:

I. Invasive or interventional imaging

II. Non –invasive imaging

I. Invasive (interventional) and minimally invasive imaging

It is defined as a medical procedure which breaks the skin in some way and it may leave a scar. Mostly used for diagnostic as well as therapeutic purposes e.g. sinoscopy, arthroscopy, laryngoscopy, angiography, angioplasty, embolization, catheterization, delivery of cytotoxic drugs, calculus destruction, percutaneous biopsy and abscess drainage.

The main advantages of interventional procedure are that it gives palliative treatment with minimum morbidity and mortality and it also avoids open surgery in unfit patients.

II. Non-invasive imaging

It is defined as a medical procedure which doesn't break the skin. It is mainly used for diagnosis but it may also use for treatment as radiotherapy for the treatment of tumors.

Types of non-invasive imaging:

1. conventional radiography (Plain x-ray)

X-rays was discovered by Roentgen in 1895, and are now used in all forms of conventional radiography as well as CT scan. The scientific unit for measurement the radiation dose (effective dose), is the millisievert (mSv). Other measurement units include rad, roentgen, and sievert. The term effective dose is used when referring to the radiation risk averaged over the entire body. Different type of tissues produces different degrees of X-ray attenuation depending on their densities e.g.

*Air (lung) → radiolucent

*Calcified tissue (Bone) → radiopaque

*Soft tissue (muscle) → Gray

*Soft tissue (Fat) → Dark gray

Radiographical views:

1. Intra oral (periapical, bitewing, occlusal)
2. Extraoral (Posteroanterior "PA", Anteroposterior "AP", Cephalometric, Oblique Lateral., Occipitomental, OPG, Submentovertex, TMJ view, etc.).

 Periapical projection:

Provides excellent anatomical detail regarding bone quality. However, it does not show all areas and is a relatively high radiation dose/image.

 Occlusal projection (Anterior, Upper oblique and Vertex occlusal):

Evaluates larger area than the periapical radiograph, localization of the impacted tooth and can assess the buccolingual width of the mandible.

 Postero-anterior skull (PA):

Performed to evaluate the skull vault, primarily the frontal bones and the jaws, asymmetry of the face, developmental abnormalities, and fracture of the mandible and nasal septum.

 Postero-anterior of the jaws (PA jaws/PA mandible):

Used to assess fractures of the mandible, lesions such as cyst or tumor in body and ramus of mandible, condylar neck fracture, and condylar hypoplasia or hyperplasia.

 Occipitomental (Water's view, sinus projection):

Used to appraise paranasal sinuses (maxillary, frontal and ethmoid sinuses) and to detect middle third (Le fort I, II, III), orbital floor, and coronoid process fractures.

 lateral oblique mandible:

Utilized to examine the body and ramus of the mandible (lesions, fracture), impacted mandibular third molars.

 Lateral skull (Cephalometric):

Used to assess skeletal jaws relationship, pituitary fossa, fractures of the cranium and the cranial base, and frontal, sphenoidal and maxillary sinuses.

 Panoramic radiograph (Orthopantomogram "OPG"):

Achieved to examine the maxillary and mandibular teeth, bone lesions, fracture (mandible, condyles, coronoid processes), and to evaluate the floor of nasal cavity and maxillary sinus.

 Submentovertex projection:

Mainly utilized to assess zygomatic arches fracture. Furthermore, to measure the thickness (medio-lateral) of the posterior part of the mandible before sagittal split osteotomy, and to appraise destructive lesions affecting the palate, pterygoid region or base of skull.

 Reverse Towne projection:

Performed to assess condylar neck fractures, condylar hypoplasia or hyperplasia, and lateral or medial condylar displacement.

 Transpharyngeal projection:

Utilized to investigate the presence of TMJ disease, particularly osteoarthritis and rheumatoid-arthritis, ankylosis, cysts, tumors, and condylar neck fracture.

 Transcranial View of TMJ:

Performed to assess internal derangements of the joint and position of the disc.

 Sialography:

Used for detection of abnormalities in the salivary gland and ducts.

Advantages of conventional radiography

1. Easy to use
2. Relatively inexpensive
3. Available
4. High spatial resolution

Disadvantages of conventional radiography

1. Ionizing radiation (X-ray).
2. It gives two-dimensional record.
3. Requires multiple projections.
4. Superimposition of various structures in the pathway of X-ray in the region of interest.
5. Difficult to detect subtle pathology or trauma (insensitive until pathology is well established, at least 30% of mineral content change is required).
6. Poor visualization of soft tissues.

2. Ultrasonography (USG)

Ultrasound means high frequency sound waves above the limit of human audibility (>20 kHz)

USG procedure

A transducer (probe) converts electrical energy into high frequency sound waves, which pass into the tissues of different densities. The echo of US is reflected back to the scanning transducer, where converted into electrical energy, which are displayed on a monitor as images.

Reflection of US waves by various tissues:

*Fluid (Urine, blood and cyst) → all the waves are allowed to pass without reflection.

*Bone and air (lung) → all the waves are reflected back and not allowed passing.

*Soft tissue (muscle, fat) → waves are partly allowed to pass.

Tissues that have higher echogenicity are called hyperechogenic and are usually represented with lighter colors on images. In contrast, tissues with lower echogenicity are called hypoechogenic and are usually represented with darker colors.

Advantages of USG

1. Outdoor procedure (portable equipment)
2. Safe (no adverse reaction)
3. Non invasive
4. Painless
5. Not expensive
6. Easily repeatable

Disadvantages of USG

Since bone and air in lung completely reflect the US waves, so any intracranial and intrathoracic lesions can't be evaluated.

