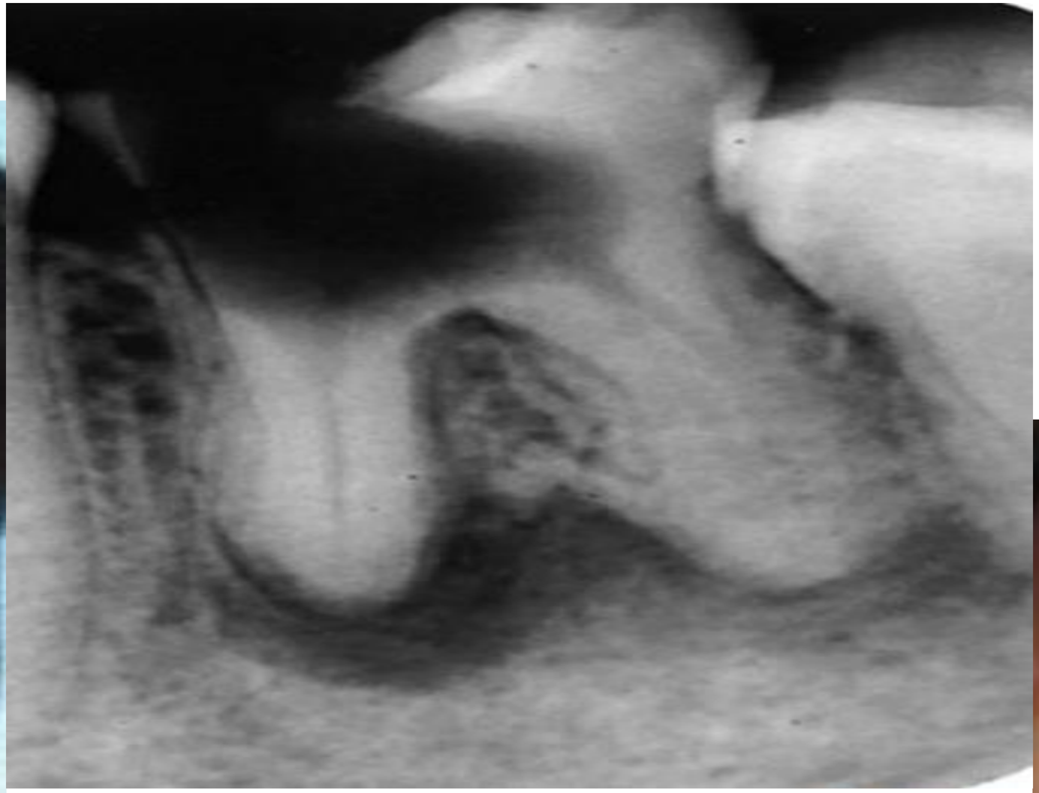
Odontogenic and fascial space infections

14-16

Prof. Abbas AY Taher

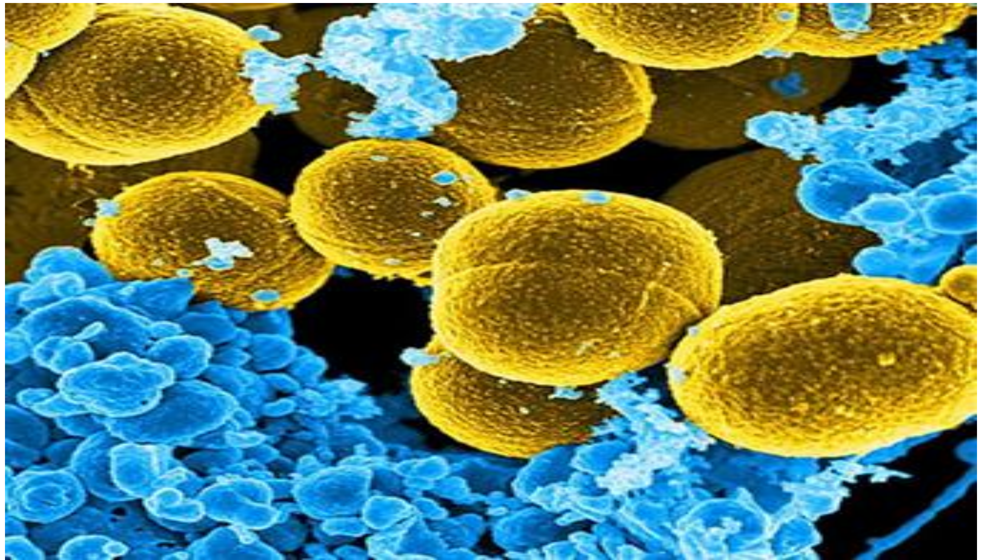
ODONTOGENIC INFECTION:

The pathology of odontogenic infections originates from the pulp and periodontal structures. Once the infection spreads from the dentoalveolar structure, it may further infect fascial faces and lead to cellulitis or abscess formation. Among the various fascial spaces, the first to be involved in odontogenic infection is the submandibular space. Similar to the masticatory space, the involvement of the submandibular space leads to severe symptoms such as **trismus**, **neck rigidity, respiratory distress**, **dysphagia**, **sialorrhea**, and **pyrexia.**

Microbiology

It was concluded that odontogenic infections were mixed aerobic-anaerobic infections. Anaerobic as well as aerobic cultures were necessary to isolate all pathogens. Successful management of these infections depends on changing the environment through **decompression**, **removal of the etiologic factor** and by **choosing the proper antibiotics.**

The mandibular 3(rd) molar was found to be the most commonly **offending tooth**, followed by the **mandibular 2(nd) molar**. The submandibular space was the most frequently involved **fascial space** both in **single fascial space infections** and **multiple fascial space infections.**
Mixed growth (**aerobic** and **anaerobic**) was seen in culture smears, only **aerobic bacterial growth was seen samples of patients** and anaerobic bacterial growth was seen in culture smears of samples of patients .
Streptococcus viridans was the most frequently isolated bacteria among the **aerobes**, whereas **Bacteroides** and **Prevotella** were the most common **bacterial species among anaerobes**.
Empirical antibiotic therapy in the form of **Co amoxiclav(Amoxicillin + Clavulanic Acid)** =(Augmentin) and **Metronidazol**e was given.
Incision and **drainage** followed by , **Endo or extraction of the offending tooth/teeth was carried out**



Pus producing (or pyogenic) infections associated with the teeth and surrounding supporting structures such as the periodontium and the alveolar bone.

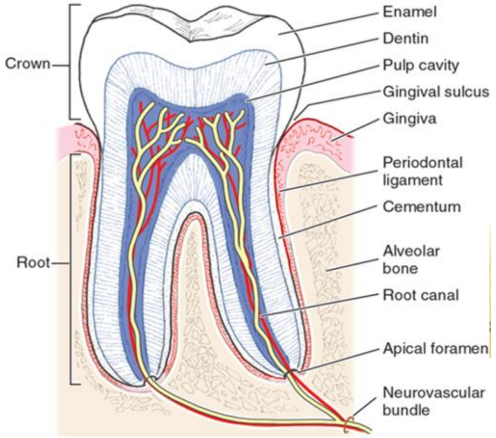
The clinical presentation of dentoalveolar infections depends on the **virulence** of the causative microorganisms, the **local** and **systemic defence** mechanisms of the host, and the **anatomical features** of the region.

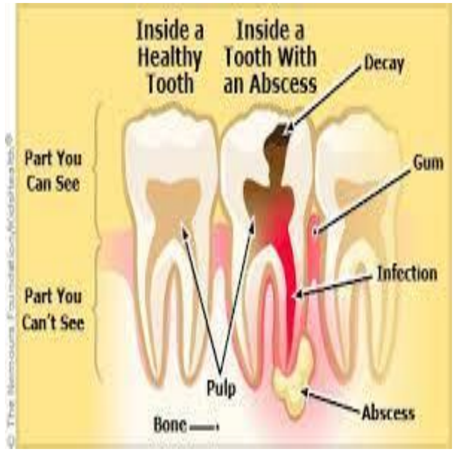
The resulting infection may present as:

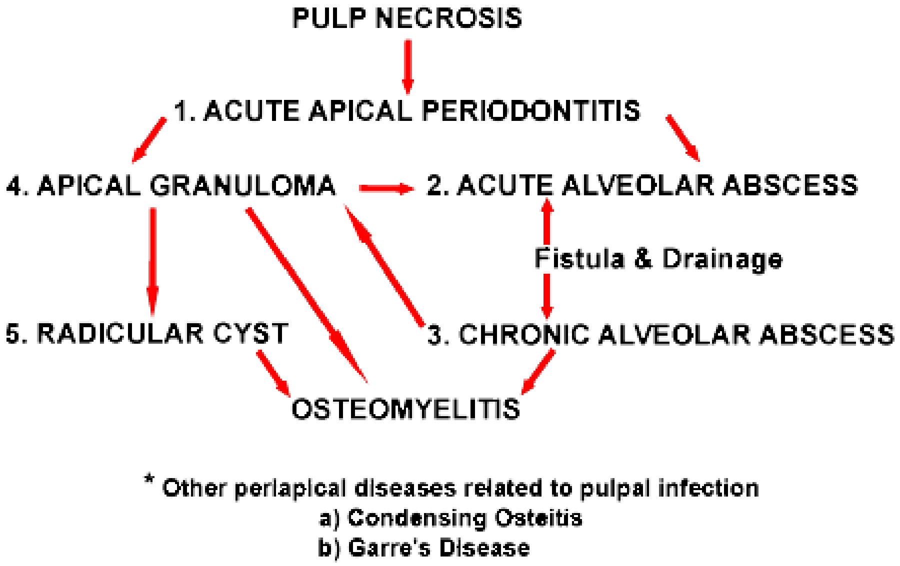
• an abscess localised to the tooth that initiated the infection

• a diffuse cellulitis that spreads along fascial planes

• a **mixture of both.**

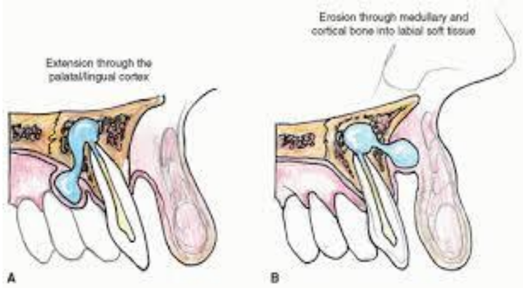






The Odontogenic infection develops by the extension of the initial carious lesion into dentine and spread of bacteria to the pulp .The pulp responds to the **infection** with **acute inflammation which causes necrosis**.

Once pus formation occurs, it may **remain localised** at the **root apex** and **develop into either an acute** or a chronic abscess, develop into a focal osteomyelitis or, as hydrostatic pressure increases, it can track along the path of least resistance to the tissue spaces. **Potential for local and distant spread.**



Local measures

Abscesses can compress other structures and reduce blood supply.

Abscesses have **poor blood supply** and **therefore antimicrobials wont reach them.**

Correct treatment is DRAINAGE and REMOVAL OF CAUSE

Drainage can be though the **tooth** or via **Hiltons method (**Incision and Drainage) to the abscess.

Drainge of abcess should be done when:

XLA and RCT no use

Find most dependent point of abscess (flactuant),and incise **through mucosa** and **periosteum**

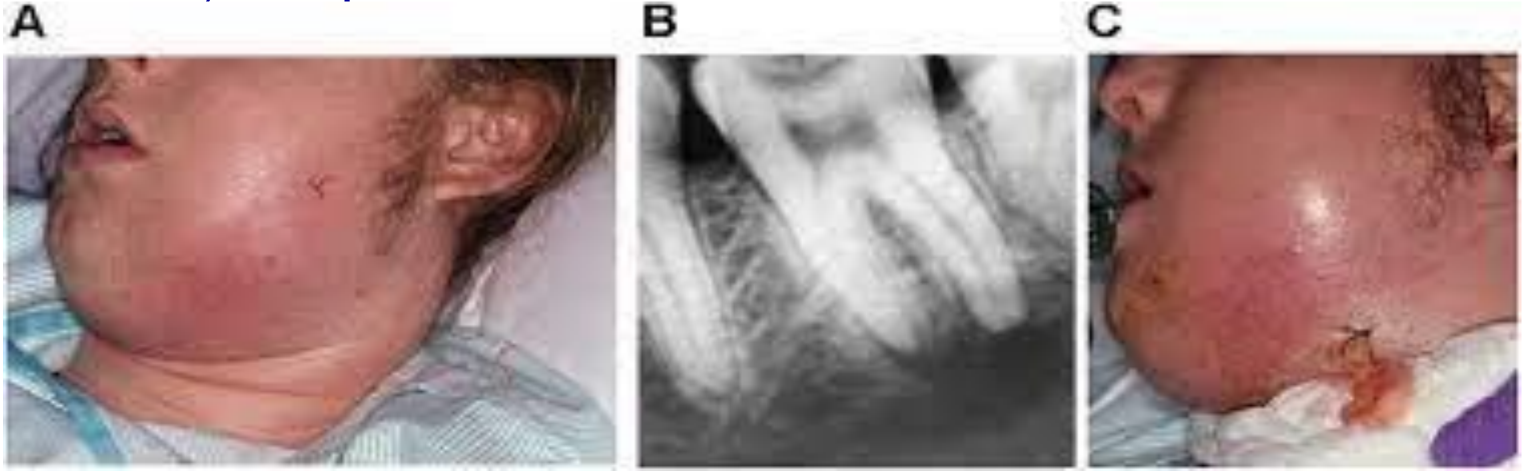
Blunt dissection to break down lobules of pus

General measures

Analgesics – paracetamol, ibuprofen, cocodamol

Supportive – fluids, rest, diet

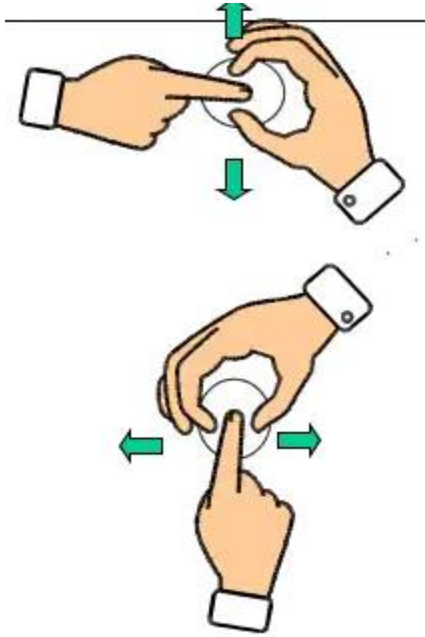
Antibiotics IF systemic involvement, cellulitis, compromised host defences involvement of fascial spaces.



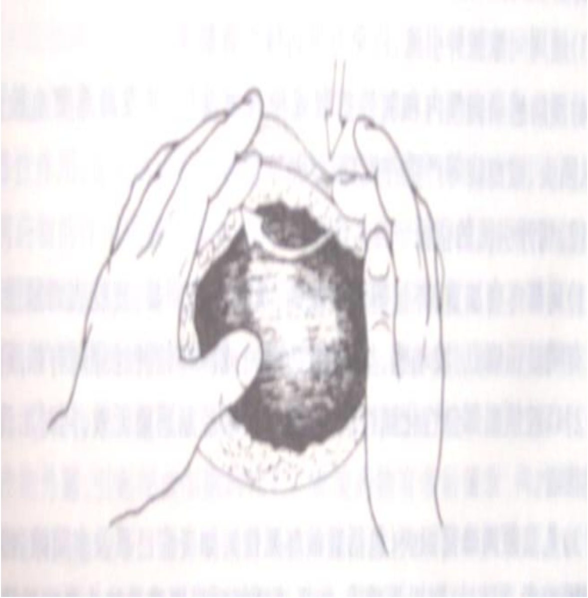
Technique Fluctuant Examination

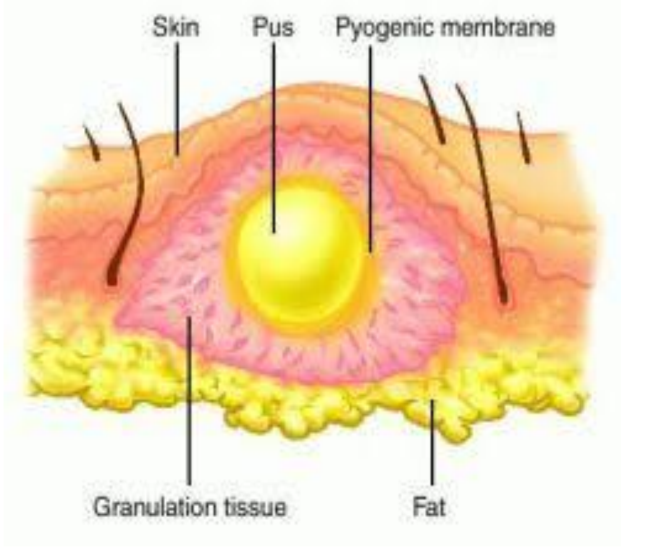
Fluctuation can be tested by placing the index and middle finger of the one hand (somewhat apart) on the swelling, with the index finger of the other hand placed in between these two fingers. Alternate downwards pressure over the lesion with these fingers.

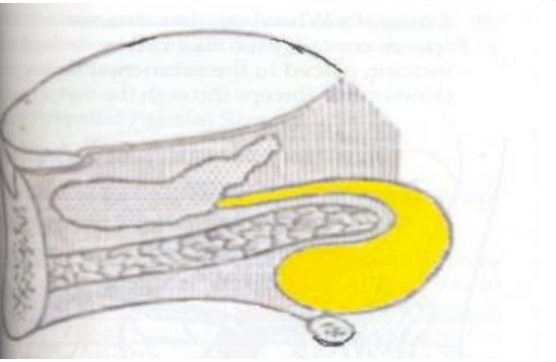
Fluctuation occurs when the finger of the one hand is forced upwards when pushing downwards with the fingers of the other hand. Use a balloon partially filled up with water to practice this technique



- Two digits are placed either side of the apex of the swelling
- Pressure is applied to the apex
- This is repeated with the digits at 90°
- The finding is positive if the two digits are pushed away in both directions
- If a swelling is fluctuant, it suggests the presence of fluid within the swelling.







Acute dental Abcess :

(1) acute suppurative pulp open early, every effort should be made to open the pulp, Pott from the drainage canal, **reducing stress**.

(2) As inflammatory exudation in periapical tissue, and sometimes open the pulp still not achieve the purpose of drainage, There was thus a need **to remove inflammation of the pulp and light to operate at this time**, to avoid the spread of infection pulp deep. After pulling myeloid not sealed to facilitate drainage.

(3) Once the abscess incision abscess limitations, we must incision, the incision under local anesthesia, the incision to be as deep as the periosteum, avoid cutting nerves and blood vessels.

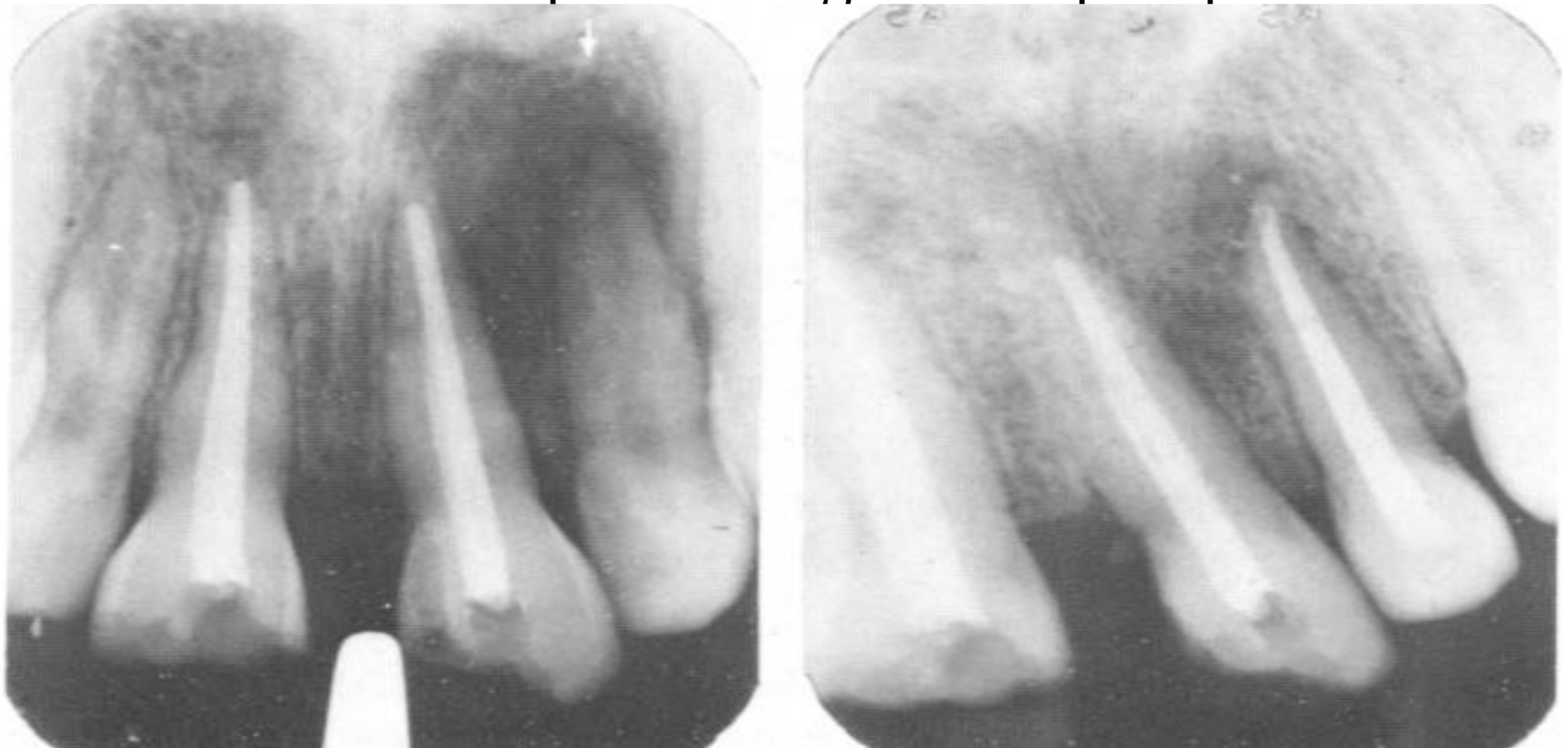
(4) use of anti-inflammatory drugs and analgesics in acute alveolar abscess significant systemic symptoms, or systemic diseases such as **diabetes**, abscesses after incision, the government should provide medical treatment and systemic antibiotic therapy.

(5) in the treatment of teeth to control inflammation, according to **extraction of teeth** or **root canal therapy**

Acute periapical abscess

The usual causes of infective:

- A carious cavity.
- Contamination of a traumatic exposure of the pulp.
- A sterile necrosis, as when the apical vessels are torn by a blow on the tooth.
- Following chemical or thermal damage to the pulp during coronal restoration.
- The necrotic pulp subsequently becomes infected via the periodontal lymphatics from the gingival crevice.
- Exacerbation of a pre-existing chronic periapical infection.



Acute periodontal abscess :

While less common than an endodontic abscess, a periodontal abscess is the third most frequent dental emergency requiring immediate intervention due to their rapid onset of pain

The causes:

- Food impaction.
- Repeated occlusal traumaAcute periodontal abscess produces redness and swelling of the gingival margin, interdental papilla and of the mucoperiosteum lateral to the tooth. The pain is usually continuous, dull or throbbing but rarely as severe as a periapical abscess. The pus usually discharges via the gingival pocket, but may produce a sinus on either the inner or outer aspect of the alveolar process.




The management of acute lesions includes establishing drainage via pocket lumen, subgingival scaling and root planing, curettage of the lining pocket epithelia and seriously inflamed connective tissue, compressing pocket wall to underlying tooth and periodontal support, and maintaining tissue contact.

There are two main etiologies . **Firstly**, in cases with a background of periodontitis, the condition is possibly due to untreated periodontitis or occurring during periodontal therapy . **Secondly**, in cases that are not related to periodontitis, the main causes are usually the presence of foreign objects and radicular abnormalitie

The management of acute lesions includes **establishing drainage via pocket lumen, subgingival scaling and root planing, curettage of the lining pocket epithelia and seriously inflamed connective tissue**, compressing pocket wall to underlying tooth and periodontal support, and maintaining tissue contact.

Treatment predominantly consists of two phases:

Acute management and definitive treatment once the acute phase has been resolved. **Acute treatment** aims to **alleviate the symptoms** and **reduce the risk of the spread of infection**.

The abscess must be drained by incising through the periodontal pocket or over the area of greatest fluctuant swelling on the gingiva.

Drainage should be accompanied by the mechanical scaling of the periodontal pocket and antiseptic rinse, removing necrotic tissue and bacterial load. This enables the host immune response to tackle the infection.




Postoperatively, warm salt water rinses are encouraged, and copious fluid intake to reduce the swelling.

Dental practitioners must reassess the lesion and develop a **long-term treatment plan** as necessary, usually consisting of periodontal therapy.

Exodontia is indicated when the clinician deems tooth **prognosis poor or hopeless**, whether from **periodontal disease** or the **destruction** caused by the abscess.

If the cause of the abscess is an **embedded foreign object**, this must be **removed through debridement**, and drainage through the gingival sulcus should be performed either with a periodontal probe or by smooth scaling.

Finally, a warm saline rinse is indicated. Follow up after one to two days.

Antimicrobial therapy will benefit patients with a compromised immune system, those where drainage is not achievable, or with systemic spread.