Doppler Ultrasound

It is used to detect and measure the rate of movement of any fluid such as blood. In "color Doppler" the direction of flowing blood is distinguished by different colors.

Applications or uses of USG

1. For detection of cervical lymphadenopathy.
2. Examination of various masses including thyroid gland, abscess, salivary glands.
3. Vascular abnormalities (Doppler US)
4. Aids in differentiation of solid or cystic masses.
5. Valuable for guided fine needle aspiration biopsy (FNA).

3. Computed tomography scanning (CT scan)

Godfrey Hounsfield in 1969 tries the first clinical application of CT in diagnostic imaging.

Tomography = Tomo (slice), graph (picture)

Early CT scanners took several minutes to acquire a single slice. In contrast, modern scanner can acquire 64 slices, 128 slices, and 620 slices in 10-50 seconds (scan time) by use of advanced powerful computers. Slices thickness of 0.5-3 mm provide very good detail of the tissues. The standard plane of CT scan is obtained in axial plane. The X-ray attenuation values of CT scan are scored from -1000 to + 1000 Hounsfield Units (HU).

It reveals: hypodense or hyperdense.

* Air (in the lungs) = -1000 HU
* Fat = -30 to -70 HU
* Water = 0 HU
* Muscle and Soft tissue = 20 to 40 HU
* Cyst, fluid and blood = 35 to 50 HU
* Bone = 300 HU cancellous bones to 1000 HU and more for dense cortical bones and tooth enamel.

The Hounsfield scale of CT is set around water measuring 0 HU and that for air is −1000 HU. Fat represents an excellent contrast in CT scan. However, the use of contrast agents can artificially increase the resolution of CT scan. Thus, intravenous contrast media (iodine) will enhance blood vessels resolution, and oral contrast (barium) will delineate bowel.

Types of CT scan:

Computed tomography can be divided into 2 categories based on acquisition x-ray beam geometry; namely: fan beam and cone beam.

* Spiral CT

This type of CT obtains a volume of contiguous slices (128-620 slices) as thin as 0.625mm in thickness within seconds. Spiral CT has many advantages over conventional or traditional CT:

1. It minimizes motion artifacts because the entire scan time can be performed during a single breath hold.
2. It reduces the X-ray dose to the patient.
3. It improves spatial resolution by giving 3D pictures.
   Sequential slices of CT scan data are stacked layer upon layer for obtaining 3D information. The 3D reconstruction is simple, since pre selection views are automatically generated; it takes 20-30 minutes. The 3D CT very useful because the surgeons can assimilate complex anatomic relationships from these images which look like anatomic dissection, so facilitate surgical planning and reduce operation time.

* Cone Beam CT (CBCT)

Applicated in dentistry since 1998. Cone Beam CT has many applications in oral and maxillofacial surgery:

- Used for preoperative planning for the placement of dental implants.
- Alveolar bone height and width can be measured easily from point to point. Selection the proper length and diameter of the implant can be easily done.
- Quality and quantity of bone at the proposed sites can be assessed.
- Precise relationship between the impacted tooth and the adjacent structures can be achieved.
- Giving 3D image and exposing the tissues to relatively small doses of radiation.

Radiation dose:

Conventional dental radiography = 1-20 μSv

CBCT = 20-500 μSv = 3-28 OPG

Medical CT = 200-2000 μSv = 33-82 OPG

Technique	Effective dose (µSv)
Conventional dental radiograph	1-20
CBCT	20- 500
CT	200-2000

X-ray	Effective dose (µSv)	Equivalent of natural background radiation in UK (2200 µSv/ year)
Intraoral radiograph	< 1.5	< 1 day
Cephalometric radiograph	< 6	1 day
Panoramic radiograph	2.7-24	< 1-4 days
CBCT (dentoalveolar)	11-674 (median=61)	2 days to 4 months (median 10 days)
CBCT (craniofacial)	30-1073 (median=87)	5 days to 6 months (median 14 days)
CT	280-1410	2-8 months

Advantages of CT scan

1. Noninvasive.
2. Quick imaging, scan time less than 1 minute, so reduced patient motion artifacts.
3. Evaluation of bony structure and soft tissue.
4. Contrast resolution of CT is much better than conventional x-ray.
5. It gives 3-D image of the body, so it gives the exact site, size of the lesion and its relation to adjacent normal structures.
6. CT differentiates various body structures from each other and demonstrates their relationship (no superimposition).
7. Used for pre- and postoperative assessments.

Disadvantages of CT

1. Ionizing radiation (x- ray)
2. Expensive
3. Children require sedation (claustrophobia).
4. Respiratory motion or body motion leads to degradation of the image.
5. Allergic reaction to contrast medium (iodine).
6. Artifact scatters due to presence of dental amalgam fillings and metallic crowns.
7. Radiation dose of medical CT exceeds that of CBCT & conventional panoramic radiographs.

Applications or uses of CT scan

1. Head and facial skeleton: fractures, osteolytic lesions, and hyperostosis.
2. Intracranial lesions: hematoma, infarction, abscess, and tumors.
3. Intraoral mass, paranasal sinuses, larynx, nasopharynx and salivary glands.
4. Cervical lymphadenopathy and neck masses.
5. Guiding the placement of FNA for biopsy.
6. Angiography (using contrast agent).

4. Magnetic resonance imaging (MRI)

Lauterbur in 1972 used MRI for the first time for medical imaging. Ogawa in 1990 discovered that MRI can be used to distinguish oxygenated blood from deoxygenated blood and named as functional magnetic resonance imaging (fMRI). The principle of MRI is based on the magnetic properties of hydrogen protons (body tissues that contain hydrogen atoms).

T1 and T2 are two distinct views which are typically generated:

In T1-weighted image; water appearing darker and fat brighter.

In T2-weighted image; fat shows darker and water brighter.

It reveals as hypointense or hyperintense

Advantages of MRI

1. Non invasive
2. Non ionizing radiation
3. Superior soft tissues contrast
4. High quality images and can select any plane.
5. Decreases the artifact scatter seen in CT due to dental fillings and metallic crowns.
6. Better differentiation of fluid from soft tissue.
7. Very accurate in detecting small tumors.

Disadvantages of MRI

1. Bone imaging limited to display of marrow, because lack of signals from cortical bone.
2. Motion artifacts due to long time procedure (about 30-60 min. full scan).
3. Expensive
4. Claustrophobia
5. Very loud continuous hammering noise when operating.
6. Powerful magnetic fields can dislodge or interfere with some metal medical devices e.g. pacemakers, ferromagnetic aneurysm clips, metal implants etc. These are absolute contraindications.
7. Tissues containing low number of suitable hydrogen protons e.g. aerated lungs, dense bone, which is poorly visualized. In such areas, CT is better in investigation.

Applications or uses of MRI for head and neck lesions

1. To define the extent of soft tissue tumor and its relation to the adjacent soft tissues or blood vessels.
2. To distinguish fluid from tumor in an obstructed paranasal sinus.
3. To evaluate cranial neuropathies.
4. To detect meningeal disease of head and spine whether inflammation or tumor.
5. Evaluate possibility of recurrent tumor.
6. Evaluate larynx, nasopharynx, and paranasal sinuses
7. To evaluate areas where artifacts may degrade the CT image, e.g. dental amalgam filling and metal crowns in mouth.
8. Guiding the placement of FNA for biopsy (use non-ferromagnetic needles)
9. Angiography.
10. Evaluation of the TMJ disc deformities.

MRI vs. CT scan

1. Resolution of soft tissue in MRI is superior to CT.
2. Resolution of bone is better with CT than MRI, because there is essentially no MRI signal from dense cortical bone.
3. The ability of MRI to differentiate a nerve from fat and CSF makes MRI the procedure of choice to visualize the anatomy of cranial nerves above and below the skull base.
4. For contrast enhancement, CT uses iodinated contrast that can be harmful in patients allergic to the contrast agent. While in MRI there is rare reaction to the contrast agent (gadolinium) which is used to enhance the resolution. The ability of MRI to change the contrast of the images; small changes in radiowaves and magnetic fields can completely change the contrast of the image. Different contrast settings will highlight different types of tissue. So, MRI is excellent replacement to CT in such patients since it is able to differentiate soft tissues and blood vessels without use of the contrast.
5. Unlike CT scan, MRI does not use ionizing radiation.
6. MRI used to evaluate areas where artifacts may degrade the CT image, e.g. dental amalgam in mouth.
7. CT can be used in patient with metal in his body, while MRI can't be used (dislodgement of ferromagnetic device).
8. CT is less expensive than MRI (CT about half the price of MRI).
9. Limited slice thickness in MRI which is 3mm, while 0.6-1mm in CT.
10. In MRI there is motion artifact due to long time procedure (CT full scan takes 5-10 minutes, while MRI full scan takes 30-60 minutes).
11. CT seldom creates claustrophobia, while in MRI; creates anxiety, claustrophobia, annoyance from the sound and the long period of scan time.
12. For purposes of tumor detection and follow-up, MRI is generally superior. While for emergency cases CT is more suitable.

5. Radionuclide (scintigraphy or skeletal scan)

Radionuclide or called radioactive isotopes is a chemical element which emit a type of radioactivity called gamma rays. It is minimally invasive method done by intravenous injection of small amounts of radioactive isotopes such as ^{131}I (Iodine) ^{99m}TC-P (Technetium labeled Phosphate) or ^{67}Gallium is used. Uptake of the radionuclide is monitored with gamma camera, in which the detectors can rotate 360^{o} around the patient, acquiring up to 128 images in different angulations to study the metabolic activity of bone.

Areas of tracer elements uptake are known as:

1. Hot lesions (hot spots): Areas that absorb more tracer elements emit lots of gamma rays may appear as red spots or "hot spots", as a picture on the computer which suggest increase metabolic activity as normal bone growth, repair, osteoblastic activity and increased vascularity, infection and cancer.
2. Cold lesions (cold spots): Areas that absorb little or no amount of tracer element emits low level of gamma rays and appear as blue or "cold spots", which suggest in decreased metabolic activity, tumor metastases, and decreased vascularity. Various other colors may be used for "in between" levels of gamma rays emitted.

6. PET Scan

Positron emission tomography (PET) is a technique that can detect a number of positrons emitting radionuclide and therefore can be used to study a variety of metabolic processes in an organ or lesion. F-18 fluoro-2-deoxy-glucose (F-18 FDG) is the most commonly used positron emitting radiopharmaceutical for PET imaging. The disadvantage of PET is that limitation to provide information in the exact localization of lesion because of absence of precise anatomic landmarks.

7. PET-CT

It is a hybrid system, more useful for detecting and grading tumors, monitoring response to therapy and postoperative recurrence.

8. SPECT Scan

Single Photon Emission Computed Tomography (SPECT) is another type of scintigraphy, but SPECT suffers from relatively low spatial resolution so that they are not effective in detecting subtle abnormalities like small tumor recurrences.