Systemic involvement presents as **fever**, **fatigue**, **cellulitis**, or **lymphadenopathy**. When selecting an antibiotic, the professional should consider susceptibility and resistance of the bacterial strain, patient allergies, and drug interactions. Amoxicillin, combined with clavulanic acid, is the **first choice of antibiotic**.

Clindamycin is recommended as an alternative for patients with **penicillin allergies**.

Periodontal surgery, including **gingivectomy** or **flap procedures**, can be used in cases of chronic periodontal abscesses.

These surgical procedures aim to **remove the residual calculus and drain the abscess**. They are especially useful in abscesses related to deep vertical defects. Periodontal flaps have been suggested in periodontal abscesses that develop after periodontal therapy, where the calculus is left subgingivally.

A pericoronal abscess:

- that food collects between the crown of the tooth and the overlying gum flap
- pressure on the operculum to express
- to the overlying pad of gum from the cusps of an opposing tooth
- The virulence of the bacteria within the pericoronal space
- Lowering of the host resistance appears

A pericoronal abscess arises around the crown of a partially erupted vital tooth, usually the 3rd molar, and therefore, resembles a periodontal abscess.

A **partially erupted** or **impacted 3rd molar (wisdom tooth)** is the most common site of **pericoronitis** and **pericoronal abscesses**.

The accumulation of food and debris between the overlying gingival flap and crown of the tooth creates a focus for pericoronitis and subsequent abscess formation.

The gingival flap becomes irritated and inflamed, and the tissue is repeatedly traumatized by the opposing molar tooth.

The inflamed gingival process may eventually become infected and form an abscess. Foul taste, inability to close the jaw, and fever may occur.

Swelling of the cheek and angle of the jaw and localized lymphadenopathy are also characteristic. More advanced disease may spread posteriorly to the base of the tongue, oropharyngeal area, and deep cervical spaces with resulting Ludwig angina and peritonsillar abscesses.

Source: Kevin J. Knoop, Lawrence B. Stack, Alan B. Storrow, R. Jason Thurman: The Atlas of Emergency Medicine, 5e

Pericoronitis:

Pericoronitis is inflammation in the soft tissues (such as the gingiva and dental follicle) surrounding the crown of a partially erupted tooth, usually the lower 3rd molar (wisdom tooth).

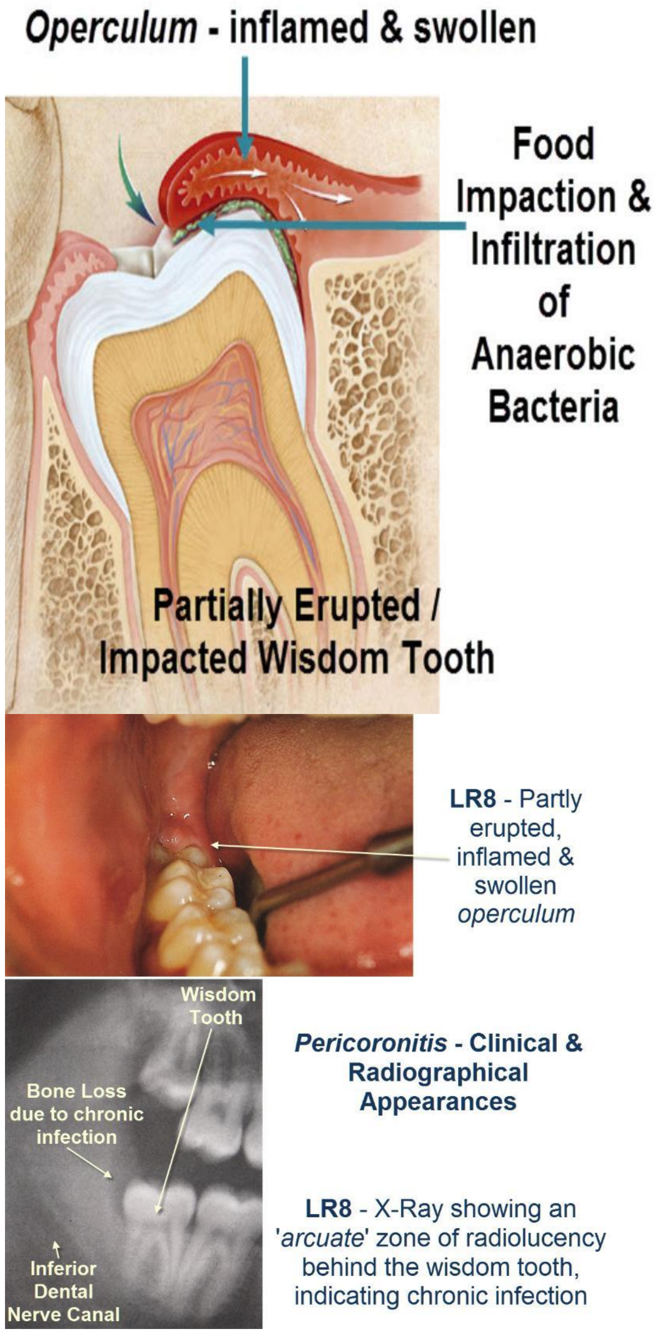
It generally does not arise in teeth that erupt normally; usually, it is seen in teeth that erupt very slowly or become impacted and it most commonly affects the lower 3rd molar.

, it is thought that bacterial infiltration into the follicular space initiates the infection. **Once the follicle of the tooth communicates with the oral cavity**

The soft tissue covering over a partially erupted tooth is known as peri-coronal flap or gingival operculum. Maintenance of oral hygiene in such area can be very difficult to achieve by normal methods of oral hygiene.

There was no significant difference between the sexes. A seasonal variation is noted with peak incidences occurring in June and December.

Bilateral (both sides) pericoronitis is rare and suggests **underlying infectious mononucleosis (Glandular Fever)**



Cellulitis: initial stage of infection:

- Diffuse, reddened, soft or hard swelling that is tender to palpation.
- Inflammatory response not yet forming a true abscess.
- Microorganisms have just begun to overcome host defenses and spread beyond tissue planes.

BACTERIA :

A streptococci of the **viridans group** and **Staphylococcus aureus** or **epidermidis are the major pathogens in alveolar or tissue space abscesses of odontogenic origin**. A mixed population of,3-4 species is usually recovered

Classifications

Pericoronitis is classified into:

- acute
- Subacute
- Chronic

Acute pericoronitis

- facial swelling is common
- Limitation of opening may be marked
- pain is throbbing and severe and may interfere with sleep
- pyrexia, severe malaise and anorexia
- The regional lymph nodes are tender and enlarged
- If untreated the infection is likely to spread to the adjacent tissue spaces.

Acute pericoronitis is associated with a wide range of symptoms including severe pain, swelling and feve

Subacute pericoronitis

- a well localized dull pain
- the gum pad is swollen, tender and red
- Sometimes pus can be seen oozing from beneath the anterior margin of the gingival flap
- There is foetor oris
- The patient may complain of slight discomfort on swallowing
- some difficulty in opening the mouth
- The submandibular lymph nodes on the affected side are enlarged and tender.

Chronic pericoronitis

Pericoronitis may also be classified as **chronic / recurrent**. Acute pericoronitis occur repeatedly and periodically. It may cause few symptoms but some signs are visible at the time of intra-oral examination. **The chronic type mostly seen with good-moderate oral hygiene.**

The main complaint is of a dull pain or mild discomfort lasting a day or two, with remission lasting many months. Cacogeusia (bad taste) may also be present. Pregnancy and fatigue are associated with an increased occurrence of pericoronitis.

Chronic Pericoronitis –

Clinical Features:

- Localised tissue swelling & redness
- Soreness
- Continuous dull pain

Localised rise in temperature
•Lymphadenitis +
The most common site of **pericoronitis is impacted or partially erupted lower 3rd molar**.
The most common cause behind peri-coronal inflammation is the entrapment of plaque and food debris between tooth crown and overlying operculum (gum flap). This is an ideal area for the growth of bacteria and it is difficult to keep clean; also, there is the constant possibility of acute inflammation of peri-coronal (around the crown of the tooth) sites. It may be due to trauma, occlusion or entrapment of foreign body below the peri-coronal flap / operculum

The patient is usually asymptomatic except for the occasional mild discomfort or bad taste due to a discharge of pus from, beneath the gum flap

Risk Factors of Pericorontis :

•Presence of unerupted / partially erupted tooth / teeth in communication with the oral cavity. Lower 3rd molars (which are vertically and disto-angularly impacted) are most commonly affected
•Presence of periodontal pocket adjacent to unerupted / partially erupted teeth
•Opposing tooth / teeth in relation to peri-coronal tissues surrounding unerupted / partially erupted tooth / teeth
•Previous history of pericoronitis
•Poor oral hygiene
•Respiratory tract infections & tonsillitis
•Age (20 – 29 year age group; the condition was rarely seen before 20 or after 40).
•Emotional stress

Management:

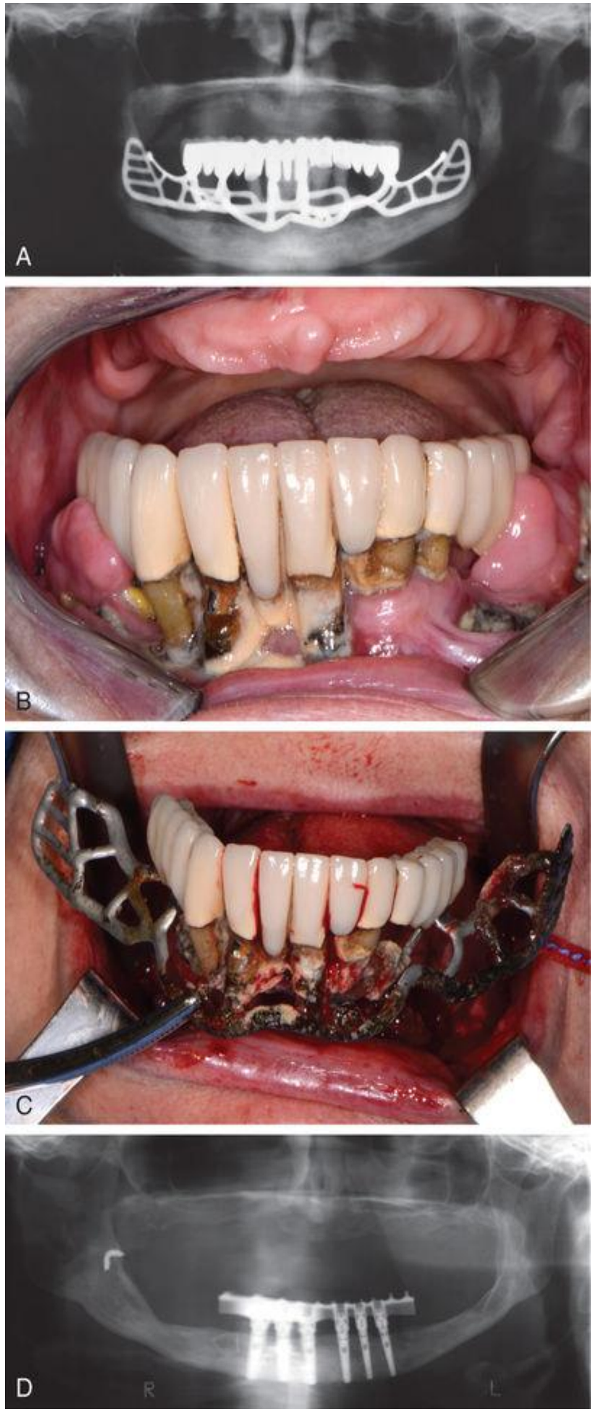
These include debridement of plaque and food debris, drainage of pus, irrigation with **sterile saline**, **chlorhexidine** or **hydrogen peroxide** & **elimination of occlusal trauma**.
Ozone has been put forward as a local antimicrobial that might be a useful adjunct in the treatment of pericoronitis; however, there is no research available to show its efficacy as yet.
In addition to local pain and swelling, if the patient is exhibiting regional or systemic signs and symptoms, **antimicrobial therapy** (such as metronidazole) is recommended; however, it should be emphasised that it is as an adjunct rather than a **1st line treatment**. These are all active against anærobic bacteria, which are the predominant microflora found in pericoronitis.
Once the acute phase of the pericoronitis has passed, operculectomy has been used as a preventive measure.

Operculectomy is a **minor surgical procedure where** the affected soft tissue/ the flap of gum over the wisdom tooth, is cut away, preventing further build-up of debris and plaque, and subsequent inflammation

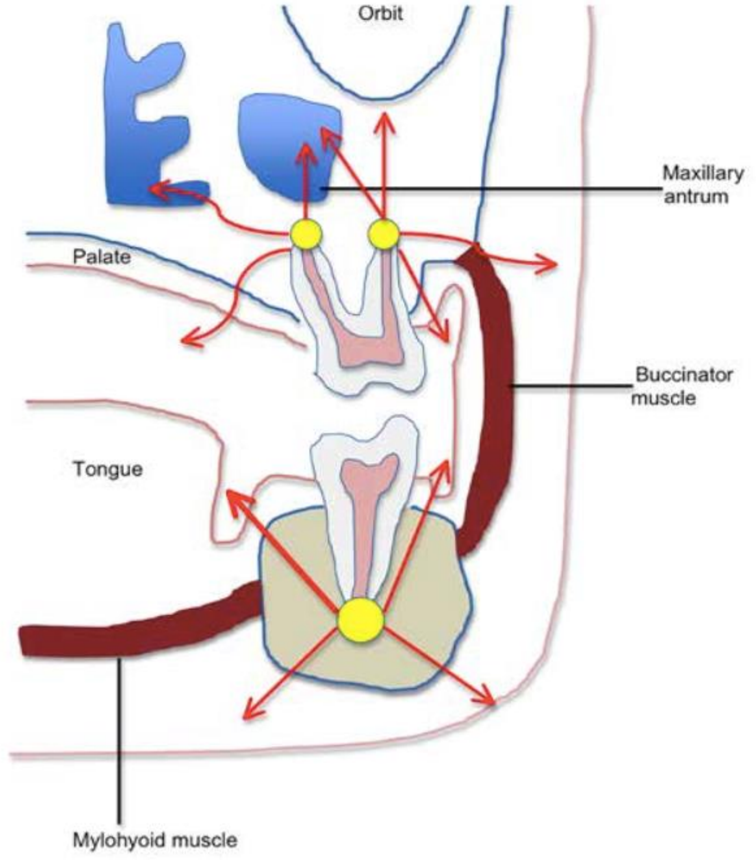
Other abscess may also arise in association with:

- Infected cysts
- Odontomes

- tooth and bone fractures
- subperiosteal and endosteal implants
- foreign bodies



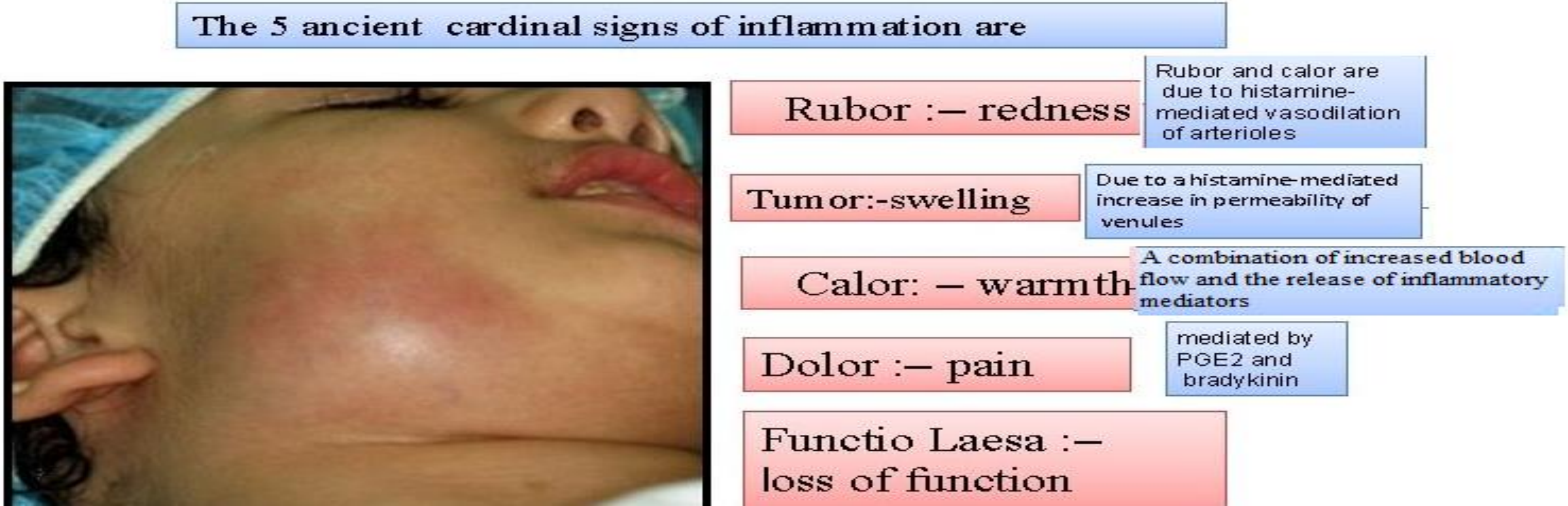
Term	Definition
Abscess	Localized collection of pus
Cellulitis	Inflammation of the subcutaneous tissues with no significant localization of pus (may later organize to form an abscess)
Sepsis	The presence of the systemic inflammatory response syndrome (temperature <36° or >38°; pulse rate >90 per minute; respiratory rate >20 per minute; and white cell count <4 or >12x109/L) plus confirmed infection
Dysphagia	The subjective sensation of difficulty swallowing
Dyspnoea	The subjective sensation of difficulty breathing



Anatomical Space	Description of Space	Signs and Symptoms of Infection in Space
Sublingual space	Bounded by the mucosa of the floor of the mouth superiorly and the mylohyoid muscle inferiorly.	Swollen, red floor of mouth. Little or no extra-oral sign of swelling. Elevation of the tongue may result in dysarthria, dysphagia and/or dyspnoea.
Submandibular space	Lies between the mylohyoid muscle, superficial fascia, platysma and skin. Contains the submandibular gland and lymph nodes.	Painful, red swelling of the neck immediately below the lower border of the mandible. Involvement of the muscles of mastication results in trismus.
Submental space	Lies between the mylohyoid muscles and the skin, just beneath the chin. Contains submental lymph nodes.	Swelling and erythema of the chin.
Submasseteric space	Bounded by the lateral border of the mandible and the medial aspect of the masseter.	Pain and swelling over the angle of the mandible plus trismus.
Parapharyngeal (lateral pharyngeal) space	Extends from the base of the skull to the level of the hyoid bone. It contains the internal carotid artery, the internal jugular vein, cranial nerves IX to XII and the sympathetic chain.	Examination of the oro-pharynx reveals deviation of the uvula to the opposite side, accompanied by displaced tonsil and lateral pharyngeal surface. There may be trimsus and swelling of the lateral neck. Involvement of vital structures may include internal jugular vein thrombosis, Horner syndrome* and meningitis.
Retropharyngeal space	This space lies behind the pharynx and oesophagus between the skull base and mediastinum.	The patient will be unwell and complain of severe sore throat. There will be dysphagia and limitation in neck movement.

Clinical Features

- Inflammation is tissue response to injury or invasion by microorganisms that involves vasodilation, capillary permeability, mobilization of leukocytes, and phagocytosis.
- Cardinal signs of inflammation:
  - Red, hot, swelling, pain, with loss of function
- Other findings: regional lymphadenopathy, fever, elevated white blood cell count, tachycardia, tachypnea, dehydration, malaise.



Factors Contributing to the Spread of Odontogenic Infections:

1-Spreading odontogenic infections **remain surprisingly common**.

2-Poor oral hygiene

3-Social deprivation

4-Tobacco smoking

5-Patients with immune suppression.

Immunity against intraoral infection is composed of three sets of mechanisms(host factors)

1-Humoral factors In presence of infection, histamine, serotonin, prostaglandins support inflammation → vasodilation and increased vascular permeability.

(Circulating immunoglobulins, along with complement, combine with microbes to form opsonins that promote phagocytosis by macrophages. ,IgA prevents colonization of microbes on oral mucosal surfaces.

2-Cellular factors (Phagocytes engulf and kill microbes, removing them, preventing replication.,Lymphocytes produce lymphokines and immunoglobulines (aids humoral),Lymphokines stimulate reproduction of other lymphocytes, and kills antigens.

3-Local factors, oral hygiene, smoking ,

Decrease one of these mechanisms and it increases the potential for infection.

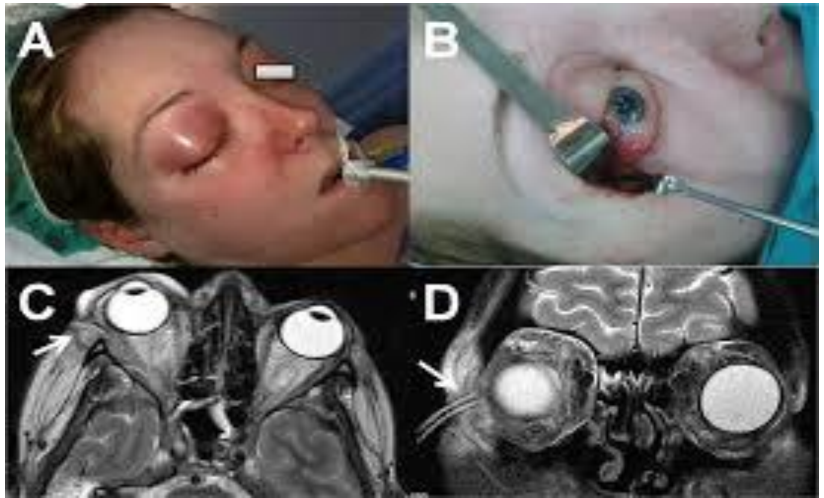
Spread to fascial spaces

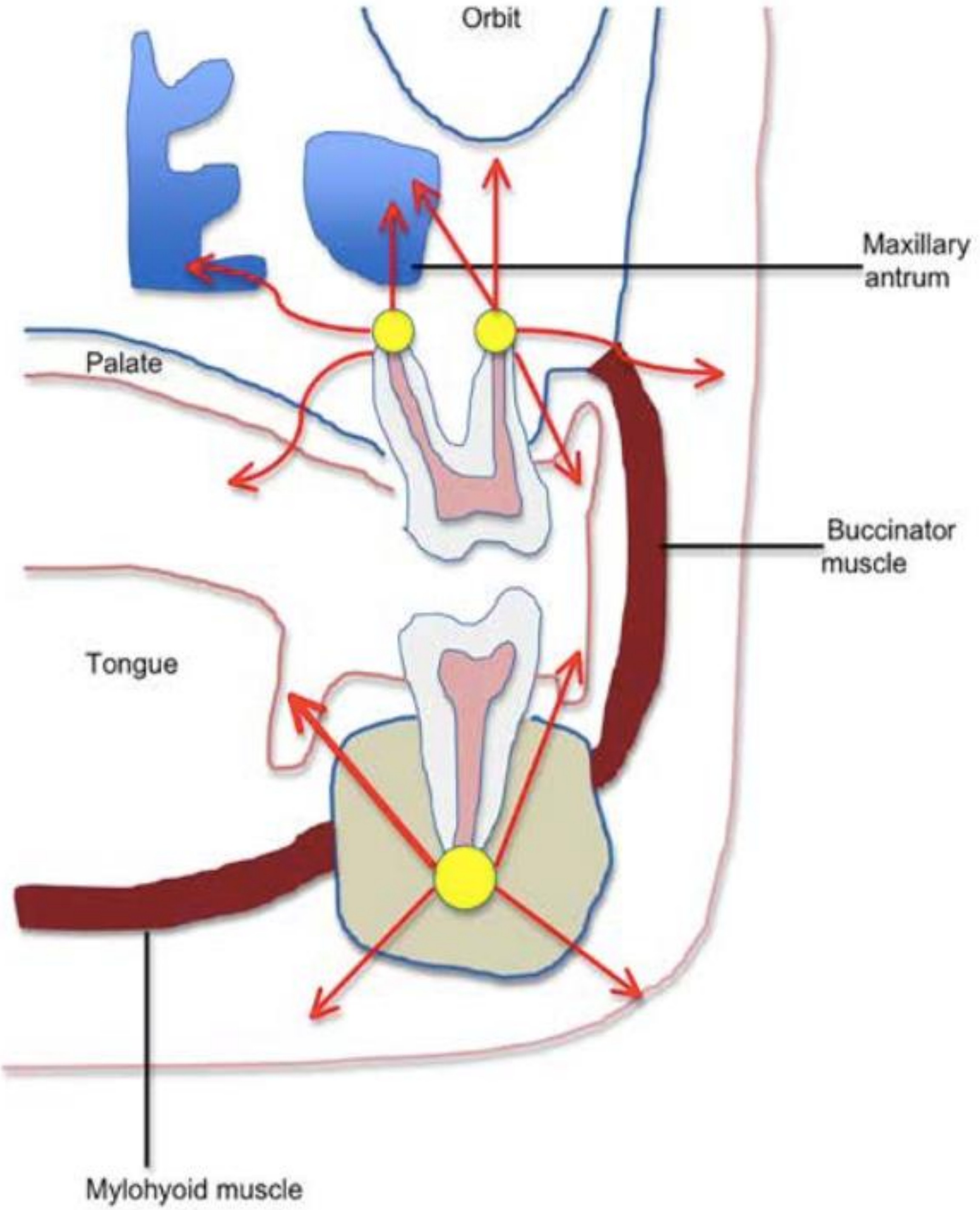
One of the most serious and potentially life-threatening complications

Whatever the cause, spread of infection through the potential spaces in the neck poses a risk to the airway.