Advantages of scintigraphy

1. Used for diagnosis and treatment
2. Isotopes are available
3. Not expensive
4. No adverse effect (radiation risk is very low compared with the potential benefits).

Disadvantages of scintigraphy

1. Limitation to provide exact localization of the lesion (overcome by hybrid system)
2. Not effective in detecting subtle tumor.
3. Radiation risk; not preferred for women who are pregnant and breast-feeding a child.
4. Long time procedure (it takes several hours for the radiotracer to accumulate in the body before imaging).

Applications or uses of scintigraphy

1. Dynamic method; for growth assessment of facial skeleton and to assess cessation of growth and fix the time for corrective orthognathic surgery.
2. To survey the presence of primary bone tumor, osteosarcoma, Ewing's sarcoma, and metastases.
3. To evaluate the extent of osseous inflammation and infection.
4. To evaluate the abnormal metabolism or growth in the skeleton (metabolic disorder).
5. To study the fate of bone grafts, rejection, vascularity, viability (bone graft follow-up).

Surgical aids to orthodontics

Surgery in the orthodontic patient will often be an integral part of a treatment plan. Surgical interventions that may be required in orthodontic treatment include: extraction of erupted teeth, management of impacted, supernumerary and dilacerated teeth, corticotomy and excision of labial frenum.
The dental and medical history, clinical examination, radiographs and consultation with the orthodontist are necessary for preoperative assessment and surgical treatment plan.

The most conditions which require surgical aids prior to orthodontic treatment are:

1. Management of Impacted teeth

Impacted tooth is one that has failed to erupt into normal functional position beyond the time usually expected for such appearance.
It occurs where there is prevention of complete eruption due to:

- Lack of space in the dental arch (main cause).
- Obstruction by another tooth.
- Development in abnormal position.
- Dense overlying bone.
- Thick fibrous tissue.
- Odontogenic cysts or tumors.

The mandibular third molar is the most commonly impacted tooth in the mouth followed by maxillary third molar, maxillary canine, mandibular canine, mandibular second premolar, maxillary second premolar and second molars.

Impacted third molars

The prevalence rate of third molar impaction varies from one population to another ranging from 16% to 68%. The presence of impacted third molars may interfere with orthodontic treatment.
The most frequently required option of treatment in orthodontic patients is surgical extraction of impacted lower third molar.

Clinical examination

After taking a thorough medical and dental history, with a special consideration to the age since it may significantly influence the management of impacted third molars, the clinical examination includes general assessment of the size and the build of the patients, patient's attitude is important as this reflects the way they will respond to the stress of surgery. General fitness is essential.

Intraoral examination should include: the mouth opening, oral hygiene, examination of the third molar, if it is visible or not, carious, the condition of the soft tissue over the impacted tooth is noted; if there are signs of pericoronitis treatment will be required before the operation, the position and the condition of the upper third molar is checked and its relationship with the lower third molar.

Radiographic examination

The radiographic examination should demonstrate:

- ✓ The whole impacted tooth.
- ✓ The investing bone.
- ✓ The adjacent tooth.
- ✓ The inferior dental (alveolar) canal.
- ✓ The anterior border of the ascending ramus.
- ✓ The relation of the maxillary third molar with the maxillary sinus

Periapical radiograph offers the highest definition and it should be used whenever possible, but the film should be positioned with care.

Orthopantomogram (OPG) is regarded as the radiograph of choice, its main advantages are; the low radiation exposure and its ability to demonstrate the entire dental arch.

Occlusal films are used in difficult cases especially in unerupted teeth in conjunction with another view at right angle, this is necessary to understand the problem in 3 dimensions.

Cone beam CT (CBCT) is indicated when on OPG there is a suggestion of a relationship between the roots of the impacted tooth and the inferior alveolar canal or the maxillary sinus or when the impacted tooth is associated with pathology.

In radiographic assessment the following features should be considered:

- ➢ Angulation of the impacted tooth.
- ➢ The depth of the impacted tooth.
- ➢ Crown features.
- ➢ Root morphology, e.g. fused roots, curved roots …etc.
- ➢ Bone density.
- ➢ Any associated pathology like cysts or tumors.
- ➢ The state of the second molar (root morphology, caries, restoration or resorption …etc.)

Surgical extraction of impacted third molars

Surgical extraction of impacted third molars is commonly done under L.A., other choices include L.A. with sedation and G.A. access is achieved either by an envelope flap or a triangular flap, which is an envelope flap with a vertical releasing incision. After flap reflection bone removal with or without tooth sectioning is needed to extract the tooth. After tooth removal any debris and the remaining follicular tissue are cleaned out, sharp bone smoothed, copious irrigation of the socket and underneath the flap is carried out and the flap is repositioned and sutured.

Complications

- Pain, edema and trismus.
- Postoperative hemorrhage.
- Infection and abscess formation.
- Alveolar osteitis (dry socket).
- Localized osteomyelitis.
- Temporal or permanent nerve dysfunction of the inferior dental nerve and lingual nerve; this is manifested as anesthesia, paraesthesia or dysethesia. Recovery of normal sensation may take few days to several months.
- Fracture mandible, uncommon, due to applying excessive force during extraction.
- Loss of the tooth or the root into the lingual space.
- Oroantral fistula which should be repaired as soon as possible.
- Displacement of tooth or root into the maxillary antrum or infratemporal fossa.
- Fracture of tuberosity.

Impacted maxillary canine

Maxillary canines are impacted in about 1%-3.5% of the population; the rate among female patients is twice as high as that in males.

If the maxillary canine fails to erupt by the age of 13 years, its position should be investigated, the orthodontist should be consulted to determine if the tooth can be brought to normal occlusion. The impacted maxillary canine can be located; palatally, buccally, within the alveolar ridge, or located labially and palatally; crown on one side and the root on the other side. It can also be horizontally, vertically or semivertically oriented.

Clinical examination

The position of the tooth may be obvious by the presence of a bulge either palatally or buccally. The palatal impaction is more common than the buccal one.

Palpation of the maxilla through the labiobuccal sulcus may reveal the presence of the bulge buccally. The lateral incisor may be proclined due to the presence of the canine labial to the root or may be retroclined if the canine is palatal.

Radiographic examination and assessment

The radiographic views that can be used include; periapical, occlusal, OPG, lateral skull view cone beam CT (CBCT) can also be used.

The periapical radiograph provides a detailed view of the tooth, surrounding bone, root formation, the presence of root resorption of the adjacent lateral incisor or the presence of any pathology.

Localization of the canine is important especially when it cannot be determined clinically. Methods of localization include:

- **Buccal object rule (parallax method, tube shift technique)**; in which two periapical films are taken, shifting the tube horizontally distally between exposures, if the unerupted tooth moves in the same direction in which the tube is shifted it is localized palatally, if it moves in opposite direction it is buccally located, in a rule called SLOB (Same Lingual Opposite Buccal).
- **Vertex occlusal projection**; which produces an axial view of the incisors, will demonstrate the buccopalatal localization of the canine.
- **Periapical-occlusal method**; uses a standard periapical view and an occlusal view to give two different views of the impacted tooth.
- **OPG**; can be used to localize impacted canine on the basis that palatally impacted canine appear magnified. It can also demonstrate the vertical angulation and its height.
- **Lateral skull view or cephalometric.**
- **CBCT.**

Options of treatment

1. **Retention or leave in situ**; indicated when:
   - ✓ The canine is asymptomatic and its extraction may lead to damage to the adjacent teeth.
   - ✓ There is absence of any pathology like infection, abnormal widening of the follicle, resorption of the adjacent roots or any other associated pathology.
   - ✓ Aesthetically acceptable.

The patient should be kept under annual review to verify that these complications have not arisen, the opinion of an orthodontist is important.

2. **Surgical exposure and orthodontic traction**; is the procedure that allows natural or orthodontically guided eruption of the impacted teeth, an active collaboration with an orthodontist is essential for planning this procedure. Certain criteria must be fulfilled:
   - ✓ There should be adequate space in the arch to accommodate the tooth.
   - ✓ There should be an unobstructed path of eruption.
   - ✓ After eruption the tooth should be in near to normal position in all planes.
   - ✓ The timing of the procedure should be as close as possible to the normal eruption time.

The approach is through a palatal envelope flap, extending from the first molar to the first molar on the other side in bilateral impaction cases, or from the first molar to the first premolar on the other side in unilateral impaction cases.
Buccally impacted teeth are approached through a 3-sided buccal flap, depending on its location.
After reflection of a full mucoperiosteal flap, the crown is exposed conservatively taking care not to expose the cementoenamel junction (CEJ) as this may result in increased incidence of external root resorption.
In palatally positioned canine, a window is excised in the soft tissue before replacing the flap, if the bracket is not attached at the same operation the window is packed with a suitable pack until it epithelializes for 2-3 weeks. In buccal approaches it is more appropriate to suture the flap above the crown (apically repositioned flap) and the area below covered with a pack to ensure that the tooth will erupt into an area of keratinized mucosa.

3. **Transplantation**; in this procedure the canine is carefully extracted and transferred to a surgically prepared socket in the dental arch with minimum delay. The transplanted tooth should be splinted in its new position for about a month with an orthodontic appliance.

It is essential to have sufficient space to accommodate the crown of the canine.
Success rate is increased when the unerupted teeth still have open apex and when the handling of the root is kept to minimum to ensure the viability of the cementum and periodontal membrane.
Endodontic treatment should be performed as soon as possible after surgery (about 6-8 weeks), periodic follow up is required to allow early detection of root resorption which is common.

4. **Removal**; surgical extraction maybe performed when the other options are unavailable. The main indications include:
   - ✓ Before construction of a dental prosthesis.
   - ✓ To permit orthodontic alignment of other anterior teeth.
   - ✓ When there is resorption of the roots of adjacent teeth.
   - ✓ When a follicular cyst has developed.
   - ✓ Infection although uncommon.

Extraction can be performed with retention of the primary canine with restorative procedures to improve esthetic contour, extraction can also be accompanied with extraction of the primary canine and orthodontic closure of the space by the first premolar. Implant supported crown can also be used to close the space created by extraction of the impacted canine and the primary canine.

Palatally positioned teeth are approached through palatal envelope flap, while buccal teeth are approached through buccal flap.
Occasionally tooth sectioning is required after bone removal and the tooth is extracted in segments.
Possible complications include:

- Palatal hematoma formation, this can be prevented by an acrylic splint to support the soft tissue.
- Perforation into the floor of the nose, but it rarely causes a problem.