It may also result in systemic compromise and cardiovascular collapse.

Furthermore, infection can spread inferiorly into the mediastinal or **pleural cavities**; or superiorly to the peri-orbital or orbital tissues, and *via* the facial vein to the **cavernous sinus.**





Initial assessment of the patient should include the following:

- Taking a comprehensive medical and dental history
- Checking for presence of fever, malaise, fatigue, dizziness or other debility
- Measuring the **pulse** and **temperature** (normal axillary temperature is **36.3-37**)
- Defining **nature location** and **extent of the swelling**
- Identifying the **cause of the infection**

Patients **admitted** to hospital with ***neck space infection*** will undergo blood tests, possible further imaging including ultrasound and/or CT scan, and eventual microbiological investigation. **Where possible, a sample of pus is obtained for microbiological culture and antibiotic sensitivity testing**. Most often, pus is obtained at the time of operation.

The acute odontogenic abscess is usually polymicrobial in nature, comprising facultative anaerobes (for example, viridans streptococci and the *Streptococcus anginosus* group), and strict anaerobes like *Prevotella* and *Fusobacterium* species.

Vital signs

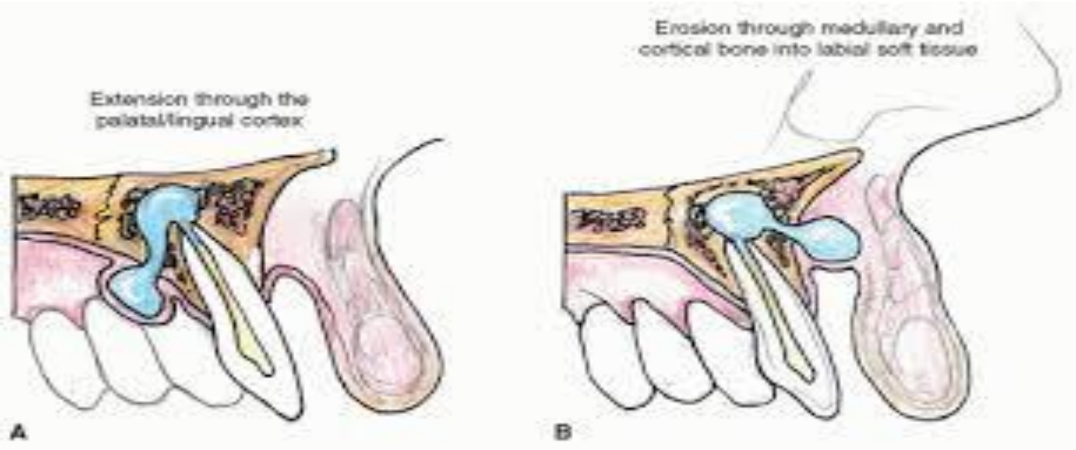
- Septic patients may be tachycardic (pulse rate >90 beats per minute) and pyrexial. If there is an abscess, a swinging pyrexia may be seen.
- The respiratory rate is a sensitive sign that may increase before an abnormality is seen in other vital signs. A rate of >20 breaths per minute is abnormal in an adult.
- The combination of a high respiratory rate, tachycardia, very high or very low temperature, with a very high or very low white blood cell count is the **systemic inflammatory response syndrome** (SIRS).
- Sepsis is defined as the presence of SIRS in addition to a confirmed infective process.
- Septic shock occurs when a septic patient remains hypotensive despite aggressive attempts at restoring the blood pressure.
- Importantly, it is well recognized that the prognosis of septic patients is improved when appropriate treatment is delivered promptly.

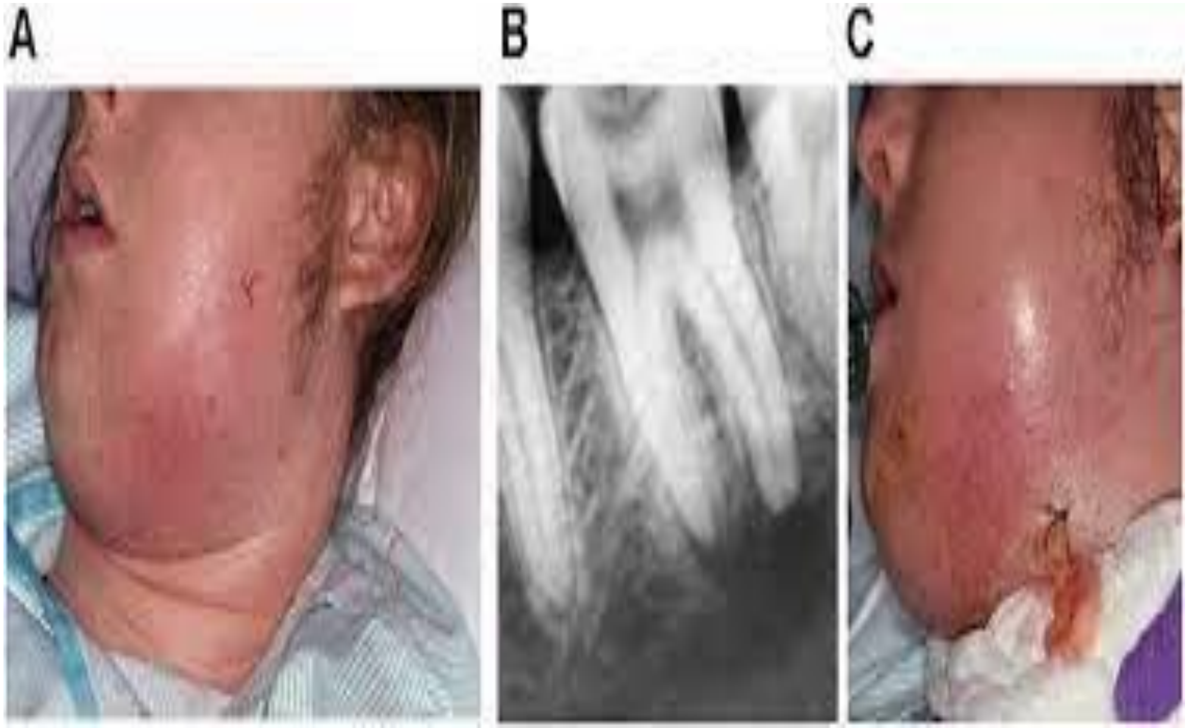
When to refer

Following this assessment the clinician should decide whether treatment can be provided in primary care or whether a referral is necessary for example if there is/ are:

- Signs of septicaemia such as grossly elevated temperature (above 39.5), lethargy, tachycardia

- Spreading cellulitis (rapidly progessing infection)
- Breathing or swallowing compromised
- Severe trismus
- Compromised host defences
- Swellings that may compromise the airway or cause difficulty in swallowing or closure of the eye
- Dehydration
- Significant trismus associated with a dental infection
- Failure to respond to previous treatment
- An uncooperative patient





Principles of the surgical management of deep neck space infection

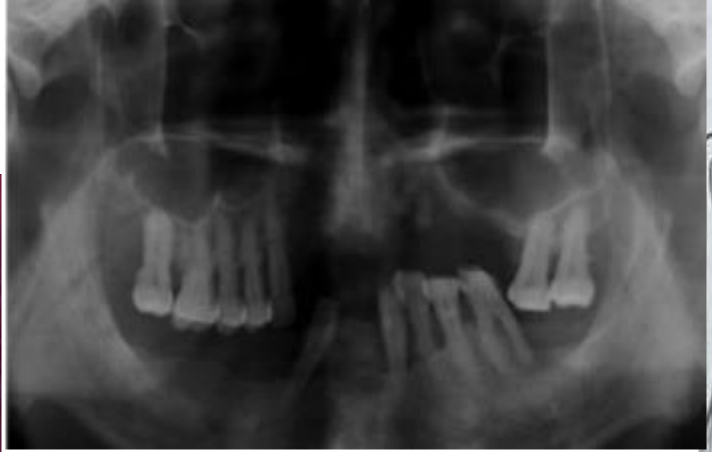
The patient presenting with suspected neck space infection should be assessed immediately for **Airway**, **Breathing**, and **Circulation (ABC).**

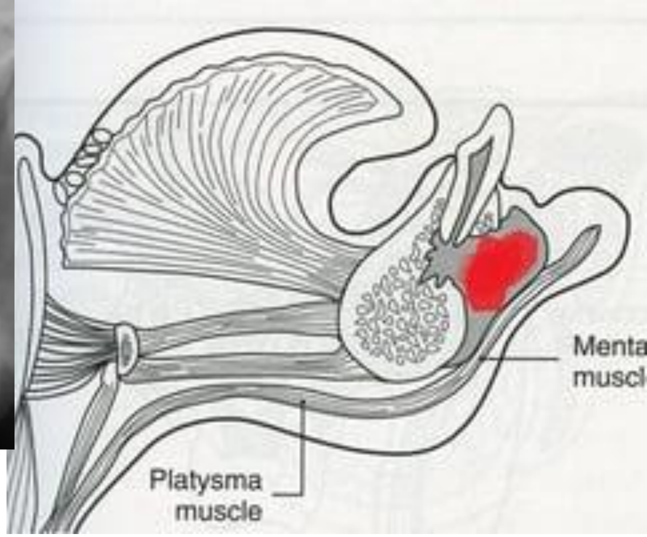
- This gauges the urgency of the need for referral to hospital.
- **A stable patient** with localised swelling and minimal soft tissue involvement is likely to be suitable for early, **local treatment including pulp extirpation** or **extraction of the tooth**, **with or without systemic antibiotics.**
- For those patients referred to hospital, the principles of establishing surgical drainage, removal of the source of infection, and systemic antibiotics also apply.
- Septic patients will be treated aggressively with fluid resuscitation and early, empirical administration of antibiotics.
- Drainage of neck space infections will usually take place in the operating theatre under general anaesthetic.
- Severe airway compromise may necessitate placement of a tracheostomy tube and post-operative admission to intensive care.
- In **Ludwig's angina**, there is often no collection of pus, but surgical exploration of the affected spaces is performed to 'decompress' the neck. Surgical drains are placed until resolution of the infection. **Corticosteroids (eg dexamethasone) may be given to help reduce the oedema associated with these infections.**
- Early recognition and management of dento-alveolar infections is critical because patients (particularly children and immunocompromised patients) can become systemically ill within a short time. If ***untreated,*** local infections can **spread**, **giving rise** to **serious life threatening sequelae.**

THANK YOU

Mandibular buccal vestibule

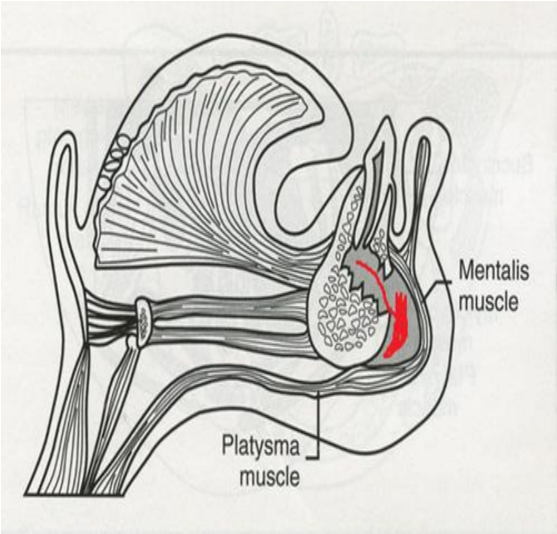
Between buccal cortical plate and mentalis muscle





Mental space Abcess :

Bounded by Mentalis muscle superiorly, Platysma muscle inferiorly These spaces may be created by pathology, e.g., the spread of odontogenic infection. Commonly the origin of the infection is an anterior mandibular tooth with associated periapical abscess which erodes through the buccal cortical plate of the mandibular at a level below the attachment of the mentalis muscle.



Spreading of infection to the surrounding spaces :

Determination of the severity of infection, **evaluation of host defences**, **surgical management**, **medical support**, **administration of antibiotics**, and **frequent evaluations of the patient** are the mainstays of the management of odontogenic infections. Bone, muscle, aponeurosis or fascia, neurovascular bundles, and skin can all act as barriers to the spread of infection. However, no tissue barrier or boundary is so restrictive or confining to universally prevent spread of infection into contiguous anatomical spaces

Three major factors must be considered when determining the **severity of an infection** of the head and neck:

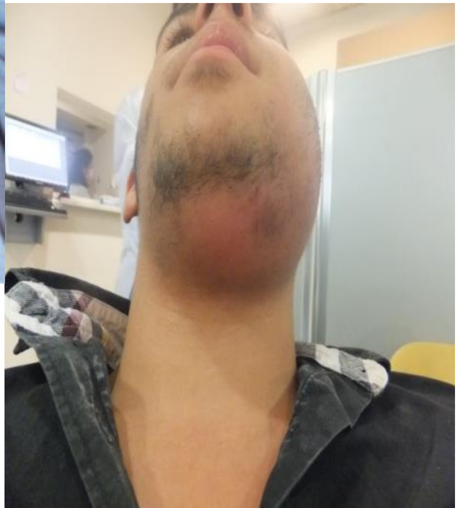
anatomical location,

rate of progression, and

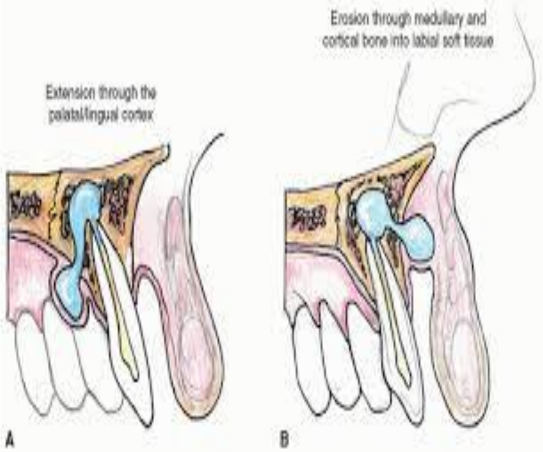
airway compromise

Anatomical Spread of Infection:

Bone, muscle, aponeurosis or fascia, neurovascular bundles, and skin can all act as **barriers** to the spread of infection. However, no tissue barrier or boundary is so restrictive or confining to universally prevent spread of infection into **contiguous anatomical spaces**



There are a number of points for spreading the infection :



Upper lip

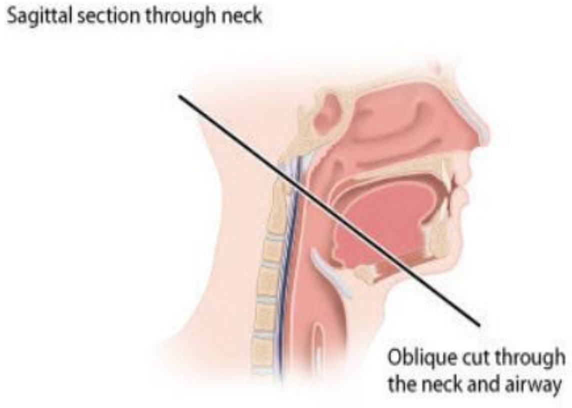
Infection at the base of the upper lip typically originates from the upper anterior teeth. It spreads to the orbicularis muscle, from the labial sulcus between the levator labii superioris muscle and the levator angularis oris muscle.

Canine fossa

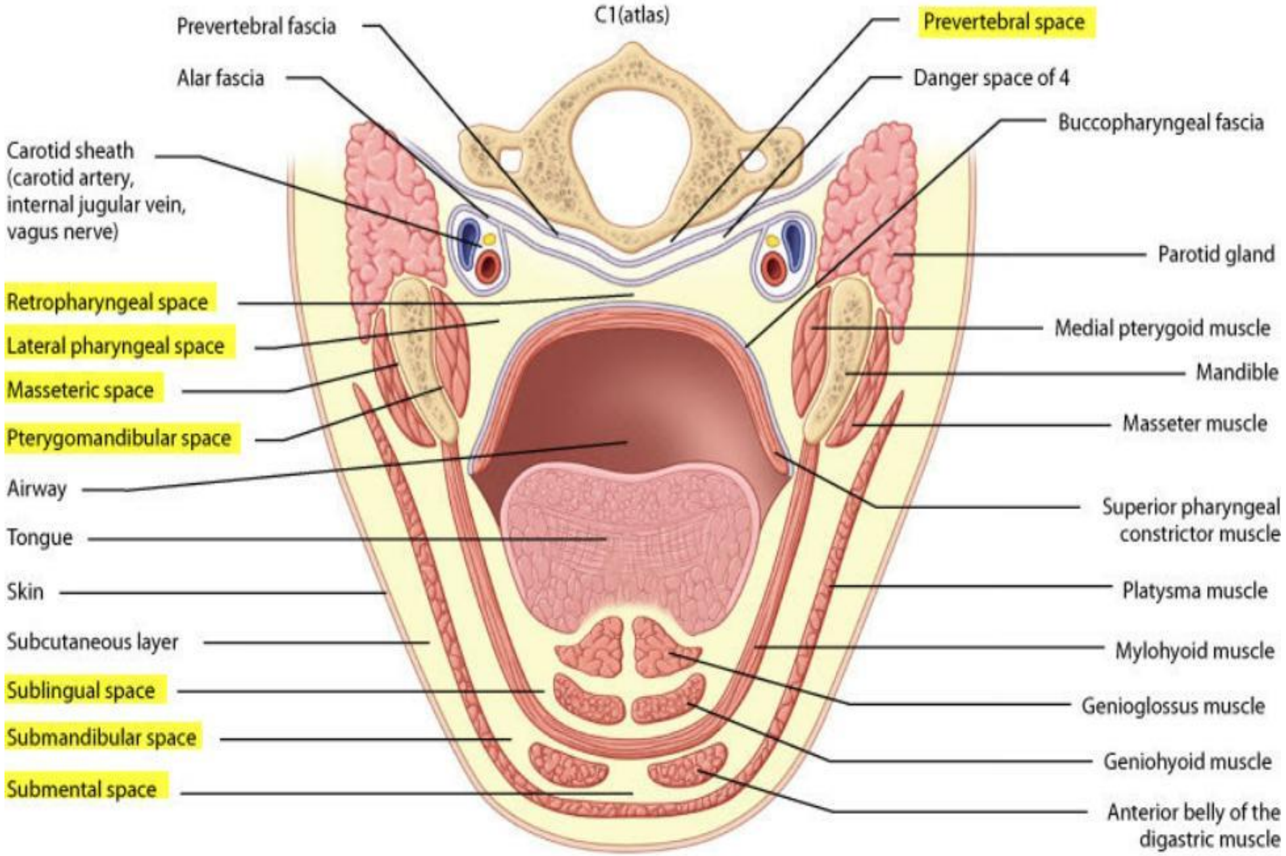
Spread of infection to the canine fossa usually originates from maxillary canine or upper premolar teeth, often presenting above the buccinator muscle attachment. These swellings obliterate the nasolabial fold. This space is in close proximity to the lower eyelids, and therefore early management is essential to avoid circumorbital infection. There is a risk of spread cranially, via the external angular vein, which may then become thrombosed

Buccal space

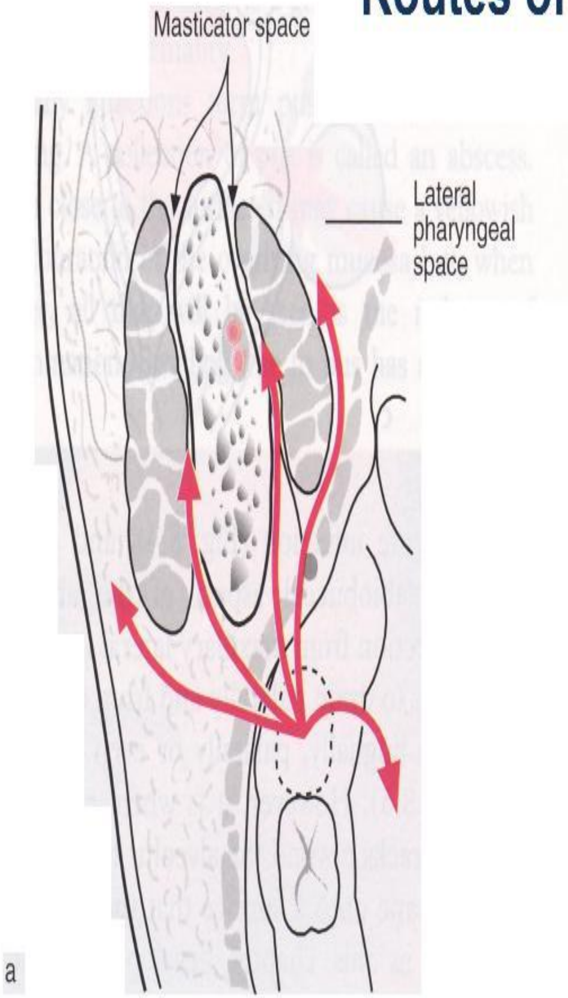
The attachment of the buccinator muscle to the base of the alveolar process can control the spread of infection in the region of the mandibular and maxillary molars. An infection spreads intraorally, superficial to the buccinator muscle, in front of the anterior border of the masseter muscle. Thus, the clinical manifestations of infection in this space are characterized by swelling confined to the cheek. However, infection may spread superiorly, towards the temporal space, inferiorly, to the submandibular space, or posteriorly, into the masseteric space. In some cases, infection may spread to the surface of the skin, leading to fistula formation

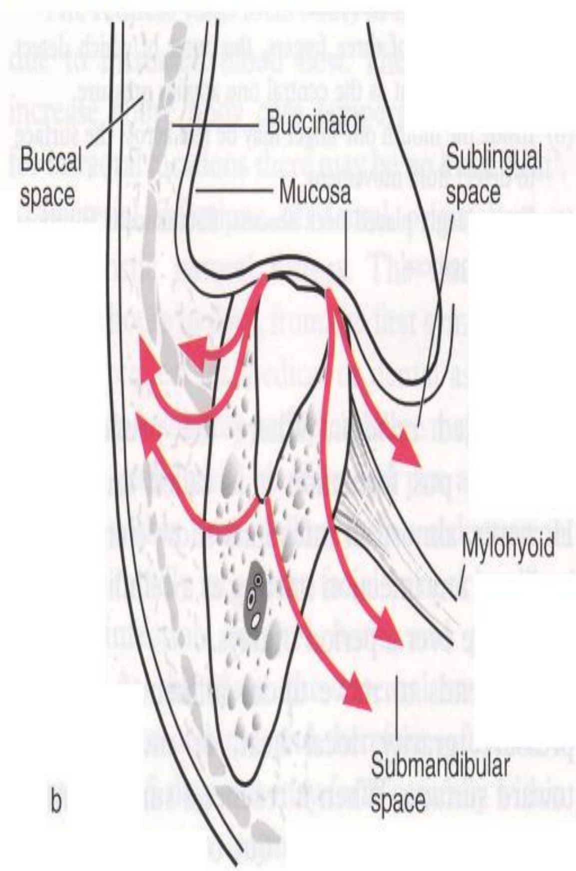


The *Fascial Spaces* seen as a transverse section cut at an oblique angle.



Routes of Spread of Infection from a Lower 3rd Molar

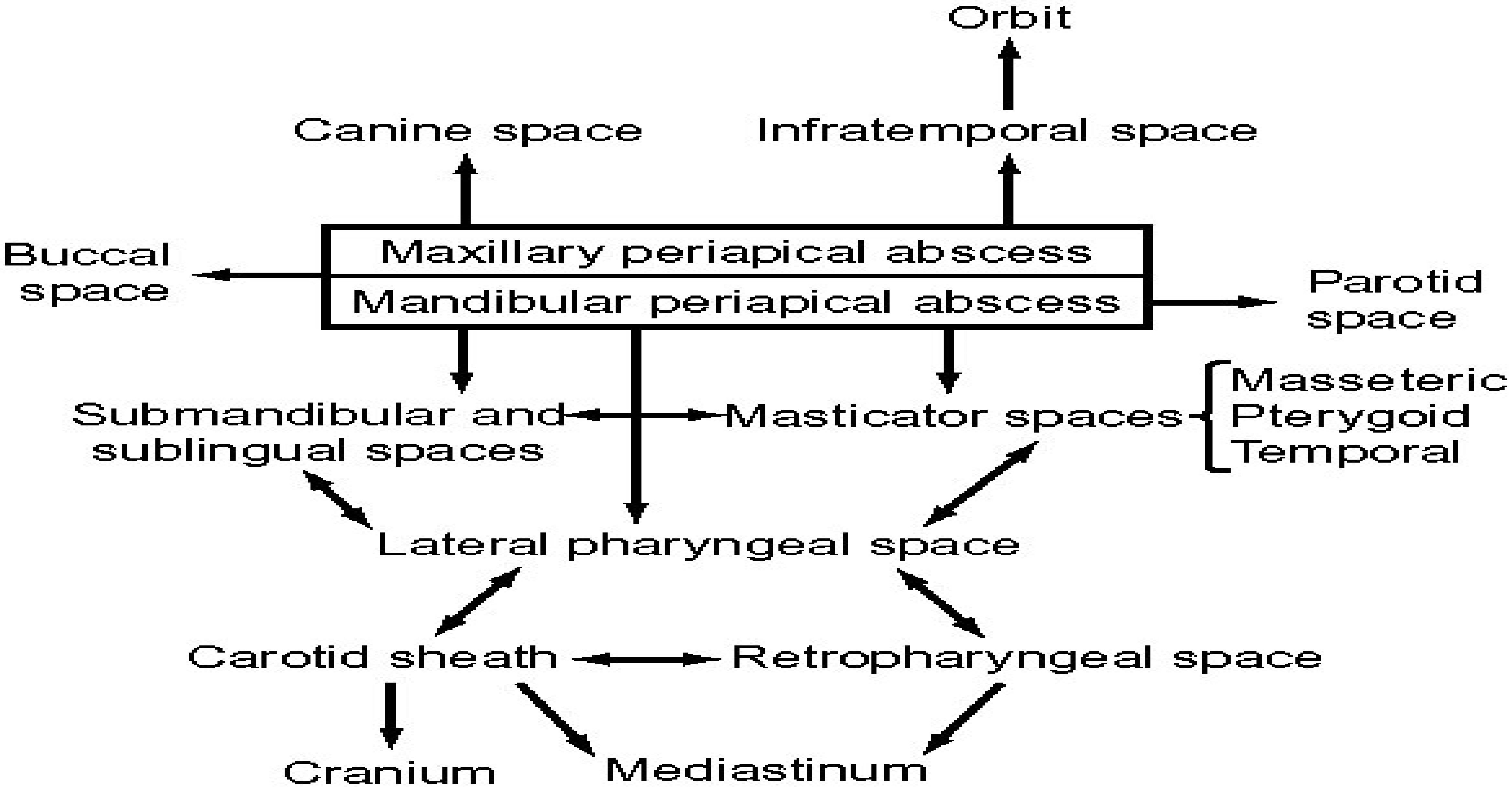




(a) When seen in horizontal section, infection may track laterally into the *buccal space*, posteriorly, either side of the mandible into the *masticatory space*, further medially into the *lateral pharyngeal space*, or lingually into the *sublingual space*.

(b) When seen in coronal sections, routes to the *buccal*, *submandibular* and *sublingual spaces* are visible.

Potential pathways of spread into deep fascial space infections of the head and neck.

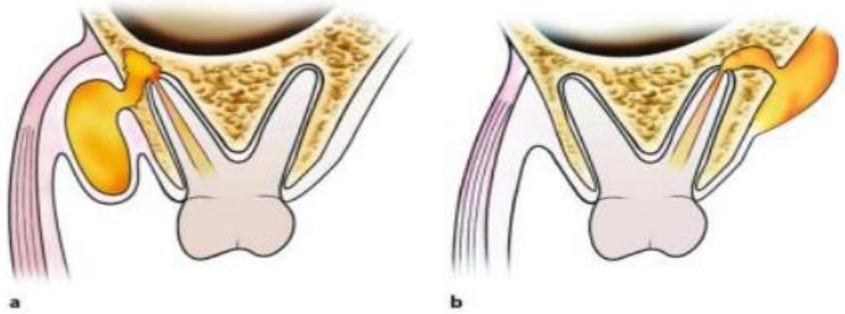


The fascial spaces around the mouth and face, which are most commonly implicated in odontogenic infections, are shown in

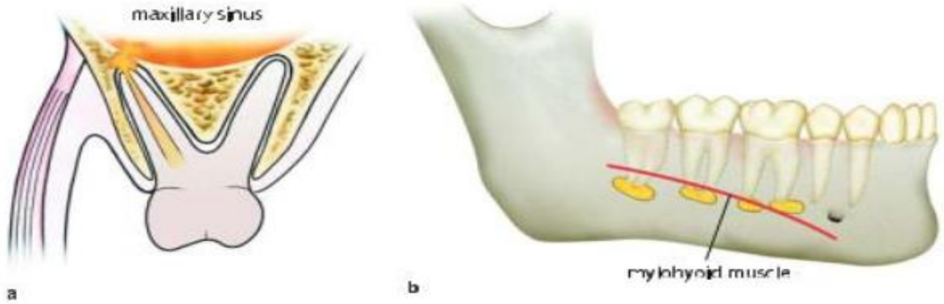
Other non-odontogenic sources of infection include suppurative parotitis, peritonsillar abscess, sinusitis, and mastoiditis.

The main clinical presentation of various orofacial odontogenic infections are summarized in

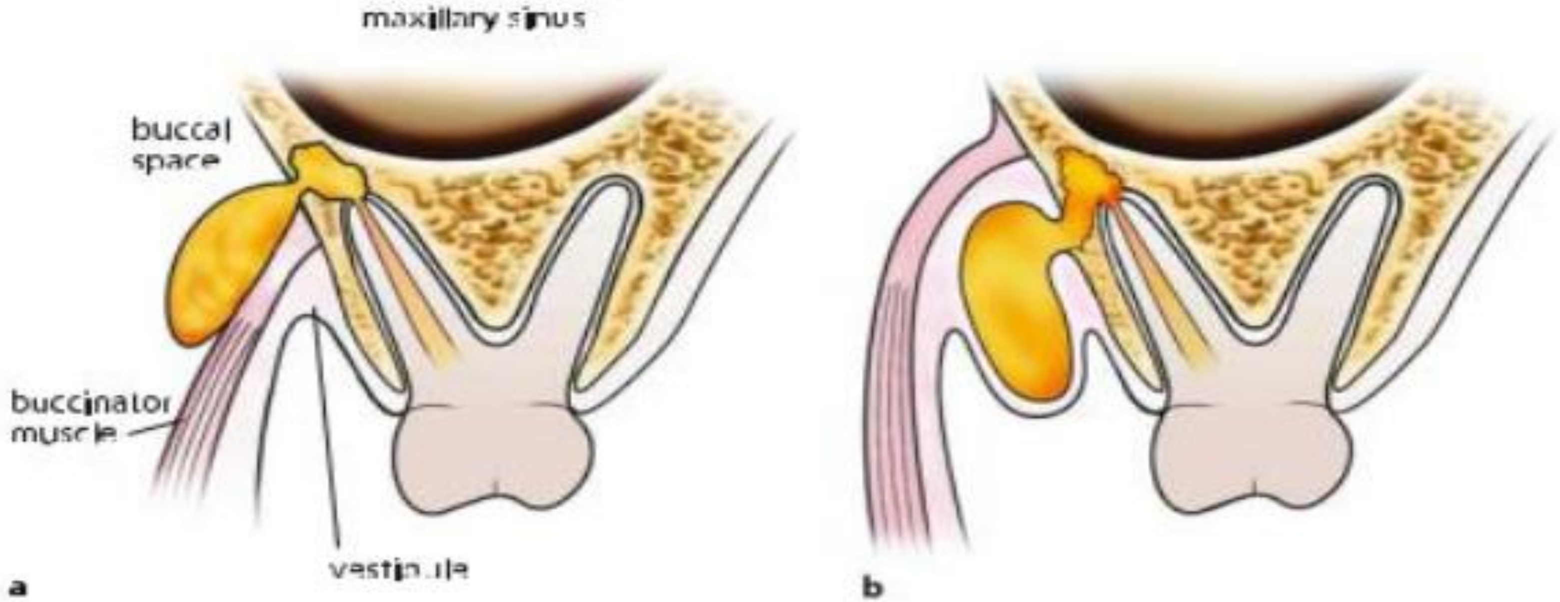
The location of these space infections can be helpful in identifying the underlying infected tooth and suggests the potential pathways for spread into deeper fascial space infections of the head and neck.



Diagrammatic illustrations showing spread of infection (propagation of pus) of an acute dentoalveolar abscess, depending on the position of the apex of the responsible tooth.
a Buccal root: buccal direction.
b Palatal root: palatal direction

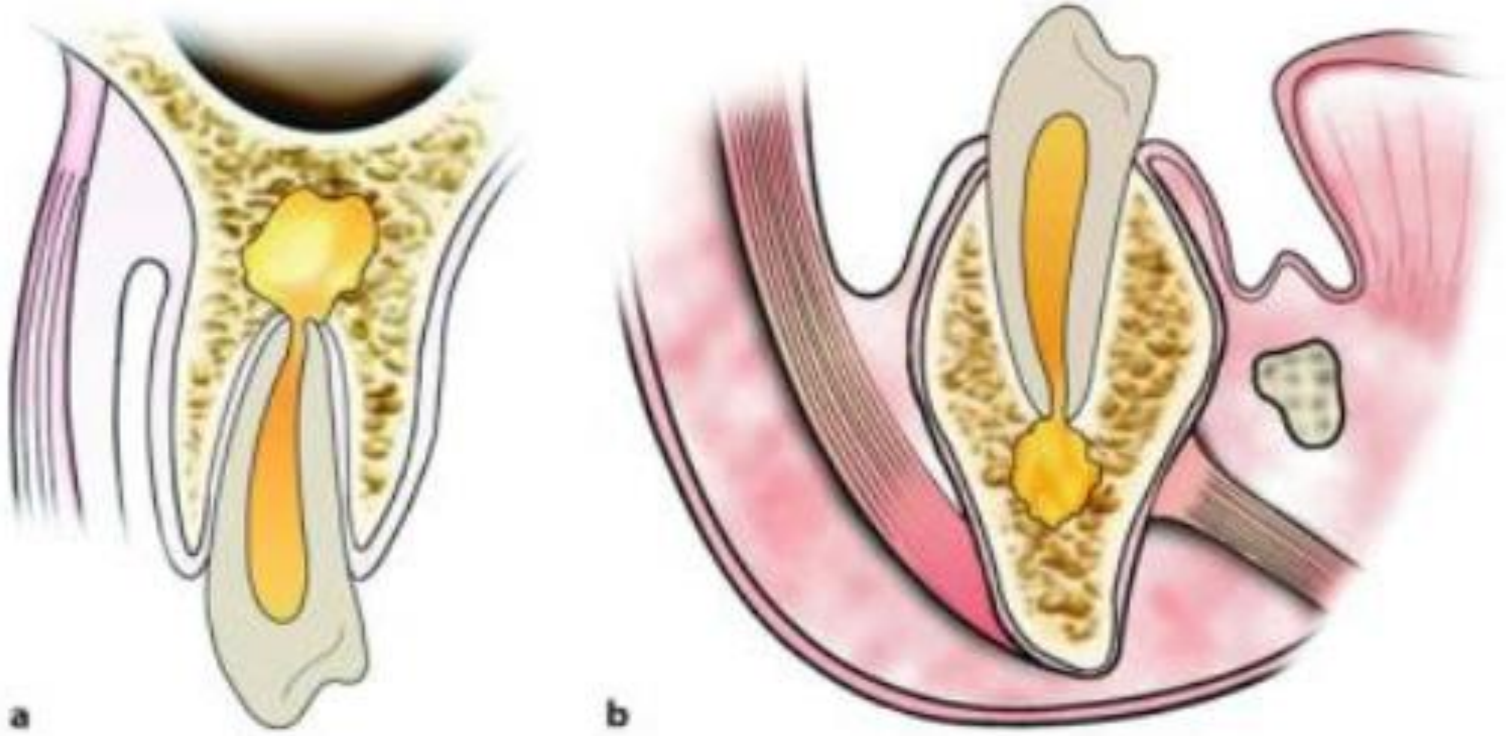


Spread of pus towards the maxillary sinus, due to the closeness of the apices to the floor of the antrum. **b** Diagrammatic illustration showing the localization of infection above or below the mylohyoid muscle, depending on the position of the apices of the responsible tooth

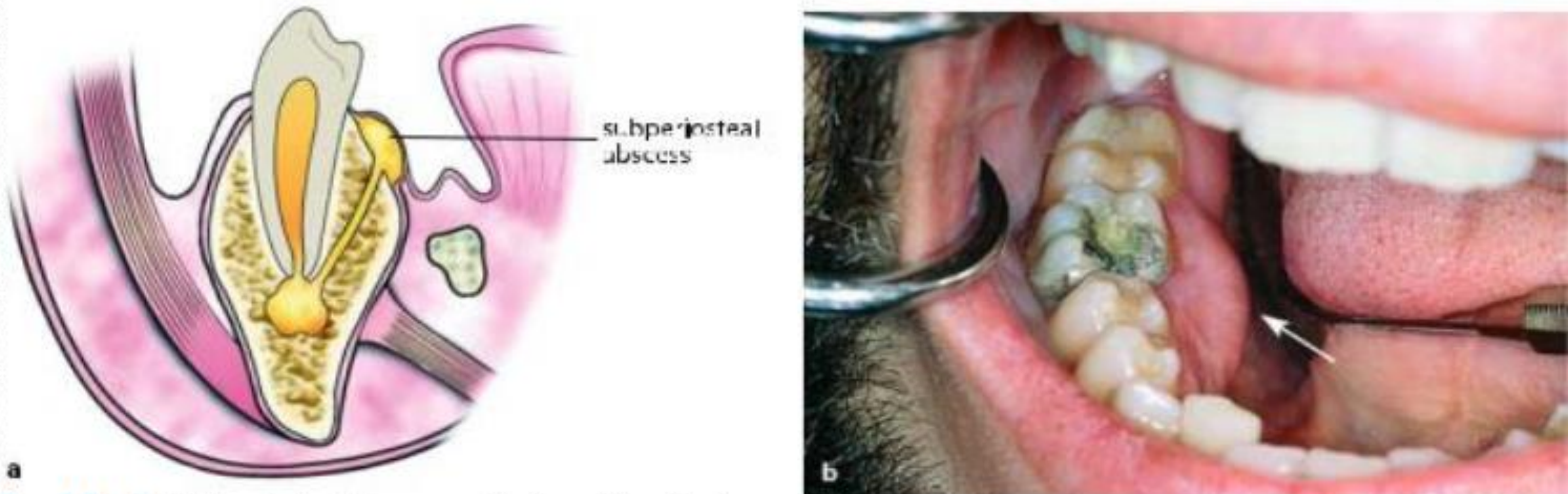


Spread of pus depending on the length of root and attachment of buccinator muscle.
a Apex above attachment: accumulation of pus in the buccal space.
b Apex beneath the buccinator muscle: intraoral pathway towards the mucobuccal fold

Intraalveolar abscess of maxilla
(a) and mandible
(b) Diagrammatic illustrations show accumulation of pus at a portion of the alveolar bone in relation to the periapical region



Subperiosteal abscess with lingual localization



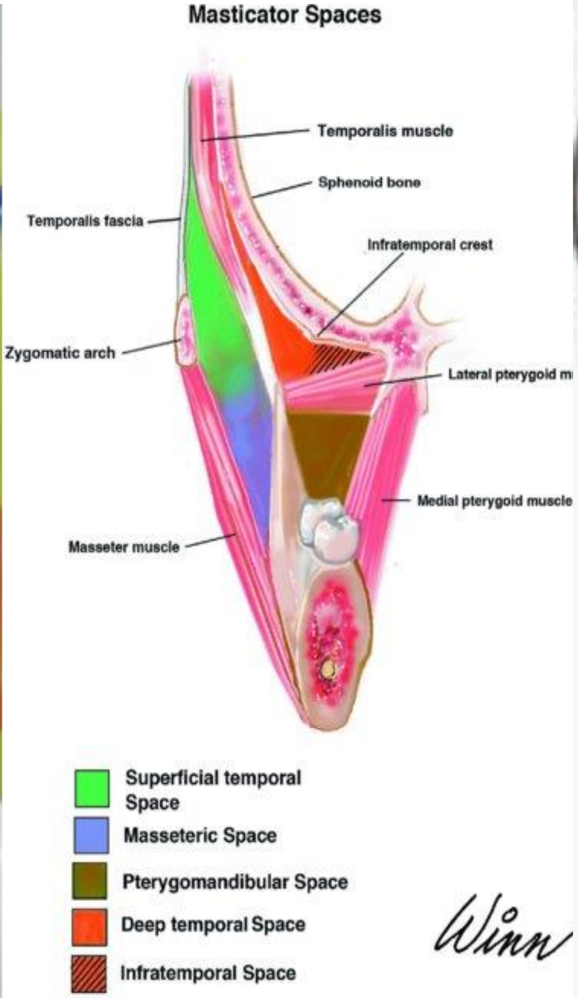
It is in direct communication with the **lateral pharyngeal space posteriorly**. **The temporalis muscle** divides the superior part of this space into two portions, the superficial temporal space, which is bounded by temporalis muscle medially, and the deep temporal space, with the temporalis muscle laterally and the periosteum of the temporal bone medially. Severe trismus due to spasm of the masseter muscle is a characteristic feature of involvement of this fascial space.

Infection abscess drainage in sub masseteric space

Infratemporal space

Extension of infection from maxillary molars can pass into this space. Infection may also spread from the pterygomandibular, parotid, or lateral pharyngeal region to the infratemporal space. The patient then complains of pain, particularly with mouth opening, some dysphagia, and difficulty with lateral mandibular movements. This space is located behind the zygomatic bone posterior to the maxilla and medial to the insertion of the medial pterygoid muscle.

The infratemporal space is bounded superiorly by the greater wing of the sphenoid and is in close proximity to the inferior orbital fissure, with a possible risk of spread of infection to the orbit.



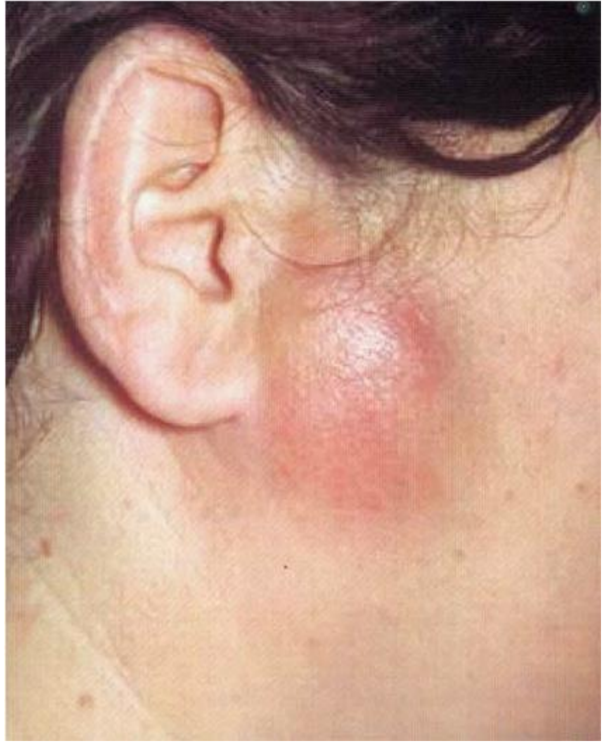
Clinical Presentation. Trismus and pain during opening of the mouth with lateral deviation towardsthe affected side, edema at the region anterior to theear which extends above the zygomatic arch, as well asedema of the eyelids are observed

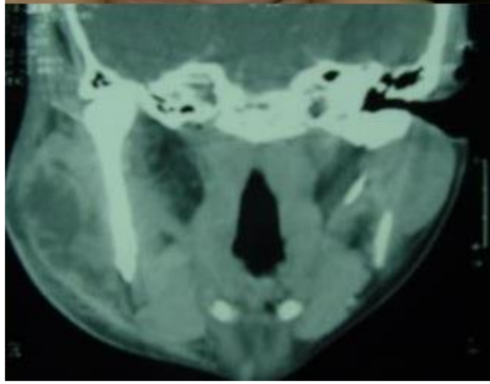
Treatment. The incision for drainage of the abscess is made intraorally, at the depth of the mucobuccal fold,and, more specifically, laterally (buccally) to the maxillarythird molar and medially to the coronoid process,in a superoposterior direction .
A hemostatis inserted into the suppurated space, in asuperior direction.
Drainage of the abscess may beperformed extraorally in certain cases. The incision isperformed on the skin in a superior direction, and extendsapproximately 3 cm.
The starting point of theincision is the angle created by the junction of thefrontal and temporal processes of the zygomatic bone.
Drainage of the abscess is achieved with a curved hemostat,which is inserted through the skin into the purulent accumulation

Infratemporal abscess. **a**Diagrammatic illustration showing the spread of the abscess into the infratemporal space. **b** Clinical photograph of an infratemporal abscess. Swelling of the region of the right zygomatic arch and edema of eyelids. **c** Incision at the depth of the vestibular fold for incision and drainage of an infratemporal abscess

Parotid space

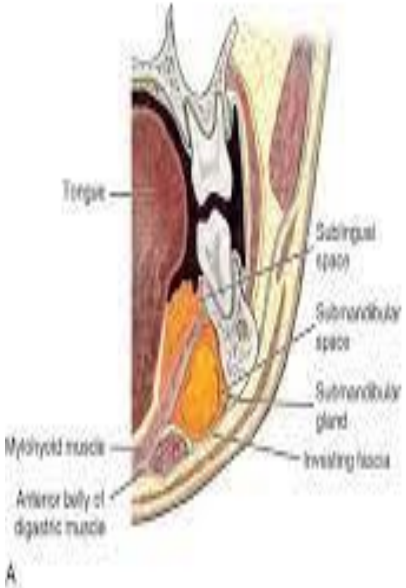
Involvement of this space may be an extension of infection in the **middle ear or the mastoid region**. Infection in the masseteric or the lateral pharyngeal space may also spread to the parotid region. Thus, the most characteristic feature of involvement of this space is swelling of the parotid gland region, below the ear lobe. This space contains several important structures that may be affected by infections. **These include the 7th cranial nerve**, the auriculotemporal nerve, the facial vein, the parotid lymph node, and, more deeply, the external carotid with its branches.

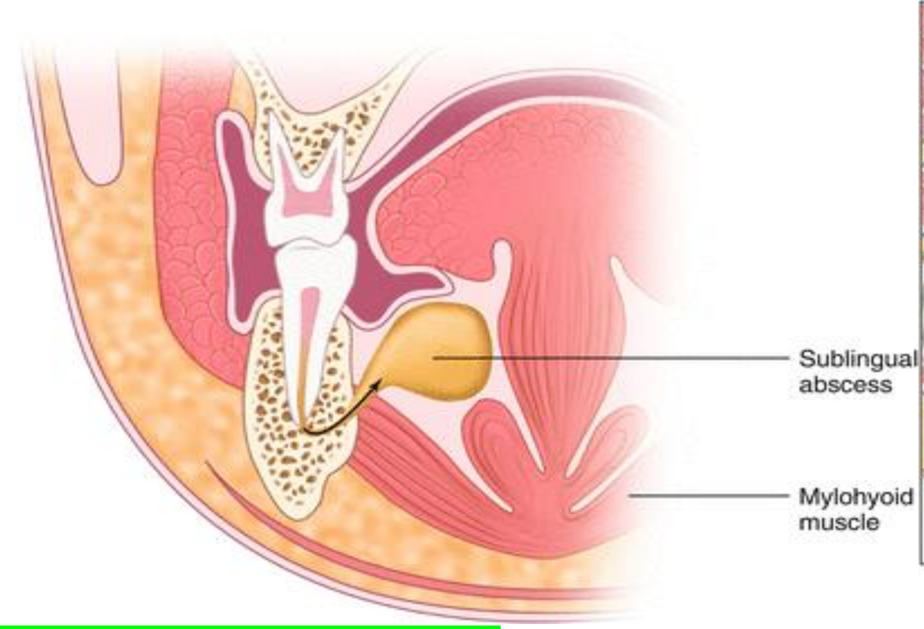


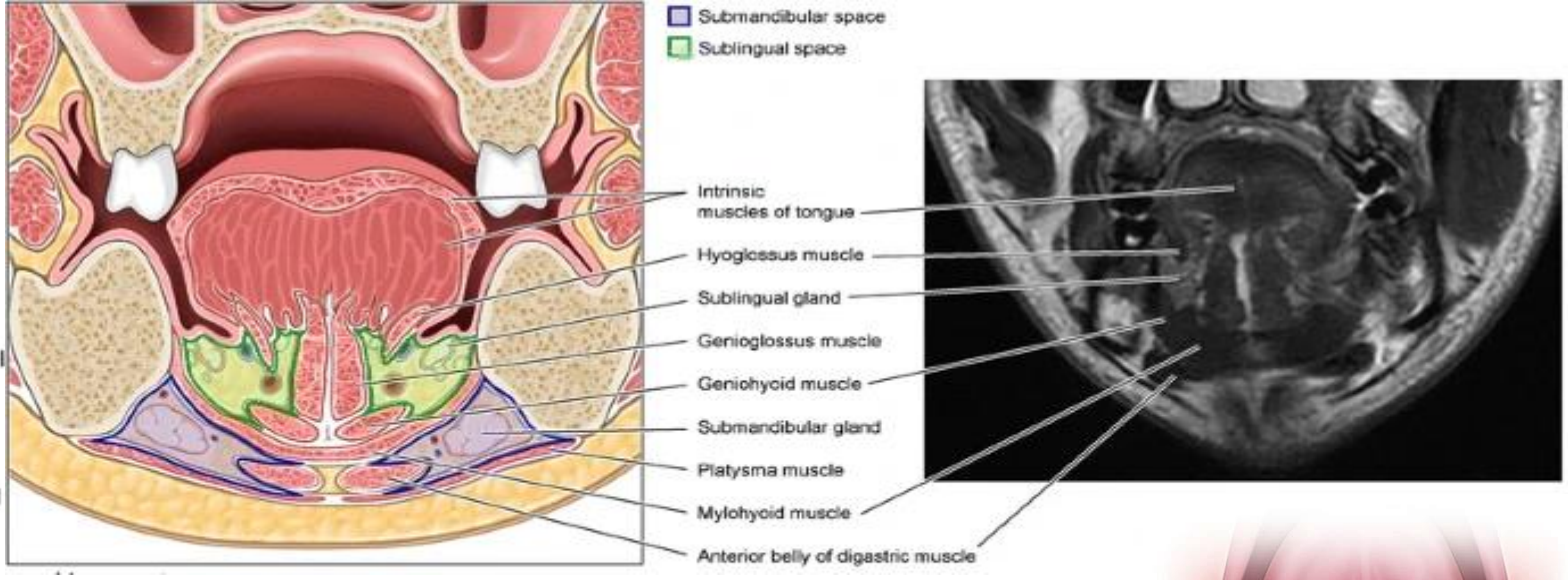


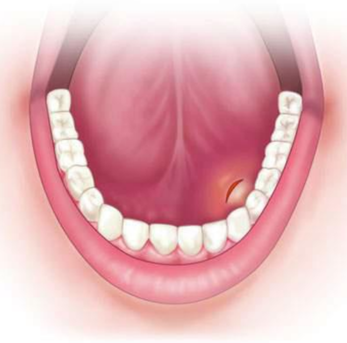
Submandibular space

This space is located below the mylohyoid muscle, medial to the ramus and the body of the mandible. It is bounded anteriorly by the attachments of the anterior belly of the digastric muscle and posteriorly by the posterior belly of digastric muscle and the stylomandibula









Sublingual space infection

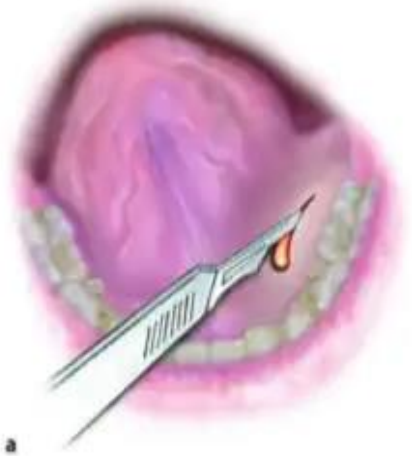
Anatomic boundaries:

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.