Impacted mandibular canines

These are less frequently impacted than maxillary canines (about 0.3% of population) and are mostly buccally located, partially erupted teeth can be removed easily using elevators or forceps.
Localization of the unerupted teeth is by periapical film, OPG, occlusal view or CBCT.
Consultation with orthodontist is mandatory when considering treatment.
Surgical extraction is by raising a buccal mucoperiosteal flap with care to avoid damaging the mental nerve, bone removal and the tooth is extracted wholly or after sectioning.
Deeply impacted teeth or those located lingually can be left in situ if they do not cause any damage to the adjacent roots or not associated with other pathologies.
Surgical exposure is either through a flap or sometimes through excision of the overlying soft tissue when the tooth is only covered by soft tissue. It is noteworthy to say that orthodontic traction is difficult since impacted mandibular canines are frequently in horizontal position near the roots of the adjacent teeth, also due to the dense bone in the region as well as the buccal position of the impacted teeth.

Impacted lower premolars

It occurs mostly due to loss of space by drifting forward of the first permanent molar after early extraction of the second deciduous molar.
Localization is by periapical film, OPG with occlusal view to demonstrate the buccolingual position or CBCT.
Removal is by raising a buccal flap, with preservation of the mental nerve, bone removal, sectioning of the tooth if needed and extraction of the tooth. Consultation with orthodontist before extraction is essential.

Impacted maxillary premolars

It is usually impacted with its crown palatally, or it may be within the arch between adjacent roots. It can be partially erupted, completely buried or the crown may be wholly exposed, in the latter case extraction is easy with an elevator or forceps. Completely impacted teeth require a palatal envelope flap extending from the second molar to the lateral incisor on the same side, bone removal and extraction of the tooth.
Buccal approach is needed in cases where the tooth is within the arch between the standing teeth, sectioning of the tooth is needed when the root is curved. Care is taken not to damage the adjacent teeth.

Impacted first and second molars

These are uncommonly impacted, their management consist of surgical extraction through a buccal flap, bone removal avoiding damage to the inferior dental nerve that may cross buccal to the neck of the teeth, sectioning of the tooth may be necessary.
Another line of treatment is the surgical uprighting, especially of the impacted second molar. This is done through buccal approach, the tooth is exposed carefully without exposing the CEJ, if the third molar is present it needs to be removed, if not, bone posterior to the second molar is removed, followed by tipping the tooth slightly posteriorly and superiorly, and the tooth can be allowed to erupt spontaneously.
This procedure is better carried out when 2/3 of the roots of the impacted second molar are developed. Teeth with fully developed roots have poor prognosis for this procedure.
Usually there is no need for fixation but RCT may be needed 6-8 weeks after surgery, also there should be no occlusal forces on the tooth in the postoperative period. Follow up for about 2 years is necessary.

2. Buried deciduous molars

These are usually ankylosed and should be removed surgically through buccal approach, bone removal and tooth sectioning if necessary.

3. Supernumerary teeth

These are more in the males than in the females, they can be present in the primary dentition as well as in the permanent dentition, 50% of the cases in primary dentition will have supernumerary teeth in the permanent dentition. Supernumerary teeth can be classified according to their position into:
Mesiodens; is situated in the premaxilla in the midline and it is commonly conical, it can have a horizontal or inverted position. Supplemental teeth may also occur in the anterior maxillary region.
Paramolar; appear in the premolar or molar region and is situated buccally to the teeth, they can be conical or supplemental.
Distomolar; appear as a fourth molar usually distal to the standing molars and they are either normal or smaller in size.

According to the shape they can be either; **conical (peg-shaped)** or **supplementary**; which have the shape and size of a normal tooth or they can have conventional shape with smaller or larger size.

Supernumerary teeth can have no effect on other standing teeth or they can cause failure of eruption of the other teeth, crowding, malposition or misalignment, resorption of the roots of the adjacent teeth or they can be associated with other pathologies (e.g. cysts).
Erupted supernumerary teeth are extracted easily especially if they are conical in shape.
If they need removal they should be localized accurately using periapical films (buccal object rule may be applied), vertex occlusal view or CBCT. OPG is needed to determine the vertical position of the tooth and its position in relation with the floor of the nasal cavity or maxillary sinus.
They are approached palatally through palatal flap or buccally, bone removal and tooth sectioning may be needed, sometimes combined palatal and buccal approach is necessary.

4. Dilacerated incisors

Trauma to the deciduous incisors especially in the 2-3 years of age can cause damage to the underlying permanent incisor tooth germ causing root development to take place at an angle.
Exposure and orthodontic traction can be performed if possible, but if not, these teeth should be removed and the lateral incisors allowed filling their space.
It is essential to seek the opinion of an orthodontist.

5. Corticotomy-assisted orthodontic treatment

It can be defined as a linear cutting technique in the cortical plates surrounding the teeth to produce accelerated tooth movement. This process differs from the osteotomy, in which cortical and cancellous bone is cut for the purpose of repositioning blocks of bone with associated teeth.

The biological mechanism behind the acceleratory effect of corticotomies has been suggested to occur due to **regional acceleratory phenomenon** in which an injury to bone results in the acceleration of all processes involved in healing which allows teeth roots to move rapidly through the alveolar bone. The reported rate of acceleration of tooth movement in human patients has been suggested to be up to 3 times the normal rate of tooth movement, but this acceleratory effect has a finite period which is reported to range from 1 to 4 months.

Indications

Corticotomy is used to facilitate orthodontic tooth movement and to overcome some shortcomings of conventional orthodontic treatment, such as the long required duration, limited tooth movement and difficulty of producing movements in certain directions. Some of the clinical applications include:

- ✓ Treatment of crowding.
- ✓ Canine Retraction after Premolar Extraction.
- ✓ Enhance post orthodontic stability.
- ✓ Facilitate eruption of impacted teeth.
- ✓ Facilitate orthodontic expansion.
- ✓ Molar intrusion and open bite correction.