Signs and symptoms of a sublingual space infection might include a **firm, painful swelling in the anterior part of the floor of the mouth**. A sublingual abscess may elevate the tongue and cause ***drooling*** or ***dysphagia (difficulty swallowing)***. There is usually little swelling visible on the face outside the mouth

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**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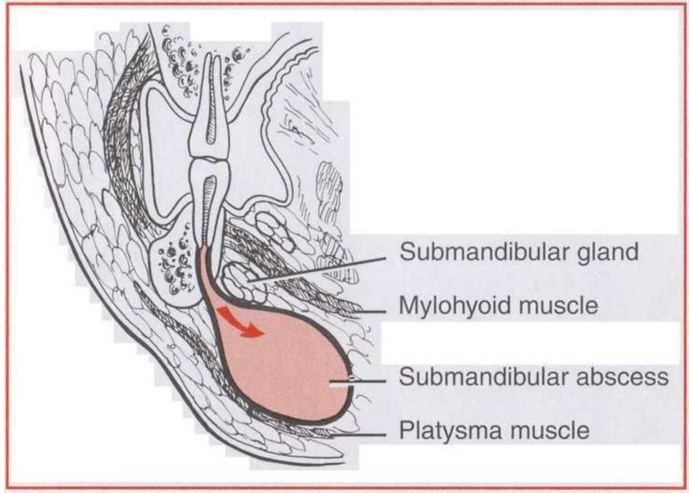
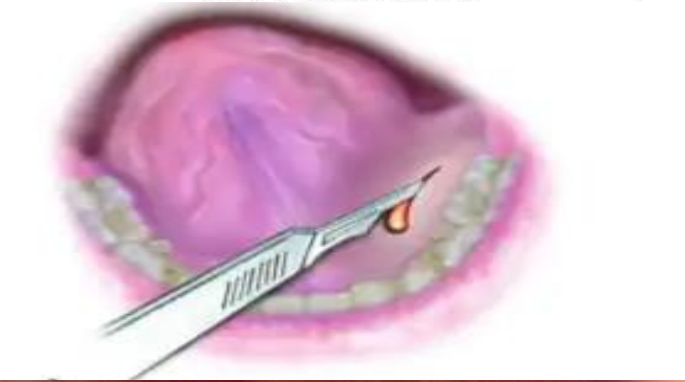
Clinically : there is erythematous swelling of the floor of the mouth that may extend through the midline since the barrier between the two sublingual spaces is weak, usually there is elevation of the tongue.

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.

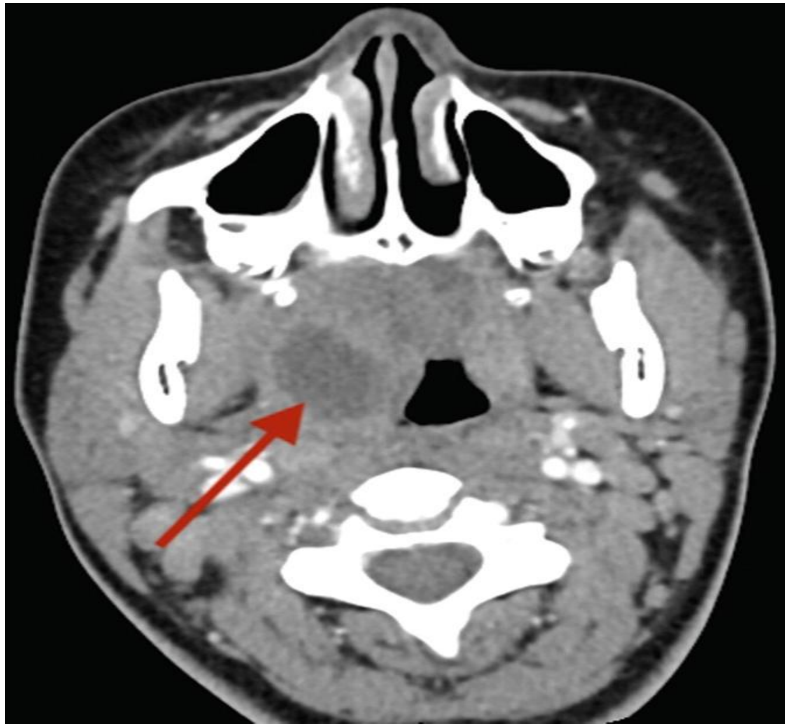


Submandibular Space. This space lies between the mylohyoid muscle and skin and superficial fascia. Primarily 2nd & 3rd molars infect it. muhadharaty.com



Parapharyngeal space infection

Parapharyngeal abscesses can also cause **trismus** and neck swelling above the level of the hyoid bone. Anterior parapharyngeal space abscesses may result in a bulging appearance of the tonsil and lateral pharyngeal wall associated with swelling in the areas of the parotid gland and angle of the mandible.



Parapharyngeal abscesses can also cause trismus and neck swelling above the level of the hyoid bone. Anterior parapharyngeal space abscesses may result in a bulging appearance of the tonsil and lateral pharyngeal wall associated with swelling in the areas of the parotid gland and angle of the mandible. In contrast, posterior parapharyngeal space abscesses result in posterior pharyngeal wall swelling. Trismus is less frequent and, when present, less severe. Posterior parapharyngeal space abscesses are also associated with a higher incidence of sepsis due to their proximity to the carotid sheath. Despite the numerous clinical findings that can use to diagnose and potentially differentiate between deep neck space infections, most cases will require imaging. The imaging modality of choice is a contrast-enhanced CT scan of the neck. Some patients will not be able to tolerate lying supine and may require endotracheal intubation prior to the imaging procedure

Needle aspiration or incision and drainage is a common method of treating abscesses in the head and neck, although intratonsillar abscesses are usually not treated surgically unless the tonsils become obstructive. Peritonsillar abscesses may be treated in a conscious patient using either needle aspiration or incision and drainage. **General anesthesia is necessary for surgical drainage of other deep neck space abscesses**. New evidence suggests that small PTAs, particularly those that are not associated with severe symptoms, can be treated medically, avoiding an incision and drainage procedure altogether .Similarly, some small, clinically mild, parapharyngeal space abscesses ,and RPAs .can be treated medically with close follow-up in case a surgical procedure becomes necessary due to progression of the illness.

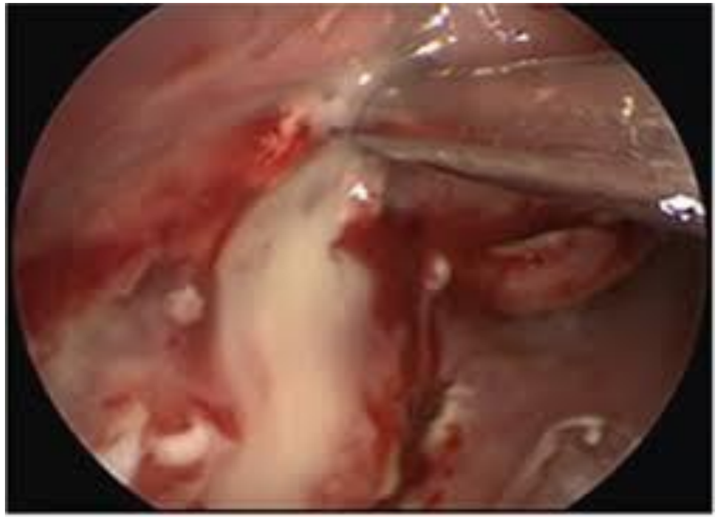
Although suppurative infections of the deep neck spaces are often regarded as being more acute and in need of surgical drainage, nonsuppurative infections can be just as severe and, in some cases, more serious than deep neck space abscesses. One of these conditions is Ludwig's angina.

This condition refers to a rapidly spreading cellulitic infection of the submental, sublingual, and submandibular areas, typically due to an infection originating in the teeth. The swelling of the sublingual space displaces the tongue superiorly and posteriorly. The swelling impairs speech and ability to swallow. As the infection progresses, the patency of the airway becomes compromised.

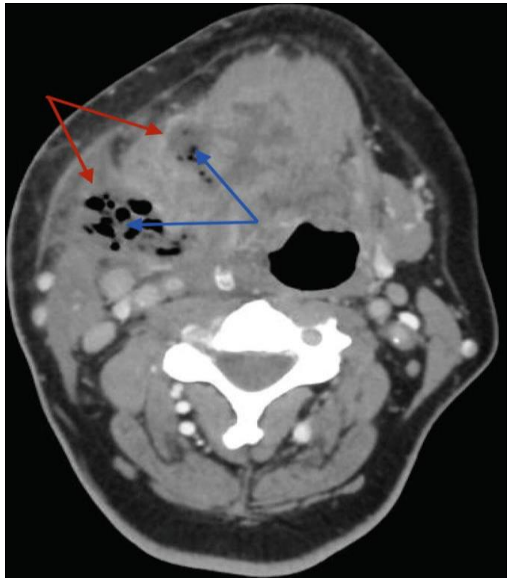
On physical examination, the patient has a muffled voice and appears distressed. Trismus may prevent examination of the oropharynx. The structures of the superior neck are indurated with a marked elevation of the tongue and floor of mouth. Ludwig's angina is a surgical emergency. The treatment priority is establishing a protected airway, typically by tracheostomy. Once the airway is protected, the infection is incised and drained and the area debrided as necessary.

Broad-spectrum intravenous antibiotics are used. Ideally the dental source of the infection is also addressed, but severe trismus typically prevents access during the incision and drainage procedure.

Intraoperative findings. (a) There was peritonsillitis containing mucus and pus from the posterior pillar on the right side. (b) Incision at the left lateral and posterior pharyngeal walls. A large amount of pus flowed out from the peritonsillar and lateral pharyngeal wall



Shown is an axial CT scan of the neck revealing hypodense areas without peripheral enhancement (red arrows) and the abnormal presence of gas pockets (blue arrows) within the soft tissue. Surgical debridement of this area revealed necrotic tissue, confirming a diagnosis of necrotizing fasciitis



Submental space:

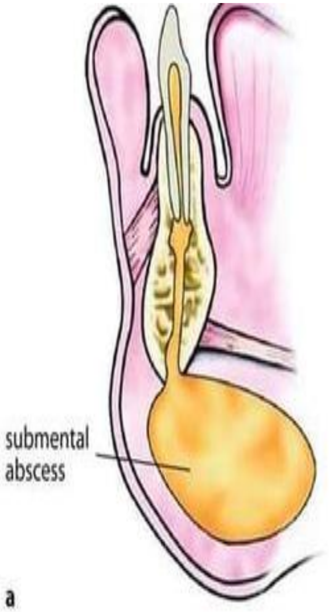
Infection can **result directly** due to infected mandibular incisor or **indirectly** from the submandibular space

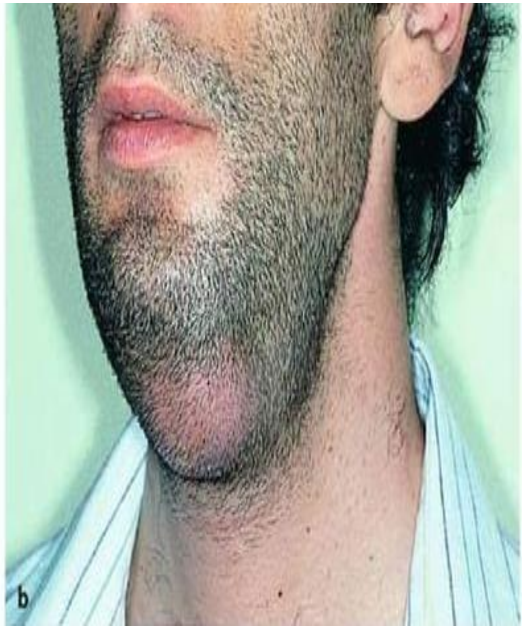
Space located between the anterior bellies of the digastric muscle laterally, deeply by the mylohyoid muscle, and superiorly by the deep cervical fascia, the platysma muscle, the superficial cervical fascia, and the skin

Dependent drainage of this space is performed by placing a horizontal incision in the most dependent area of the swelling extraorally with a cosmetic scar being the result

Treatment:

All patients had undergone surgical drainage for impending airway compromise and required airway support postoperatively. It was not always evident to the investigators why a particular strategy was chosen, and these groups were not likely identical. Airway loss occurred more commonly in the intubated patients, but this characteristic was not statistically significant.





Maxillary Sinusitis of Endodontic Origin

Many factors and conditions can contribute to the development of odontogenic sinusitis. These include spread of endodontic infection in the maxilla, periapical cysts, granulomas, and displacement of foreign bodies into the sinus such as endodontic materials, tooth fragments, implants, or augmentation grafts.

The AAE recently published a position statement distinguishing the **difference** between maxillary sinusitis of dental origin (MSDO) and maxillary sinusitis of endodontic origin **(MSEO)**, indicating that the treatment and management of the two may differ. MSEO has been characterized as sinusitis that is secondary to endodontic periradicular disease and excludes other **dental etiologies** of odontogenic sinusitis.

Patients with MSEO frequently present **without dental symptoms or complaints** and typically seek care from **their primary care physician or ear/nose/throat (ENT) physician due to their sinonasal symptoms**.

Therefore, if mucosal abnormalities exist or there is obstruction of the sinus, **physicians and dentists must not rule out an odontogenic source of infection**.

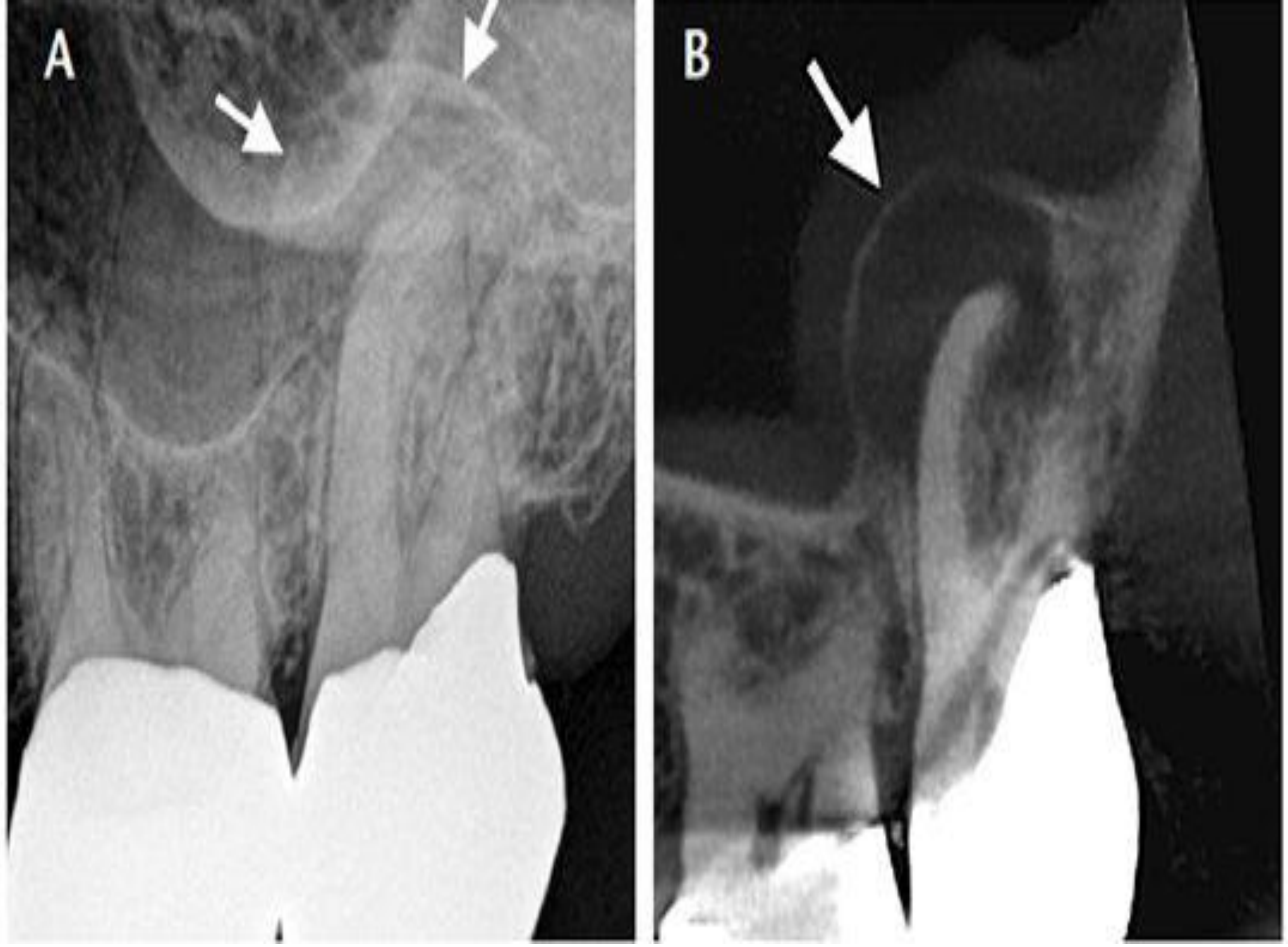
Otolaryngologists and dentists should always suspect an odontogenic origin for sinusitis cases and refer to an endodontic specialist if needed.

Biofilms involved in **odontogenic sinusitis** are generally characterized as polymicrobial. Acute bacterial sinus infections of non-odontogenic sources typically involve nasal cavity aerobic commensals, including ***Staphylococcus aureus***, ***Haemophilus influenzae***, ***Moraxella catarrhalis***, and others.**In odontogenic** sinusitis, the sinonasal environment changes to that characteristic of chronic sinus infections with predominance of anaerobic bacteria.The most common isolates found were oropharyngeal flora that were also **predominate in periodontal and endodontic infections**. These included ***Peptostreptococcus, Fuso-bacterium, Prevotella spp.*, and *Porph-yromonus spp***.The differences in microbial communities and source of infection explains why patients with odontogenic sinusitis require different approaches to treatment

Radiographic Examination:

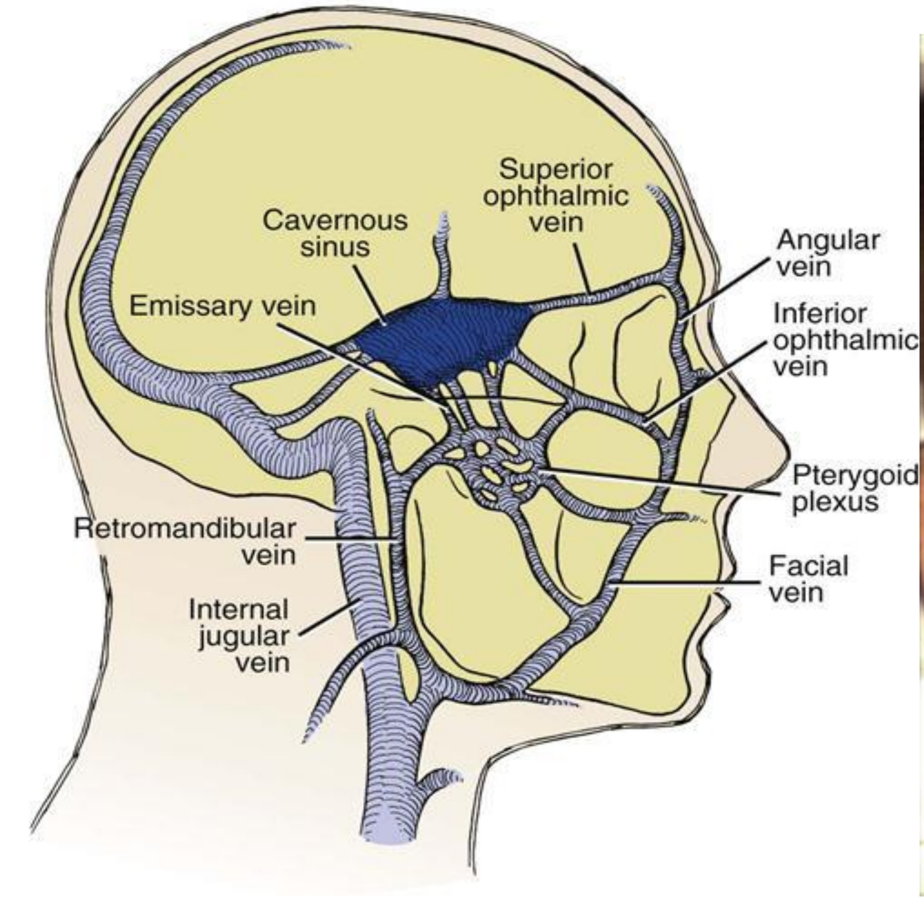
While periapical radiographs are the most widely used imaging modality in endodontics, the posterior maxilla presents significant and unique interpretation challenges when using conventional two-dimensional imaging. Anatomic structures — such as the zygoma, palatal process, maxillary sinus and buccal cortical plate — are often superimposed onto the dental roots, obscuring or concealing periapical infection. Conventional periapical radiographs also do not consistently reveal mucosal thickening or fluid in sinuses, which are of important diagnostic value in MSEO.

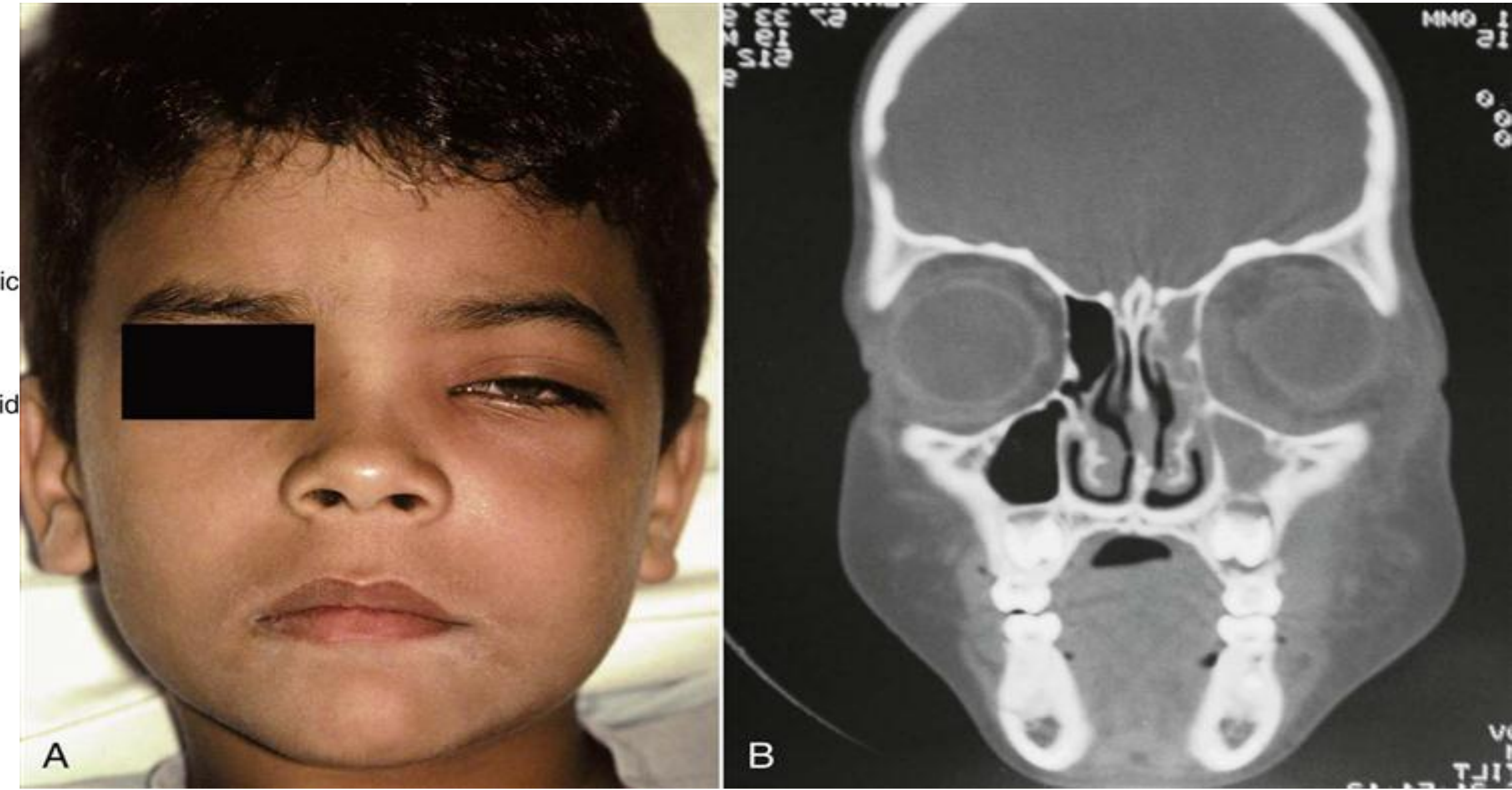
The increased availability of CBCT in dental offices in recent years has allowed for improved recognition of odontogenic sinusitis. In all three of the cases in this study, periapical infection was missed on routine dental examination and was not discovered until CBCT imaging was performed. Once the source of infection was established and the appropriate treatment was performed, the patient's dental and sinonasal symptoms resolved. Collectively, the literature strongly supports interdisciplinary action among physicians, dentists, and dental specialists to provide a patient-centered approach for treatment and management of maxillary odontogenic sinusitis**Further progression of the disease process can cause a partial or total sinus obstruction**



Periapical radiograph of a left maxillary second molar with a periapical osteoperiostitis or "halo" lesion (arrows) over the mesiobuccal root (A); the clinical examination confirmed pulpal necrosis. Sagittal cone beam computed tomogram of the same necrotic molar displaying the periapical osteoperiostitis (arrow), with associated mucosal edema of the left maxillary sinus (B).

Periapical or periodontal infections of posterior maxillary teeth may erode superiorly through the floor of the maxillary sinus. Approximately 20% of cases of **maxillary sinusitis are odontogenic**.
Odontogenic maxillary sinus infections may also spread superiorly through the ethmoid sinus or the orbital floor to cause secondary periorbital or orbital infections.
Periorbital or orbital infection rarely occurs as the result of odontogenic infection, but when either does occur, the **presentation is typical**: redness and swelling of the eyelids and involvement of the vascular and neural components of the orbit. This is a serious infection and requires aggressive medical and surgical intervention from an oral-maxillofacial surgeon and sometimes other specialists.





CLINICAL EXAMINATION

A thorough clinical endodontic examination is essential for diagnosing or ruling out a potential endodontic source.When diagnosing a possible endodontic etiology in patients with sinusitis, the clinician must look carefully for any teeth with pulpal necrosis, and evaluate all prior endodontic treatments for possible failure in the suspected quadrant. Because MSEO is a bacterial disease, typically only teeth with an **infected necrotic pulp** or **failing endodontic treatment** will cause significant **sinonasal disease** or **sinonasal symptoms**. When examining maxillary posterior teeth with existing root canal treatment, providers must carefully look for any untreated or suboptimally filled canals, inadequate core restorations, or leaking coronal restorations that may provide evidence of endodontic failure and a bacterial source for MSEO.

TREATMENT

The objectives for treating MSEO are removal of pathogenic microorganisms, their by-products, and pulpal debris from the infected root canal system that are responsible for the sinus infection. Appropriate treatment options include nonsurgical root canal therapy, **periradicular surgery** (when indicated), intentional replantation, or extraction of the infected tooth. Patients should be informed of all treatment options and the prognosis of each, including the risks of no treatment.

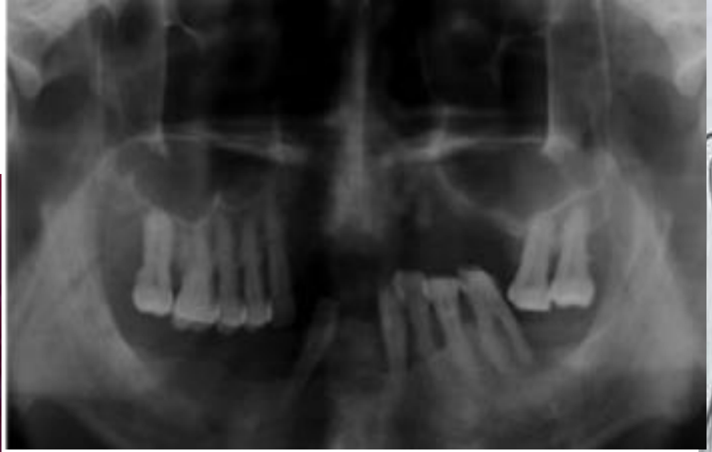
Clinicians performing endodontic treatment in the posterior maxillary dentition should have extensive knowledge of maxillary root canal anatomy, the necessary armamentarium (including magnification), and requisite clinical skill considering the anatomic complexities and challenges in this region. Maxillary molars typically have the most complex anatomy in the dentition, and inadequate root canal treatment, particularly missed mesiobuccal canal systems, is a common cause of endodontic failure in maxillary molars.

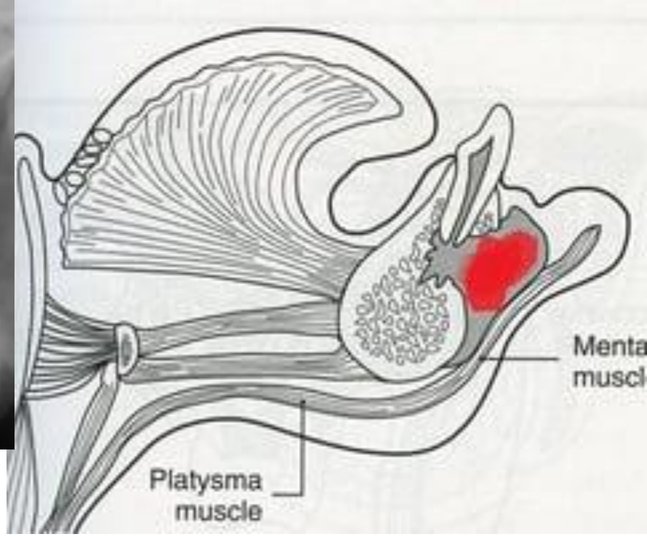
The close anatomic proximity of maxillary molar root apices to the floor of the maxillary sinus can lead to persistent MSEO if canals are left untreated or root canal failure occurs in these teeth. Endodontists are specialists in managing complex root canal systems and should be heavily relied upon for root canal treatment of maxillary molars.

Use of systemic antibiotics to manage MSEO should follow the guidelines set forth in the *AAE Guidance on the Use of Systemic* ***Antibiotics in Endodontics***. Apart from spreading infections, antibiotic therapy is unwarranted in the treatment of MSEO and ineffective as a definitive solution. While antibiotic therapy may offer temporary relief of symptoms by improving sinus clearing, its sole use is inappropriate without definitive debridement and disinfection of the root canal system.

Mandibular buccal vestibule

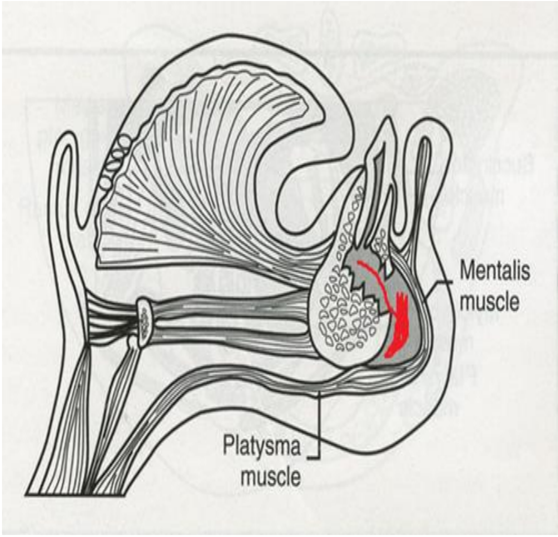
Between buccal cortical plate and mentalis muscle





Mental space Abcess :

Bounded by Mentalis muscle superiorly, Platysma muscle inferiorly These spaces may be created by pathology, e.g., the spread of odontogenic infection. Commonly the origin of the infection is an anterior mandibular tooth with associated periapical abscess which erodes through the buccal cortical plate of the mandibular at a level below the attachment of the mentalis muscle.

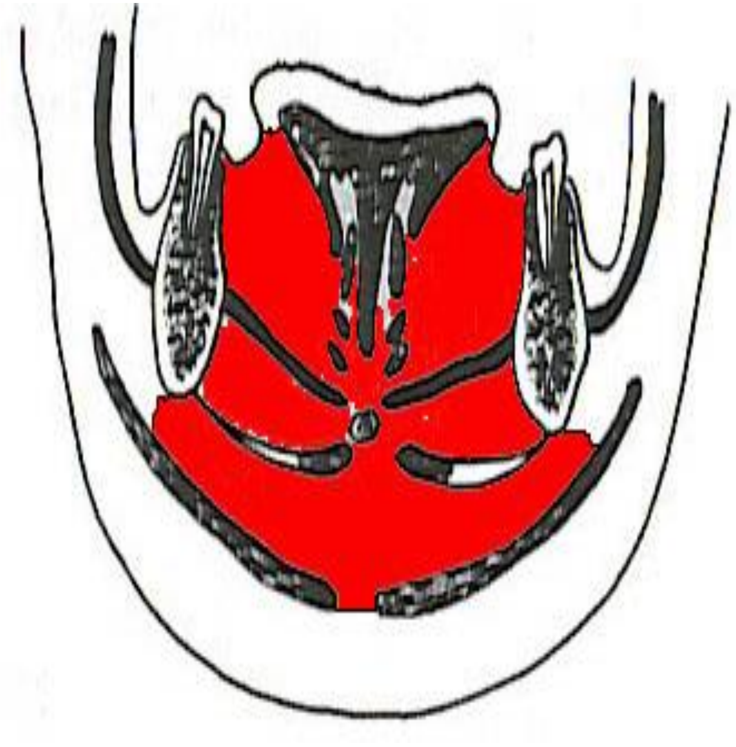


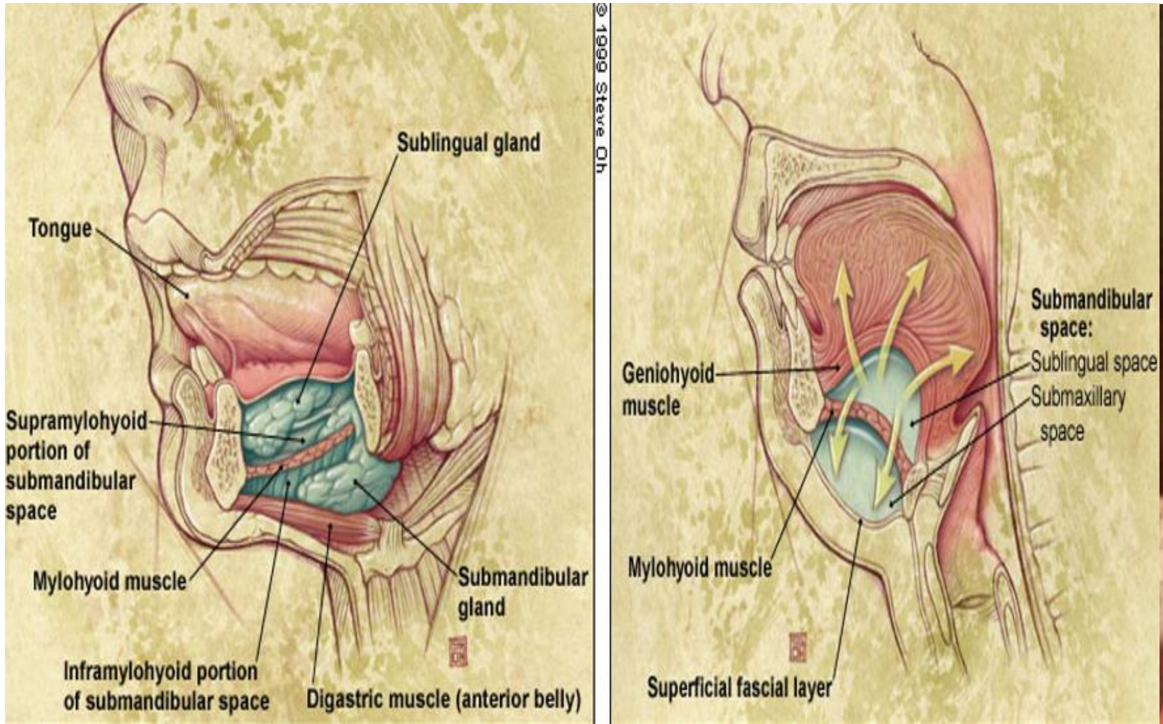
THANK YOU

COMPLICATIONS OF ODONTOGENIC INFECTIONS:

Complications include **Ludwig's angina,** **airway obstruction, mediastinitis, necrotizing fascitis, cavernous sinus thrombosis, sepsis, thoracic empyema, Lemierre's syndrome, cerebral abscess, orbital abscess, and osteomyelitis**. The incidence of these "space infections" has been greatly reduced by **modern antibiotic therapy**

Ludwig's angina





Ludwig’s Angina

Ludwig’s Angina is **infection in multi-space, considered** severe form of cervico-fascial infection / cellulitis which usually arises from the **lower second or third molars (wisdom teeth).**

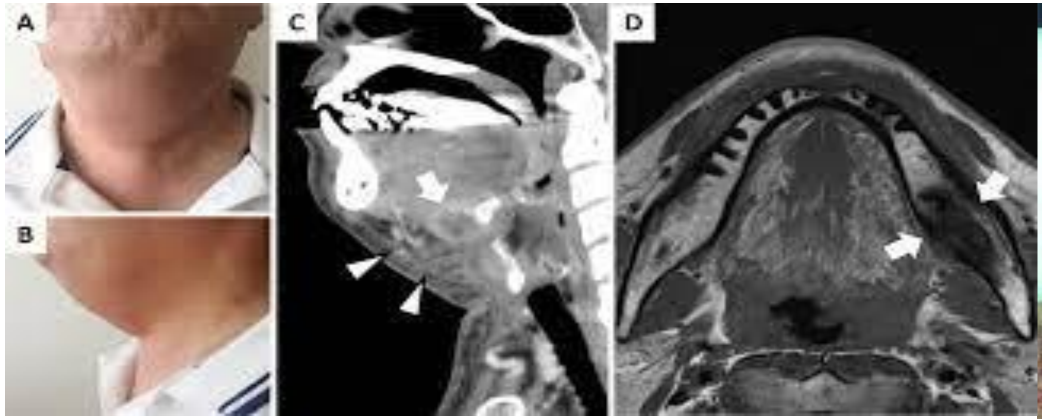
Deep fascial space infections cause gross inflammatory exudates (a fluid with a high content of protein and cellular debris which has escaped from blood vessels and has been deposited in tissues or on tissue surfaces, usually as a result of inflammation. It may be septic or non-septic) and tissue **œdema (swelling),** associated with **fever** and **toxæmia (blood poisoning).** Before the advent of antibiotics, the mortality was high and the disease is still life-threatening if treatment is delayed.

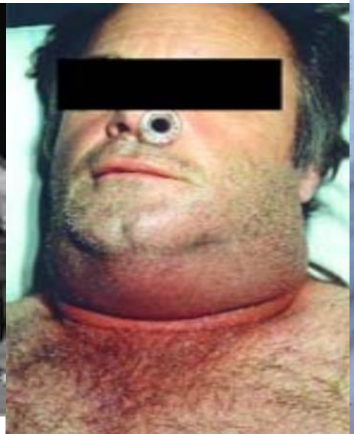
The main fascial spaces involved in Ludwig’s Angina are the sublingual, submandibular and para-pharyngeal.

Normally, the spaces both side of the midline (ie bilateral) are effected.

The characteristic features are:

- diffuse swelling, pain, fever and malaise.
- The swelling is tense and tender, with a characteristic ‘board-like’ firmness.
- The overlying skin is taut and shiny.
- Pain and œdema (swelling) that limit opening the mouth and often cause dysphagia (difficulties in swallowing)
- Systemic upset is severe, with worsening fever, toxæmia (blood poisoning) and leucocytosis.
- The regional lymph nodes are swollen and tender.
- In Ludwig’s Angina particularly, airway obstruction can quickly result in asphyxia.






A
B


A
B

The Cervicofacial necrotizing Fasciitis can always be a cause of death if not intervened promptly; this extensive disease entity is commonly caused by an Immunocompromized state of host, malnutrition, diabetes, and ignored dental infections, surgical and non-surgical trauma.

The mainstay of the treatment includes **prompt surgical debridement**, **intravenous antibiotics**, **fluids and electrolytes management** and **analgesia** along with meticulous attempt to reverse the immunocompromized state of the patient, thus improving their general condition.

Pathology

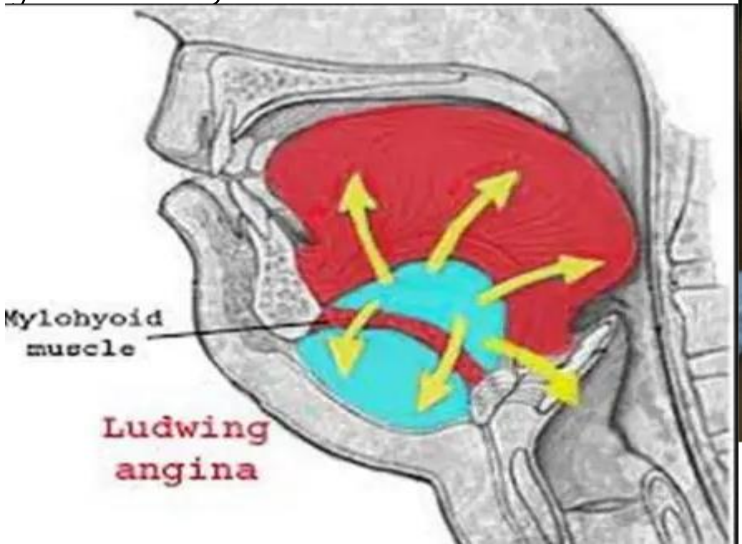
Anærobic bacteria are primarily **responsible** and **infection mainly spreads from mandibular third molars (lower jaw wisdom teeth) whose apices (root tips) are closely related to several fascial spaces**.

Fasciæ, covering muscles and other structures are normally adherent but can be spread apart by inflammatory exudate.

Spaces created in this way are almost avascular (do not have a blood supply) and inflammatory exudate carries bacteria widely through them. It involves the sub-lingual and sub-mandibular spaces bilaterally (on both sides) almost simultaneously; it readily spreads into the lateral pharyngeal and pterygoid spaces and can extend into the mediastinum. The main features are rapidly spreading sub-lingual and sub-maxillary cellulitis with painful, brawny swelling of the upper part of the neck and the floor of the mouth on both sides. With involvement of the para-pharyngeal space, the swelling tracks down the neck and œdema can quickly spread to the glottis.

Swallowing and opening the mouth become difficult and the tongue may be pushed up against the soft palate. The latter or œdema of the glottis causes worsening respiratory obstruction.

The patient soon becomes desperately ill, with fever, respiratory distress, headache and malaise.

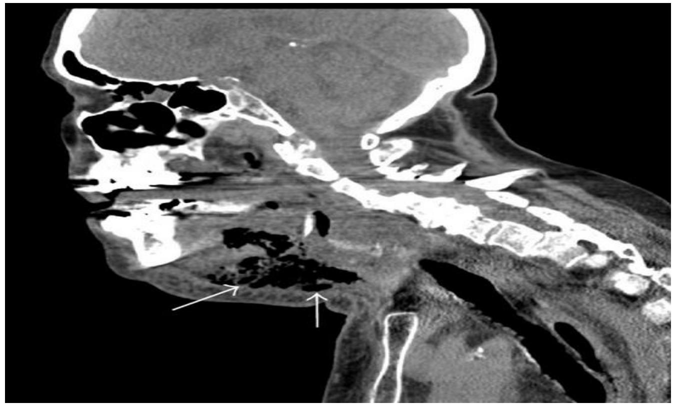


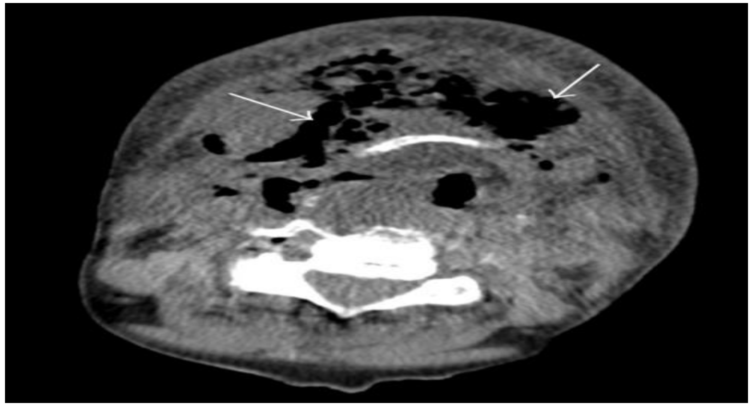
Management

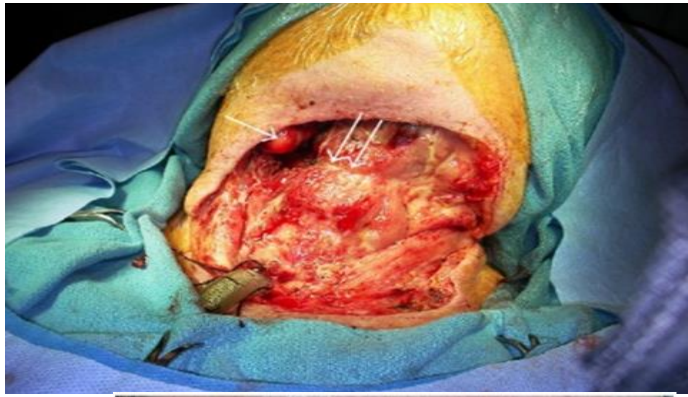
The main requirements are:

- immediate admission to hospital
- procurement of a sample for culture and sensitivity testing
- aggressive antibiotic treatment
- securement of the airway by tracheostomy if necessary, and
- drainage of the swelling to reduce pressure.

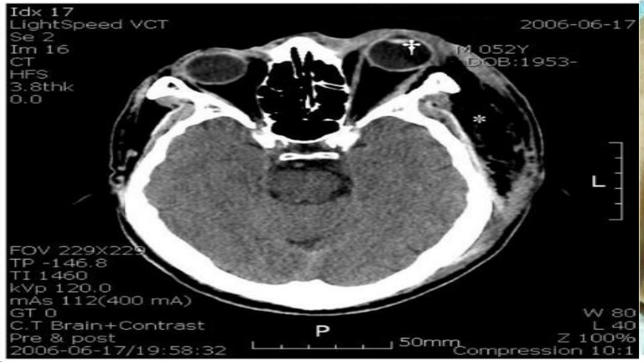
Ludwig's Angina Complicated by Cervical Necrotizing Fasciitis



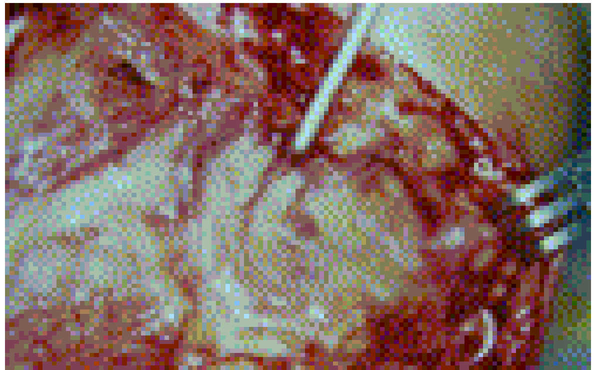


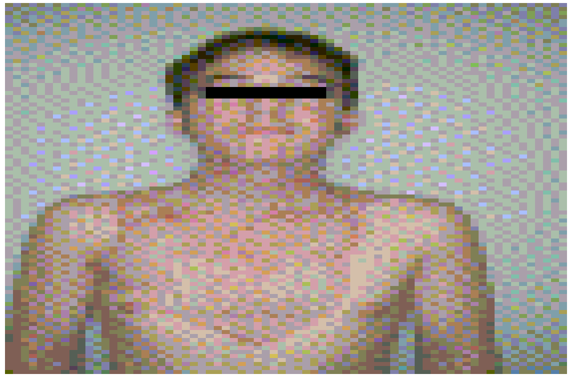


Necrotizing Fasciitis of Odontogenic Origin



Pediatric necrotizing fasciitis complicating third molar extraction



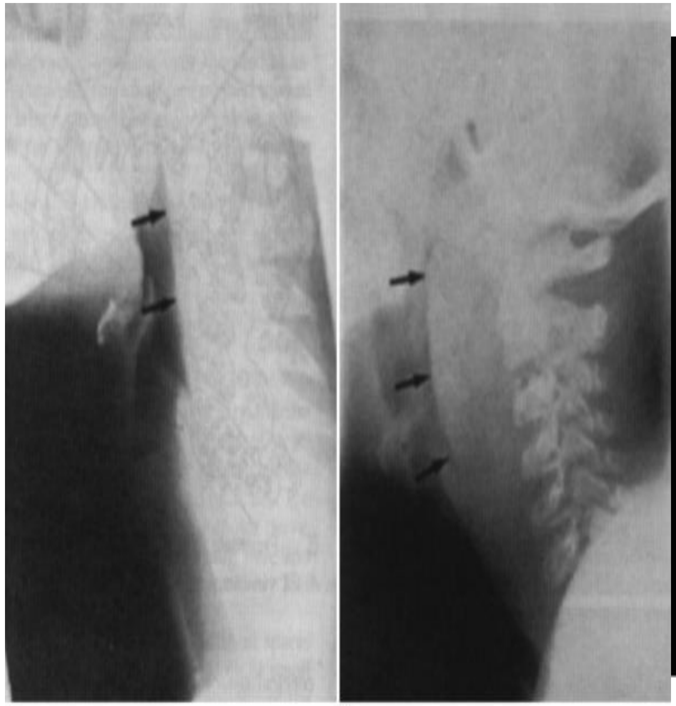


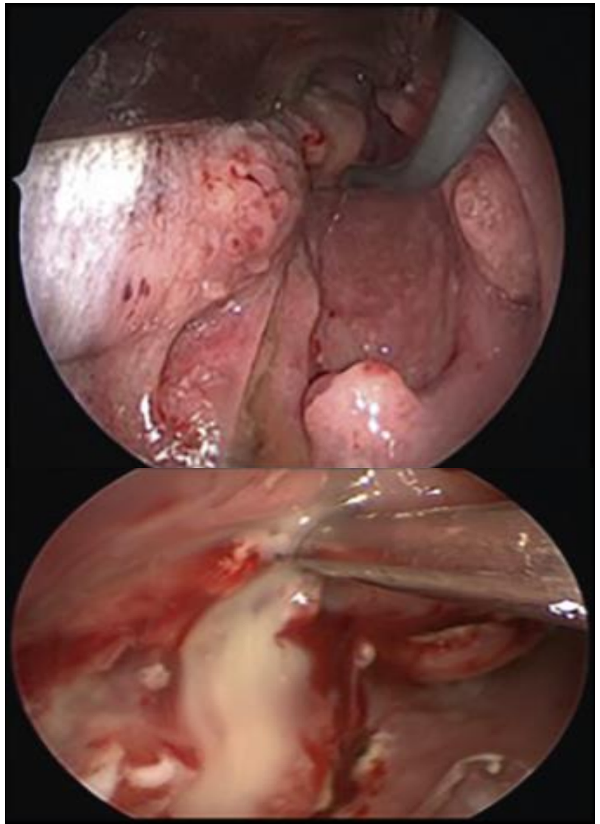
Lateral Pharyngeal Space Abscess

A parapharyngeal abscess is a deep neck space abscess of the parapharyngeal space (or pharyngomaxillary space), which is lateral to the superior pharyngeal constrictor muscle and medial to the masseter muscle. This space is divided by the styloid process into anterior and posterior compartments

Symptoms include fever, sore throat, painful swallowing, and swelling in the neck.