Corticotomy is contraindicated in patients with active periodontal disease or gingival recession.

Surgical procedure

Various surgical interventions have been described:

- ➢ The traditional corticotomy procedure entails raising full-thickness buccal or buccal and palatal/lingual mucoperiosteal flaps and performing vertical linear interradicular corticotomy cuts (about 0.5 mm in depth), with or without joining horizontal subapical corticotomy cuts, or by drilling multiple holes that penetrated the cortical plate instead of linear cuts.
- ➢ More conservative techniques involved making vertical interproximal soft tissue incisions without raising soft tissue flaps and making corticotomy cuts through these incisions using piezosurgical instrument or a hard tissue laser.
- ➢ Grafting material may be added on the cut alveolar bone in some cases especially where alveolar bone is considered thin.

After surgery orthodontic force can be applied either immediately or within 2 weeks after surgery.

Complications

Possible complications include:

- ❖ Adverse effects on periodontium.
- ❖ Adverse effect on vitality of the pulp.
- ❖ Root resorption.

6. Labial frenectomy

A frenum is a small band or fold of mucosal membrane that attaches the lips and cheeks to the alveolar process and limits their movement. The maxillary midline frenum connects the mucosa of the maxillary alveolar process and central incisors to the upper lip. Histologically it contains elastic fibers and collagen tissue components. It is a dynamic structure that changes in shape, size and position during growth.

Histological studies show that collagen fibers of labial frenum disrupt the trans-septal periodontal fibers of the central incisors which is related to midline diastema.

Management of diastema is by orthodontic treatment, in some cases labial frenum is hypertrophic inhibiting orthodontic closure necessitating surgical removal before the end of orthodontic therapy, although surgical removal of the frenum before orthodontic treatment is reported to lead to a more rapid crown approximation but it is believed that abundant granulation tissues may complicate diastema closure.

Frenectomy is the total removal of the frenum. Various surgical techniques have been proposed:

- The simple excision technique.
- The Z-plasty technique.
- Localized vestibuloplasty with secondary epithelialization.
- The laser-assisted frenectomy

The simple excision technique

It is performed under local anesthesia; a narrow elliptical incision around the frenal area down to the periosteum is completed. The frenum is then sharply dissected from the underlying periosteum and soft tissue, and the margins of the wound are gently undermined and reapproximated.
Placement of the first suture should be at the maximal depth of the vestibule and should include both edges of mucosa and underlying periosteum at the height of the vestibule beneath the anterior nasal spine to allow for adaptation of the tissue to the maximal height of the vestibule. The remainder of the incision is closed with interrupted sutures. The excision closest to the alveolar ridge crest may require periodontal dressing and will undergo secondary epithelialization. The advantage of this technique is that it is easy to perform while its main disadvantages are scar tissue formation and relapse.

The Z-plasty technique

The excision of the frenum is done as in the previous technique, after excision of the frenum, two oblique incisions are made in a Z fashion, one at each end of the previous area of excision. The two flaps are then undermined and rotated to close the initial vertical incision horizontally, it is a more demanding procedure and more aggressive but it results in less scar formation.

Localized vestibuloplasty with secondary epithelialization

It is indicated in case of wide frenal attachment, an incision is made through mucosal tissue and underlying submucosal tissue. A supraperiosteal dissection is completed by undermining the mucosal and submucosal tissue, the edge of the mucosal flap is sutured to the periosteum at the maximal depth of the vestibule and the exposed periosteum is allowed to heal by secondary epithelialization.

The laser-assisted frenectomy

Frenectomy can be performed by laser. Diod laser, Nd:YAG laser, Er:YAG laser and CO_2 Laser have been reported. The main advantages of laser are; less bleeding during surgery, no need for suturing or periodontal dressing, with minimal postoperative swelling and discomfort.

ORAL SURGERY

LECTURE 1 **ا.م.د.احمد فاضل ابراهيم القيسي**

Rheumatologic and Connective tissue disorders

Rheumatologic (or rheumatoid) disorders include much more than "arthritis" and encompass a large group (nearly 100) of disorders that affect bones, joints, and muscles. Arthritis is a nonspecific term that means "inflammation of the joints." Often arthritis is used interchangeably with rheumatism or rheumatoid arthritis (RA) to denote aches, pains, and stiffness in the joints and muscles, but these terms are not synonymous nor inclusive. Rheumatologic disorders include (RA), osteoarthritis (OA), psoriatic arthritis (PsA), systemic lupus erythematosus (SLE), juvenile rheumatoid arthritis (JRA), scleroderma (SD), Sjögren syndrome (SS), gout, ankylosing spondylitis, Lyme disease, giant cell arteritis (GCA or temporal arteritis), and fibromyalgia syndrome (FMS).

RHEUMATOID ARTHRITIS

Rheumatoid arthritis is an autoimmune disease of unknown origin characterized by symmetric inflammation of joints, especially of the hands, feet, and knees. The severity of the disease varies widely from patient to patient and fluctuates over time within the same patient. Disease onset usually occurs between ages 35 and 50 years. RA is more prevalent in women than in men by a 3:1 ratio.

Oral Complications and Manifestations

The most significant complication of the oral and maxillofacial complex in RA is TMJ involvement, which is found in up to 45% to 75% of patients with RA. This may present as bilateral preauricular pain, tenderness, swelling, stiffness, and decreased mobility of the TMJ, or it may be asymptomatic. Periods of remission and exacerbation may occur, as with other joint involvement. Fibrosis or bony ankylosis can occur; therefore, treatment should be initiated promptly.