An anterior space abscess can cause lockjaw (spasm of jaw muscle), and hard mass formation along the angle of the mandible, with medial bulging of the tonsil and lateral pharyngeal wall. A posterior space abscess causes swelling in the posterior pharyngeal wall, and lockjaw is minimal. **Other structures within the carotid sheath may be involved**, causing **rigors**, **high fever**, **bacteremia**, **neurologic deficit**, or a **massive haemorrhage** caused by carotid **artery rupture**





EXTRA ORAL, INTRA ORAL APPROACH FOR ABCESS DRAINAGE

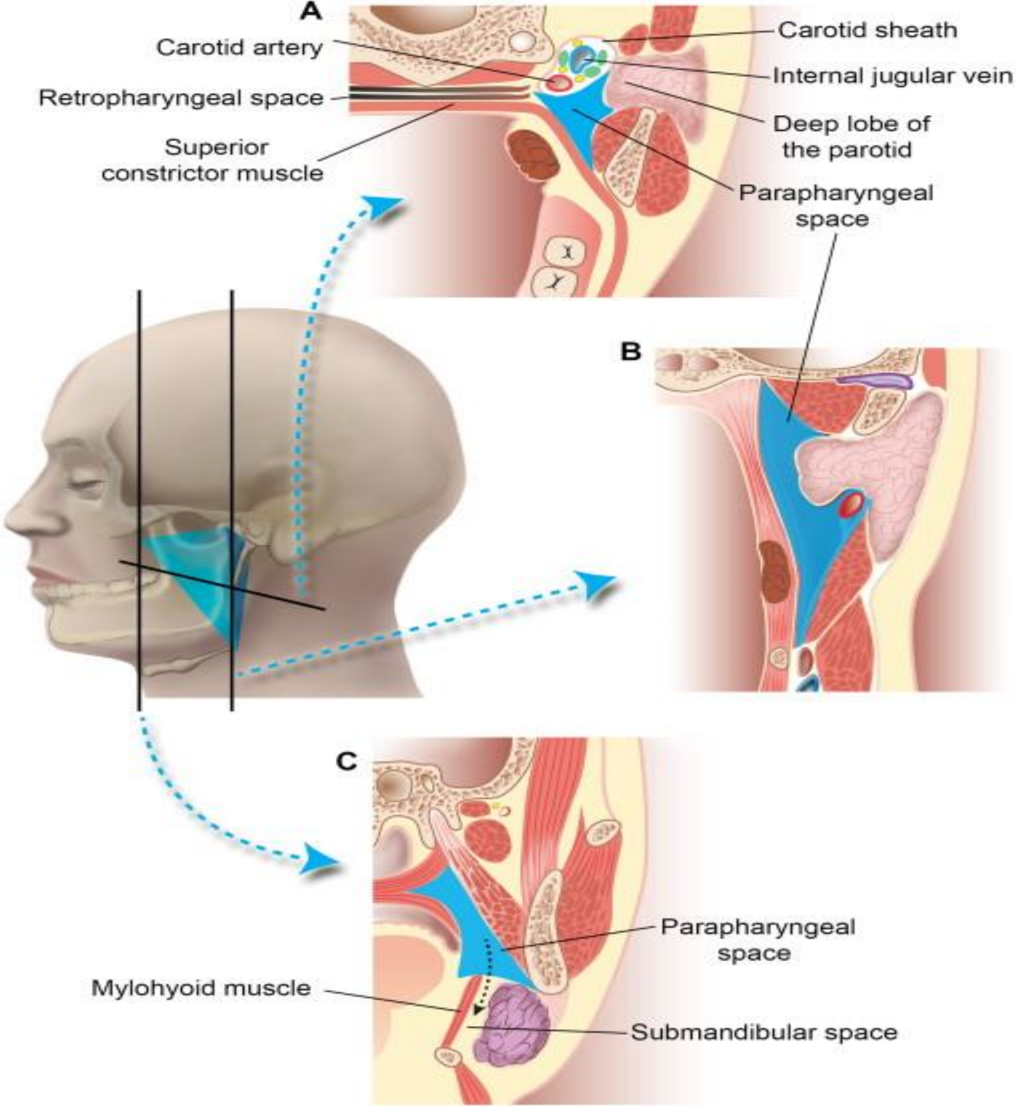
lateral Pharyngeal Space :

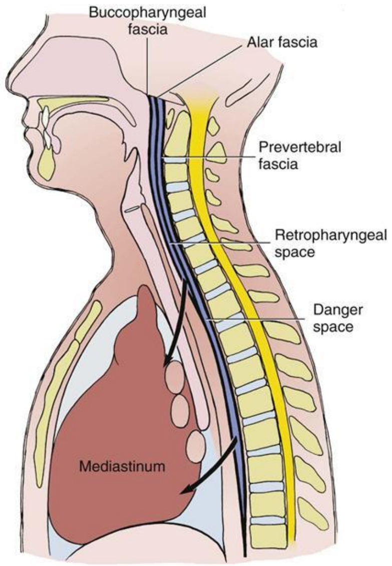
Patients who have infection of the lateral pharyngeal space have **several serious potential problems.**

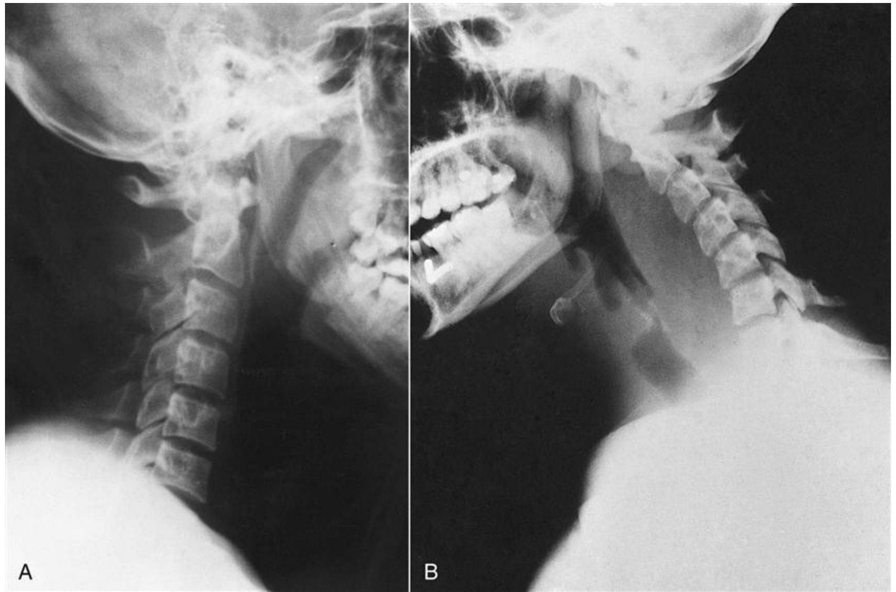
When the lateral pharyngeal space is involved, the odontogenic infection is severe and may be progressing at a rapid rate.

Another possible problem is the **direct effect** of the infection on the contents of the space, especially those of the posterior compartment. **These problems include thrombosis of the internal jugular vein**, **erosion of the carotid artery** or its branches, and interference with cranial nerves IX, X, and XII.

A third serious complication arises if the infection progresses from the lateral pharyngeal space to the retropharyngeal space or beyond



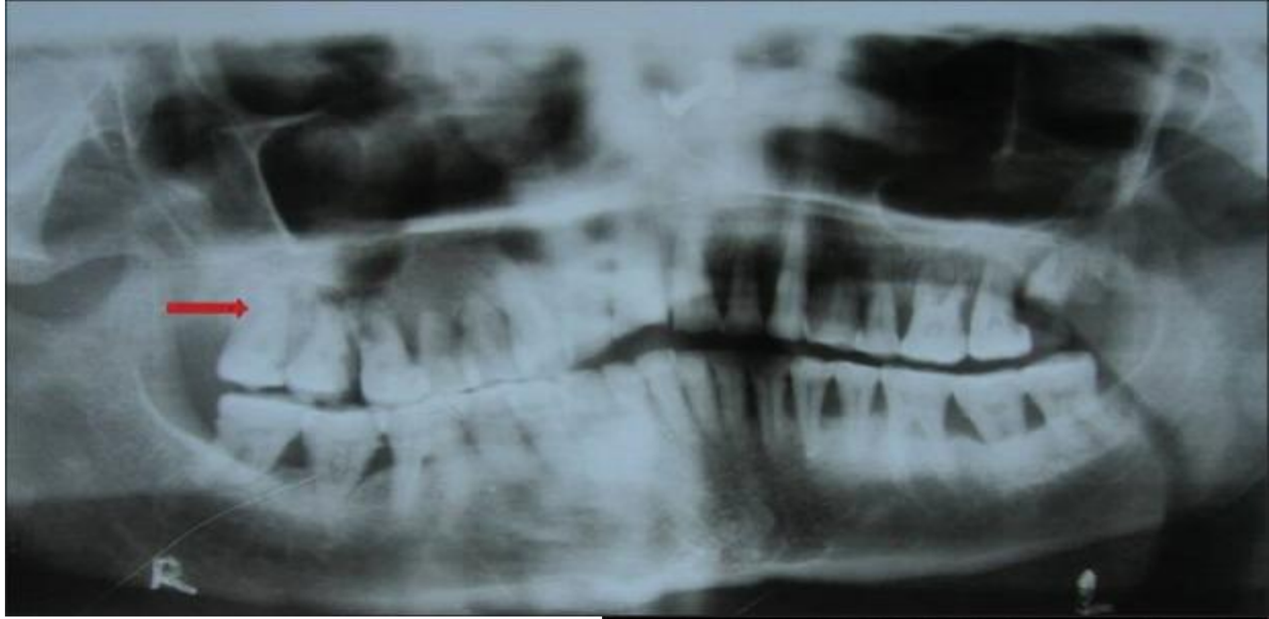


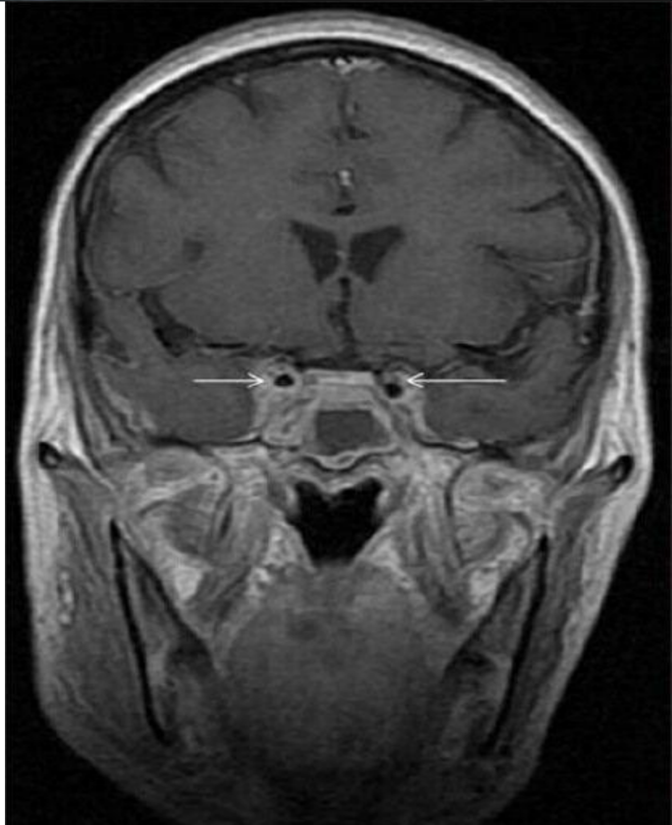


The normal retropharyngeal soft tissue shadow is narrow (6 mm or less) at C2 and 20 mm or less at C6 on plain radiographic films. **B,** When the retropharyngeal space is infected, the soft tissue becomes substantially thicker, and the width of the oropharyngeal air shadow decreases

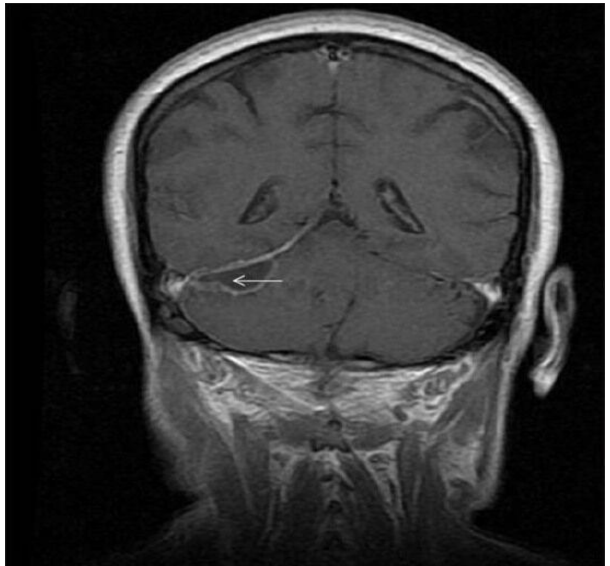
Septic cavernous sinus thrombosis (CST)

Septic CST can be defined as thrombophlebitis of the cavernous sinus of infectious origin. Facial infections and paranasal sinusitis are among the most important causes of septic CST. However, less commonly it may be caused by pharyngeal, otogenic, and odontogenic infections Septic cavernous sinus thrombosis (CST) related to dental infection is a rare clinical event. The septic CST is a disease of high morbidity and mortality. The prompt diagnosis and timely treatment of septic CST is cornerstone of successful outcome. The dental infection should be given due attention, as to prevent CST. In this case report of immunocompetent female, we highlighted the role of dental abscess in producing bilateral CST and facial palsy. **The close collaboration between dentist and neurologist and early institution of antibiotics led to complete recovery at follow-up after 3 months**. The dental infection should never be neglected as it is the interface of serious intracranial complication like CST.





Coronal contrast-enhanced T1 WI showing multiple defects in the enhancing bilateral cavernous sinuses (arrows)



MRI-brain showing right cerebellar abscess (arrow)

Pathology of SCST

The most important dural sinuses are cavernous sinuses, sagittal sinuses, straight sinus, transverse sinuses, and sigmoid sinus, which are all interconnected. The right and left cavernous sinuses are situated on the lateral aspect of the **sella turcica**. They extend from the superior orbital fissure to the petrous apex of the temporal bone. They are interconnected by anterior and posterior intercavernous sinuses that encircle the pituitary gland.

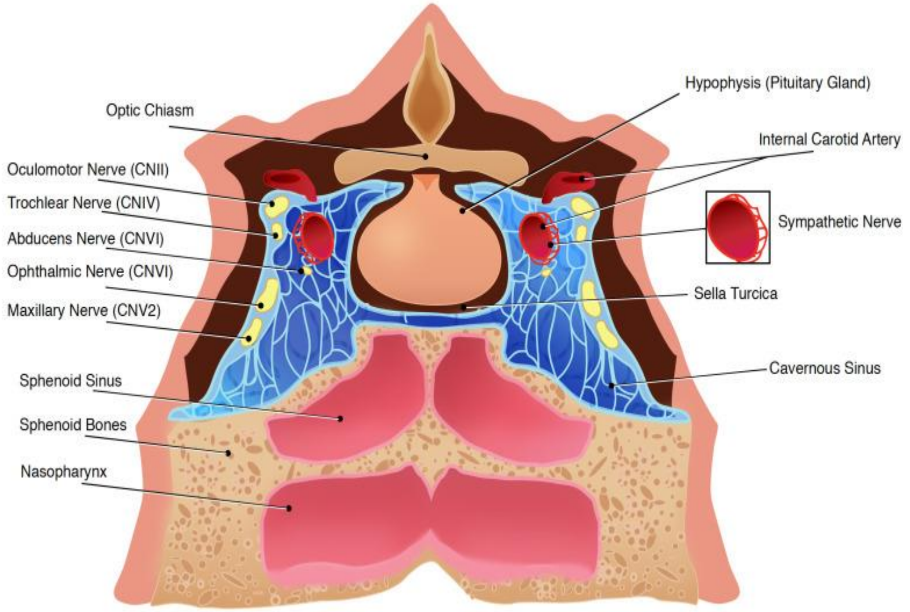
Cavernous sinuses receive blood from the **ophthalmic veins, the superficial middle and inferior cerebral veins**, and the **sphenoparietal** and **sphenoid sinuses**.

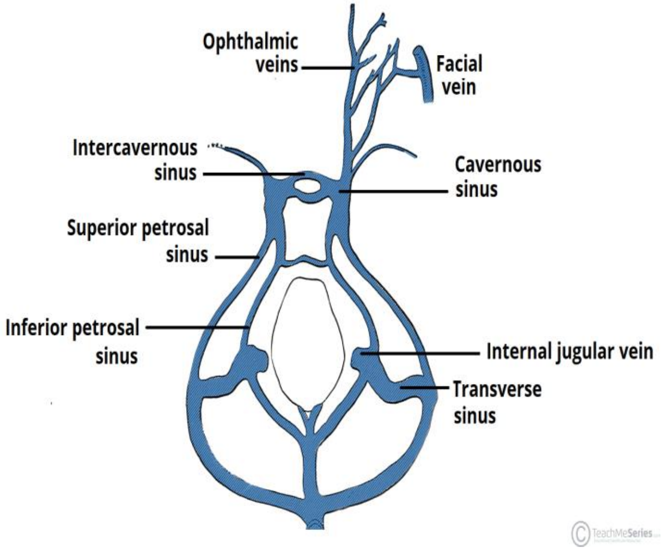
The cavernous sinuses **in turn drain into** the **pterygoid venous plexus** via **emissary veins**, and **into the internal jugular vein** and the **sigmoid sinus** via **the inferior and the superior petrosal sinuses respectively**.

Absence of valves in the cavernous sinuses and their connections favor bidirectional spread of infection. This can give rise to extensive thrombi throughout the network of sinuses.

Most commonly, CST results from spread of infection from the **sinuses**, especially the **sphenoid**, **ethmoid**, and **frontal sinuses**, or from the middle one third of the face. **Less commonly infection from teeth**, **nose**, **tonsils**, **soft palat**e, and **ears may constitute primary source of the infection**.

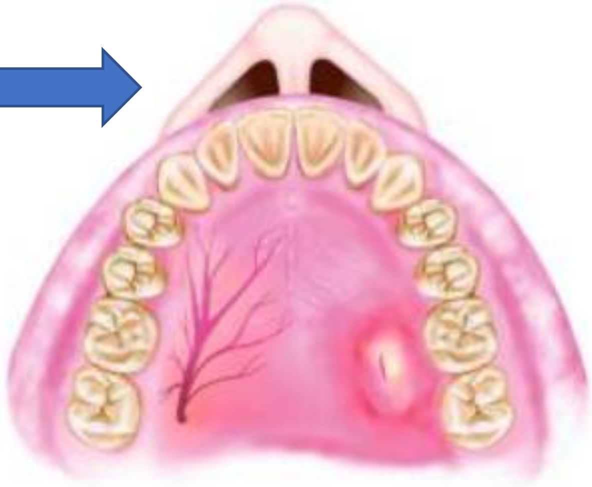
The dental infection most commonly **spreads via** the **pterygoid venous plexus**, where an infected thrombus may **extend or disseminate septic emboli**





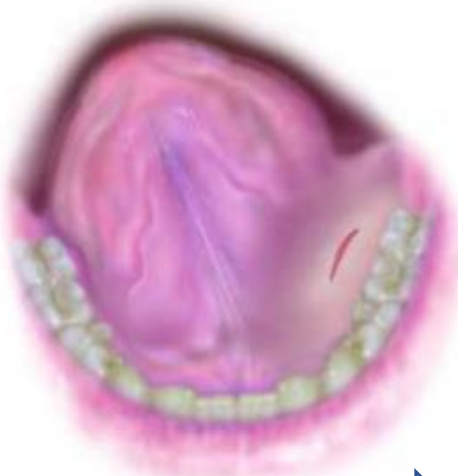
Antibiotics are the main treatment for cavernous sinus thrombosis. Treatment will be started as soon as possible, even before tests have confirmed if a bacterial infection is responsible. If tests later show that a bacterial infection did not cause the condition, antibiotic treatment may be stopped

Incision for drainage of a palatal abscess, parallel to the greater palatine vessels



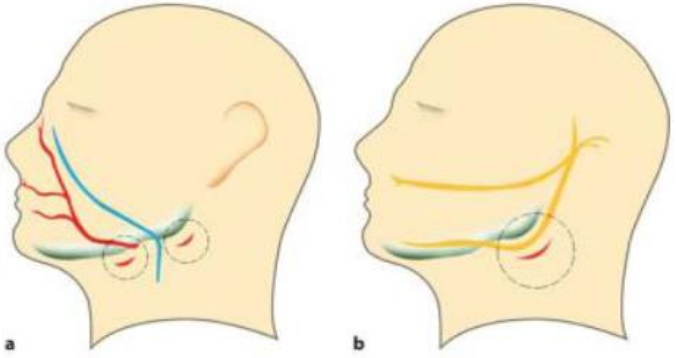
D-Antisepsis of the area with an antiseptic solution before the incision.

E-Anesthesia of the area where **incision and drainage** of the abscess are to be performed, **with the block technique together with peripheral infiltration anesthesia at some distance from the inflamed area**, in order to **avoid the risk of existing microbes spreading into deep tissues**.