Clinically, patients may present with tenderness over the lateral pole of the condyle, crepitus, and limited opening. Radiographic changes initially may show increased joint space caused by inflammation in the joint. Later, these inflammatory changes progress to erosive degenerative changes and changes in size and shape of the joint and can involve both the condyles and the fossa. A potential dental complication is the development of an anterior open bite, caused by destruction of the condylar heads and loss of condylar height. Although palliative treatment such as interocclusal splints, physical therapy, and medication may prove to be helpful, surgical intervention can become necessary to decrease pain, improve appearance, or restore function.

There is recent evidence that patients with RA have a higher incidence of periodontal disease. Therefore, meticulous oral hygiene combined with more frequent dental prophylaxis will be more effective in reducing periodontal problems.

An additional complication that may be seen in patients with RA is severe stomatitis that occurs after the administration of drugs such as gold compounds, penicillamine, or immunosuppressive agents. Stomatitis may be an indication of drug toxicity and should be reported to the physician. Treatment for this problem should include consideration for changing the offending drug and palliative mouth rinses, diphenhydramine elixir, or a topical emollient such as Orabase.

Dental management:

1- **Patient Evaluation and Risk Assessment**

a- Evaluate and determine whether rheumatoid or joint disorder exists.

b- Obtain medical consultation if disease is poorly controlled or undiagnosed or if the diagnosis is uncertain.

2- **Drugs**

   a- Analgesics: If patient is taking aspirin or another NSAID or acetaminophen, be aware of dosing and the possibility that pain may be refractory to some analgesics; dosing and analgesic choices may need to be modified in consultation with the physician.

   b- Antibiotics: Provide antibiotic prophylaxis if needed in accordance with ADA (2015) guidelines. (Patients who are taking gold salts, penicillamine, sulfasalazine, or immunosuppressive agents are susceptible to bone marrow suppression, which can result in anemia, agranulocytosis, and thrombocytopenia.)

   c- Anesthesia: No issues

   d- Allergy: Allergic reactions or lichenoid reactions are possible in patients taking many medications.

3- **Bleeding:** Excessive bleeding may occur if major surgery is performed on patients who take aspirin or other NSAIDs. Bleeding usually is not clinically significant and can be controlled with local hemostatic measures. (only patient who is taking both aspirin and a corticosteroid may be at greater risk for bleeding**).**

4- **Chair position:** Ensure comfortable chair position. Consider shorter appointments, and use supports as needed (e.g., pillows, towels).**(** The patient also may be more comfortable in a sitting or semisupine position, as opposed to a supine one

5- **Devices:** Patients who have a prosthetic joint replacement should be managed according to ADA (2003) guidelines (mentioned later in the lecture).

6- **Investigations**: Obtain blood cell count with differential if surgery is planned for patients taking gold salts, penicillamine, antimalarials, or immunosuppressives. If patient is taking corticosteroids—secondary adrenal suppression is possible.

7- **Emergencies:** If surgery is performed, supplemental techniques may be necessary to control bleeding.

8- **Follow-up:** Monitor dental and periodontal health; routine follow-up evaluation is appropriate

Management of Patients with Prosthetic Joints Undergoing Dental Procedures:

Clinical Recommendation:

1- Generally, patients with prosthetic joint implants, prophylactic antibiotics are not recommended before dental procedures to prevent prosthetic joint infection.
2- patients with a history of complications associated with their joint replacement surgery who are undergoing dental procedures that include gingival manipulation or mucosal incision, prophylactic antibiotics should only be considered after consultation with the patient and orthopedic surgeon.
   * To assess a patient's medical status, a complete health history is always recommended when making final decisions regarding the need for antibiotic prophylaxis.

Reasons for that above Recommendation:

1- There is evidence that dental procedures are not associated with prosthetic joint implant infections.
2- There is evidence that antibiotics provided before oral care do not prevent prosthetic joint implant infections.
3- There are potential harms of antibiotics, including risk for anaphylaxis, antibiotic resistance, and opportunistic infections such as Clostridium difficile.
4- The benefits of antibiotic prophylaxis may not exceed the harms for most patients.
5- The individual patient's circumstances and preferences should be considered when deciding whether to prescribe prophylactic antibiotics prior to dental procedures.

High-Risk Patients with Prosthetic Joints

1- Immunocompromised or Immunosuppressed Patients
   - Inflammatory arthropathies: rheumatoid arthritis; systemic lupus erythematosus; disease-, drug-, or radiation-induced immunosuppression
2- Other Patients
   - Insulin-dependent (type 1) diabetes
   - First 2 years after joint replacement
   - Previous prosthetic joint infections
   - Malnourishment
   - Hemophilia

Suggested Antibiotic Prophylaxis Regimens

1- Patients Not Allergic to Penicillin: Cephalexin, Cefradine, or Amoxicillin
   - 2 g orally 30 minutes to 1 hour before the dental procedure.
2- Patients Not Allergic to Penicillin and Unable to Take Oral Medications: Cefazolin or Ampicillin
   - Cefazolin 1 g or ampicillin 2 g intramuscularly or intravenously 30 minutes to 1 hour before the dental procedure.
3- Patients Allergic to Penicillin: Clindamycin

- 600 mg orally 30 minutes to 1 hour before the dental procedure.

4- Patients Allergic to Penicillin and Unable to Take Oral Medications: Clindamycin

- 600 mg intravenously 30 minutes to 1 hour before the dental procedure.