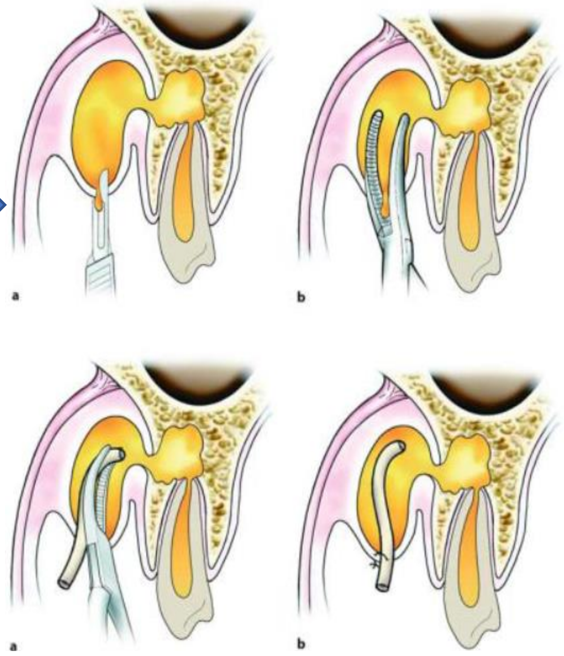


F-Planning of the incision so that:

–Injury of ducts (Wharton, Stensen) and large vessels and nerves is avoided



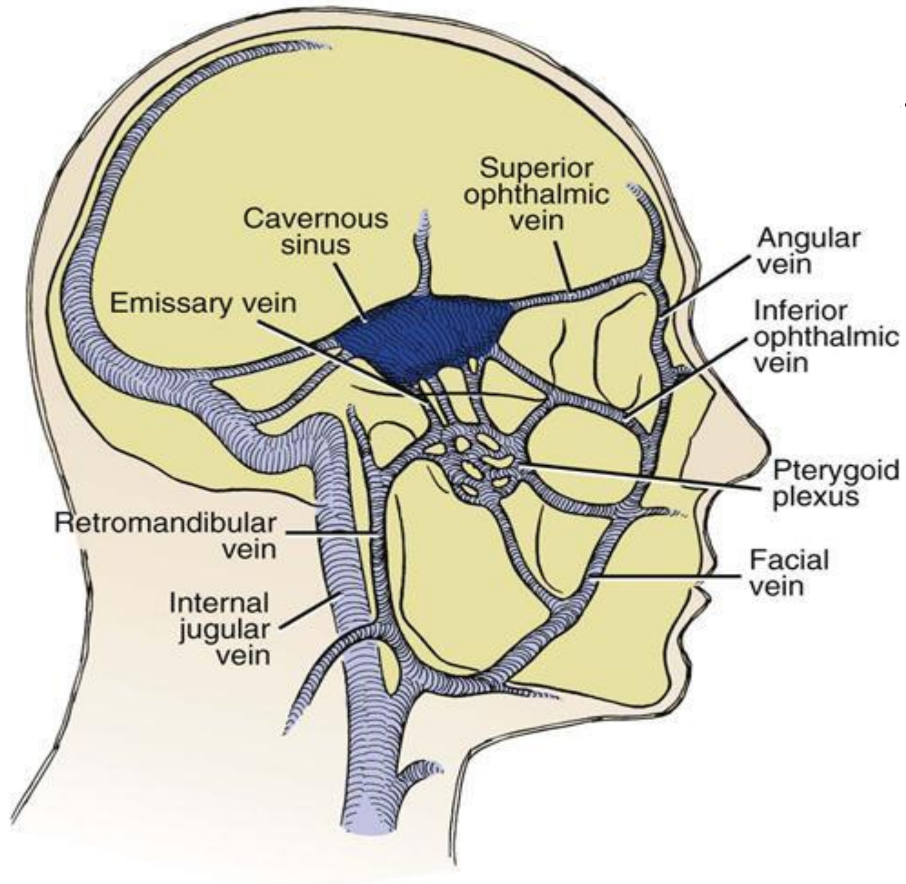
–Sufficient drainage is allowed. The incision is performed superficially, at the lowest point of the accumulation, to avoid pain and facilitate evacuation of pus under gravity–The incision is not performed in areas that are noticeable, for esthetic reasons; if possible, it is performed intraorally.



G-Incision and drainage of the abscess should be performed at the appropriate time. This is when the pus has accumulated in the soft tissues and fluctuates during palpation, that is when pressed between the thumb and middle finger, there is a wave-like

When maxillary odontogenic infections **erode into the infraorbital vein in the infraorbital space** or **the inferior ophthalmic vein via the sinuses**, they can follow the **common ophthalmic vein through the superior orbital fissure** and **extend directly into the cavernous sinus**. This is the anterior route to the cavernous sinus. Intravascular inflammation caused by the invading bacteria stimulates the clotting pathways, resulting in a septic cavernous sinus thrombosis. Cavernous sinus thrombosis is an unusual occurrence that is rarely the result of an infected tooth. Like orbital cellulitis, cavernous sinus thrombosis is a serious, life-threatening infection that requires aggressive medical and surgical care.

Cavernous sinus thrombosis has a high mortality even today. illustrates the rapid extension of a case of orbital

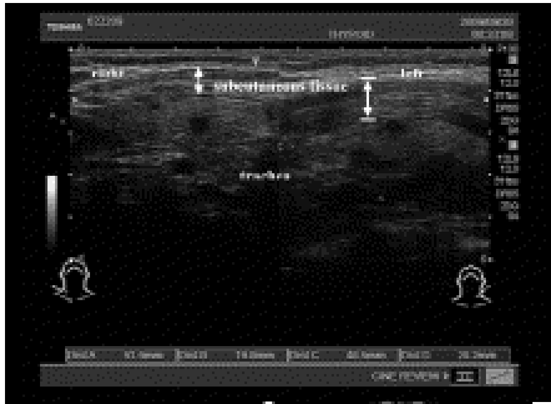


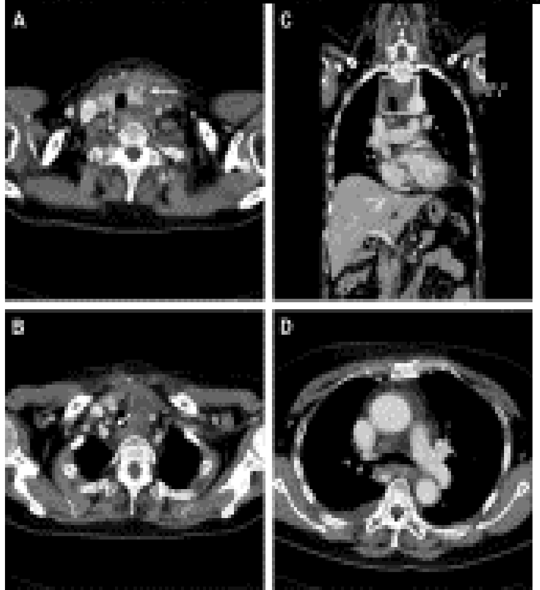
fortunately, the most vulnerable structure in the cavernous sinus, the

.

Acute descending mediastinitis secondary to an odontogenic infection

Acute mediastinitis is a **life-threatening infection** involving the mediastinal connective tissue that fills the interpleural space and surrounds the adjacent thoracic organs. The majority of cases result from **oesophageal perforation**, or as a consequence of a post-operative infection after sternotomy. Rearely from dental infection :





Three criteria for the diagnosis of mediastinitis of **odontogenic origin**

1-Clinical evidence of **severe oropharyngeal infec**tion.

2-Characteristic radiological features of mediastinitis (radiographs of the chest will generally show gas in the tissues, air fluid levels, and mediastinal widening).

Establishment of the relationship between **mediastinitis** and the **oropharyngeal process**.

There are three primary routes of the infection spreading from the **neck to the mediastinum**:

A-Via the pretracheal space which descends into the anterior mediastinum. Its fascia fuses with the pericardium and the parietal pleura. This explains the occurrence of empyema and pericardial effusion in our case.

2-Via the viscerovascular space, and

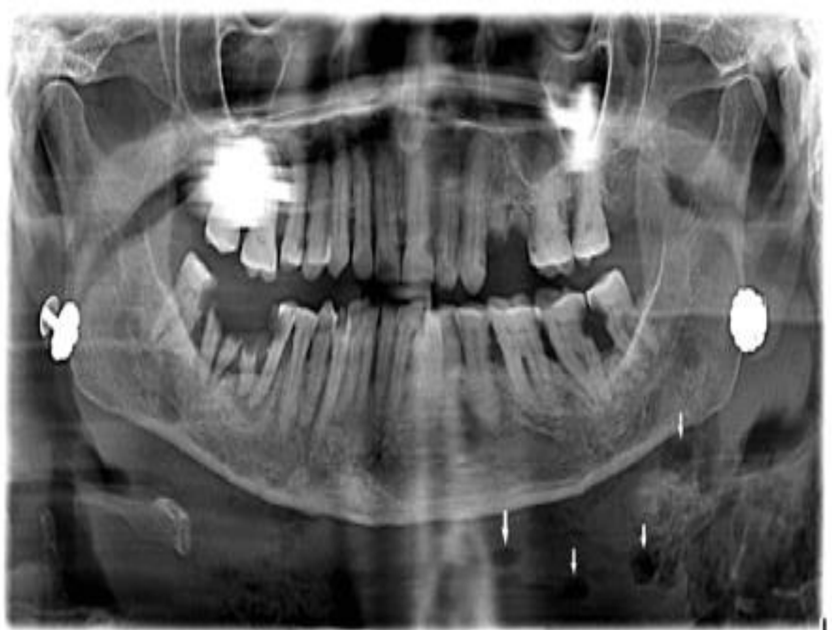
3-Via the retropharyngeal space.

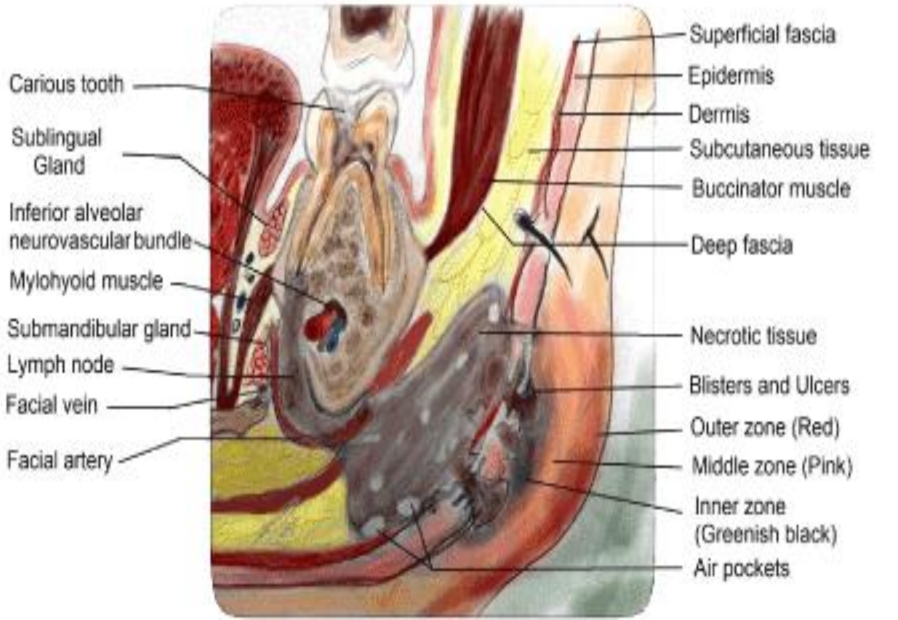
This is the most frequent route for descending mediastinitis to spread into the posterior mediastinum and the diaphragm

Cervicofacial necrotizing fasciitis of odontogenic origin

Cervicofacial necrotizing Fasciitis (NF) is a **rare**, **fatal**, **rapidly progressive infection of soft tissue** that often presents as necrosis of the superficial fascia with undermining of the subcutaneous tissue and necrosis of the skin.

The term necrotizing fasciitis was first coined by Wilson in 1952. The mortality rate of NF has been reported to be as high as 40% . Being a rare entity, it is very important for an Oral and Maxillofacial Surgeon to possess knowledge of the **cardinal sign**s and symptoms of this disease and to initiate the treatment as early as possible.





General Principles in Treatment of Oral and Paraoral Infections

1-Remove the cause.

2-Establish drainage.

3-Institute antibiotic therapy.

4-Supportive care, including proper rest and nutrition.

Goals of management of odontogenic infection:

Airway protection

Surgical drainage#

Medical support of the patient

Identification of etiologic bacteria

Selection of appropriate antibiotic therapy

Principles for choosing appropriate antibiotic

causative organism

sensitivity

specific, narrow-spectrum antibiotic

least toxic antibiotic

drug history(success, allergic and toxic)

Antibiotic Therapy

Removal of the cause, drainage, and supportive care more important than antibiotic therapy.

Infections are cured by the patient's defenses, *not* antibiotics.

Risks of allergy, toxicity, side effects, resistance and superinfection causing serious or potentially fatal consequences must be considered.

Oral infections are typically polymicrobial.

Antibiotic effectiveness dependent upon adequate tissue (not serum) concentration for an appropriate amount of time.

Antibiotics should be prescribed for at least one week – adequate tissue concentration achieved in 24-48 hours, with bacteriocidal activity occurring over the next 3-5 days.

Principles of antibiotic administration

Proper dose ,Proper time interval, Proper route of administration(oral, parenteral), Combination antibiotic therapy

How to judge the pus formation?

Characteristic

Three stages

Inoculation

↓

Cellulitis

↓

Abscess

- Duration--- >5 days
- Palpation---Fluctuant
- Appearance---Reddened
- Needle aspiration
- B-ultrasound
- CT

Surgical drainage and incision

- How to judge the pus formation?
- Purposes of surgical drainage and incision
- Principles of surgical drainage and incision

Fundamental Principles of Treatment of Infection:

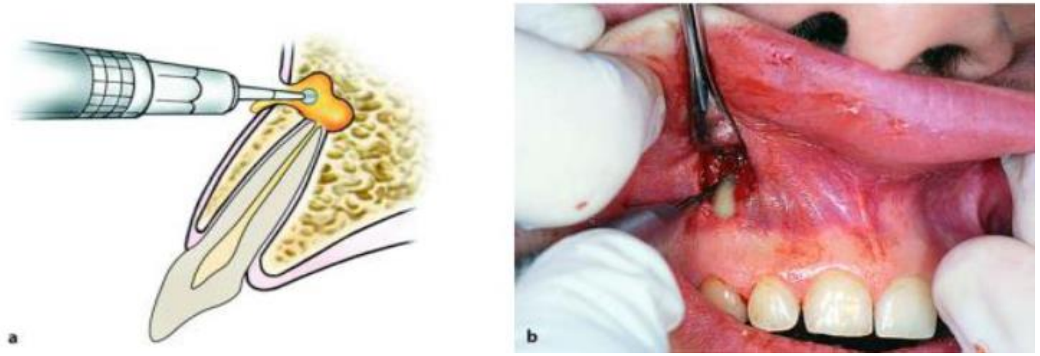
In order to treat an **acute dentoalveolar infection** as well as a **fascial space abscess correctly**, the following are considered **absolutely necessary:**

A-Take a detailed medical history from the patient.

B-Drainage of pus, when its presence in tissues is **established**.
This is achieved

(1) by way of the root canal,
(2) with an intraoral incision,
(3) with an extraoral incision, and
(4) through the alveolus of the extraction. Without evacuation of pus, that is with administration of antibiotics alone, the infection will not resolve.

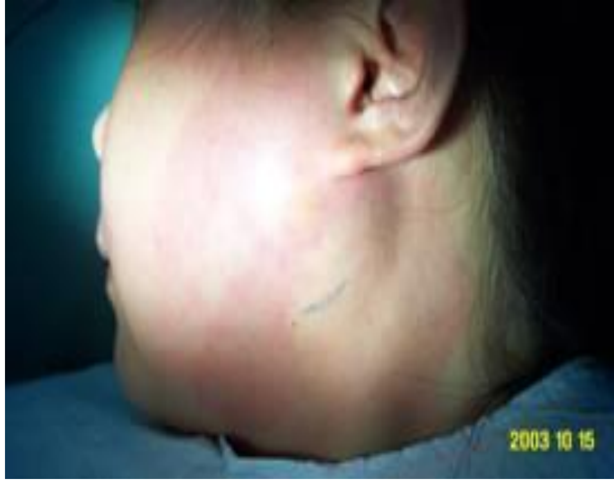
C-Drilling of the responsible tooth during the **initial phase of inflammation**, to drain exudate through the root canal, together with heat therapy. In this way, spread of inflammation is avoided and the patient is relieved of the pain.
Drainage may also be performed with **trephination of the buccal bone**, when the root canal is inaccessible.



Trephination of buccal bone for drainage of an abscess. **a** Diagrammatic illustration. **b** Clinical photograph

Purposes of surgical drainage & incision

- Rid the body of toxic purulent material
- Decompress the tissues
- Allowing better perfusion of blood containing antibiotics and defensive elements
- Increased oxygenation of the infected area
- Place the incision in an esthetically acceptable
- Place the incision in a dependent position to encourage drainage by gravity
- Dissect bluntly through deeper tissues and explore all portions of the abscess
- Place a drain and stabilize it with sutures



Therapeutic considerations:

Although **surgical drainage of loculated pus** and **dental extraction are the mainstay of therapy**, **antibiotic** treatment is required to **halt local spread of infection and prevent hematogenous dissemination**.

Anti-infective agents are generally indicated if **fever** and **regional lymphadenopathy** are present or **when infection has perforated the bony cortex and spread into surrounding soft tissues**.

Severely immunocompromised patients are particularly at risk for rapidly spreading orofacial infections and bacteremia. Empiric antimicrobial therapy should be initiated promptly in such patients.

The choice of specific antimicrobial regimens for odontogenic orofacial infections is empirical based on anticipated **causative pathogens** and **immune status of the host**.

This is because specimens obtained extraorally to avoid contamination by the oral commensal flora are rarely available.

Odontogenic orofacial space infections are usually polymicrobial involving both strict anaerobes and facultative bacteria within unique ecosystems of the dental plaque and gingival crevice.

The most **prevalent anaerobic bacteria include gram-positive cocci**, such as **Peptostreptococcus spp**., and **gram-negative rods**, such as Bacteroides spp., Fusobacterium spp., Prevotella spp. and Porphyromonas spp. The most prevalent aerobes are facultative gram-positive cocci such as Streptococcus mutans, and viridans streptococci. Facultative gram-negative bacilli and S. aureus (including MRSA) are uncommon in immunocompetent hosts but may be more important in immunocompromised patients

In healthy and **systemically well patients** without trismus, infections of low-risk spaces can be initially treated in a primary care dental practice, **while infections spreading to higher risk spaces should be managed more aggressively and may need to be treated in a secondary care centre** ., like

Ludwig's angina

Is rapidly and frequently **fatal progressive gangrenous cellulitis** and **ooedema of the soft tissues of the neck and floor of the mouth**.**The mortality rates have reduced significantly with the introduction of antibiotics**, improved oral and dental hygiene, **and timely surgical intervention.**

The initial immediate management usually includes

a)positioning the patient in an upright position and administering oxygen 15 litres/minute, **while referring to the hospital. Even a mild pericoronitis**

b) if left untreated may progress to the formation of an abscess that could expand in the submandibular and submental spaces

c) or further to the neck and pretracheal spaces

d) requiring extensive drainage and airway protection though a tracheostomy

Antibiotic therapy

Penicillin (bacteriocidal) drug of choice for treatment of odontogenic infections (5% incident of allergy).

Clindamycin (batericiodal) 1st line after penicillin; effective against anaerobes; stop taking at first sign of diarrhea.

Cephalosporin (slightly broader spectrum and bacteriocidal); cautious use in penicillin-allergic patients → cross-sensitivity; if history of anaphylaxis to penicillin, do not use.

Erythromycin (bacteriostatic) good 2nd line drug after penicillin; use enteric-coated to reduce GI upset.

Metronidazole (bacteriocidal) excellent against anaerobes only.

Augmentin (amoxicillin + clavulanic acid) kills penicillinase-producing bacteria that interferes with amoxicillin; expensive.

Single or combined therapeutic depending on the severity of infection, normal person or immunocompromised person. ,

Immunocompromised Host may need more stronger antibiotics like :

Cefotaxime 2 g IV q 6h, OR

Orceftriaxone 1 g IV q 12h, OR

Orcefepime 2 g IV q 12h; OR

Ciprofloxacin 400 mg IV q 12h OR

Imipenem 500 mg IV q 6h; OR

Meropenem 1 g IV q 8h

plus

Metronidazole 500 mg IV q 6h;

LOCAL ANAESTHESIA IN OROFACIAL INFECTION

The in efficient so not is anesthesia Local ,Therefore. **pH acidotic** of because pus of presence of effect the augment to needed is analgesic potent a .dissection tissue deep allow to anesthetic local the agonist-muacting short-ultra strong a is **Remifentanil and sedation** and **analgesia for used been has which also has It**.
Anumber of reasons why local anaesthesia may not work as well as it should do.
The reasons are:
1.poor technique
2.anatomical variation
3.local infection (a “hot tooth”), acidic environment of the abcess .
4.hypersensitivity due to fear
5-some forms of Ehlers-Danlos syndrome(**Ehlers–Danlos syndromes** (EDS) are a group of genetic connective-tissue disorders.[1] Symptoms may include loose joints, joint pain, stretchy velvety skin, and abnormal scar formation.[1] These can be noticed at birth or in early childhood.[2] Complications may include aortic dissection, joint dislocations, scoliosis, chronic pain, or early osteoarthritis.[1][3]
EDS occurs due to variations of more than 19 genes that are present at birth.[1] The specific gene affected determines the type of EDS.[1] Some cases result from a new variation occurring during early development, while others are inherited in an autosomal dominant or recessive manner.[1] Typically, these variations result in defects in the structure or processing of the protein collagen.
6-having red hair!

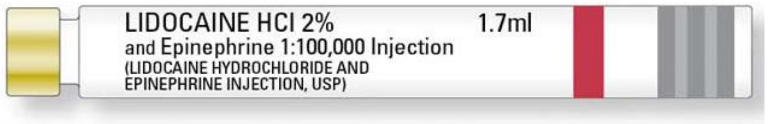
Buffered Local Anesthetic?

The local anesthetic used in dental treatment is the medicine that provides the numbing effect that makes your tooth comfortable. Whether your tooth is causing pain and needs a root canal or is not hurting, but needs a procedure to save it, a local anesthetic is administered by your dentist.

The local anesthetic or “novocaine” as it is commonly called is injected into the soft tissue, and within minutes begins to “numb” the tooth, which blocks all pain impulses. The anesthetic medicine is packaged and sold to dentists in small **sterile glass carpules** and **typically contains a small amount of epinephrine** within the enclosed solution.

The epinephrine works by partially constricting or closing the blood supply near the injection site, so that the local anestheic solution is not “drawn away” by the patient’s blood supply.

Placing the local anesthetic near the **tooth makes the tooth stay numb long enough to complete the dental treatment** and **maintains the potency or profoundness of the numbing effect to keep the patient comfortable during the procedure**



One downside to a local anesthetic that contains the **epinephrine vasoconstrictor is that the ph or “acid-base balance**” tends to be more on the **acid side of the equation** as delivered by the manufacturer. This results in **two minor**, but manageable problems: **First**, as the local anesthetic is initially injected into the patient’s soft tissue, the **lower “acid” ph results in a slight stinging effect felt by the patient**. Due to the fast action of most local anesthetics, the surrounding tissue becomes numb quickly and subsequent injections, even at the **lower ph are not felt by the patient**. **Secondly,** also due to the lower ph, less number of anesthetic chemical molecules are initially released into the area that is being numbed. Because of this effect, multiple injections may be required and a time **delay of the local anesthetic to profoundly numb an inflammed or infected tooth.** The human body’s soft tissues eventually **neutralizes or “buffers” the lower ph of the local anesthetic** back to a neutral balance that gets and keeps a **patient profoundly numb.**

If you don't buy ready made buffered LA ,an alternative process is to buffer or neutralize the local anesthetic carpule immediately prior to its use. **This allows the following benefits:**

(1.) The manufacturer's normal shelf life is maintained.

(2.) There is no "sting" felt by the patient when the local anesthetic solution is injected, sometimes only a slight pressure sensation.

(3.) The onset of the numbing effect is almost immediate–no waiting for 20-30 minutes for it to work.

(4.) The profoundness of the local anesthesia effect is markedly stronger, especially in inflammed or infected teeth.

(5.) The number of carpules needed to adequately get and keep a tooth numb is reduced, therefore resulting in greater patient safety, comfort, minimal procedural interruptions for repeat injections and less post-operative pain.

How is the buffering procedure performed? It is quite simple and has been used in general medical procedures for decades. Here are the steps:

•1. A sterile dental anesthetic carpule (1.8cc) is loaded into a standard dental syringe.

•2. The dentist then extrudes and discards approximately 10% of the solution (0.18cc) out of the anesthetic cartridge.

•3. Using a separate sterile plastic syringe and needle, approximately 1cc of 8.4% sterile sodium bicarbonate (an alkaline base solution) is drawn into the 3cc syringe from the bottle of sodium bicarbonate.

•4. Remove the needle from the 3cc syringe containing the sterile 8.4% sodium bicarbonate and insert the dental cartridge's anesthetic needle into the open ended syringe of sodium bicarbonate and proceed to aspirate 0.18cc of sodium bicarbonate back into the dental syringe anesthetic carpule.

•5. This process essentially replaces approximately 10% of the local anesthetic solution of the anesthetic carpule with the 8.4% sodium bicarbonate. When the two solutions are mixed together, the local anesthetic solution (low ph- "acid") + sodium bicarbonate (high ph- "base") they immediately balance each other out and the final solution becomes a neutral ph=7.0, which is equal to the body's ph of 7.0. This final, neutralized solution is used to numb the patient's tooth. Ph paper is used to verify the correct ph of the final solution: the color of the ph paper will change from yellow (new carpule: ph=3.8) to green (buffered carpule: ph=7.0).

The above 5 steps are repeated for each anesthetic carpule used to numb the patient as per standard local anesthetic practice. As mentioned earlier, once a local anesthetic carpule is buffered, it must be used on a patient typically **within a half hour of the buffering technique**, or **the "shelf life" of the anesthetic carpule diminishes rapidly and must be discarded**. Once practiced, the Doctor can be quite proficient in this technique and it can be easily incorporated into daily practice.

Along with topical anesthetics to numb the **oral tissues prior to the first injection**,

The potential benefits of buffering local anesthetic solutions prior to injection, such as decreased injection pain, faster onset, and greater depth of anesthesia, may be particularly advantageous in patients who have difficulty achieving profound anesthesia for clinical dentistry, and for anesthetizing infected areas.

Dentists can effectively buffer local anesthetic preparations using commercially available mixing systems or by utilizing a hand-mixing technique. Rather than using a remove and replace technique, practitioners may consider a direct injecti v v on technique, **adding 0.1 mL of 8.4% sodium bicarbonate** directly into any local anesthetic cartridge regardless of local anesthetic concentration

Dental Local Anesthesia using in abcess drainage

Numbing depends on the pH of the tissue.When there is an abscess (an acute area of infection), the pH drops and the environment becomes acidic. Local anaesthetic is very pH sensitive.

Even in a normal environment, it seeps into nerve fibers slowly, which is why local anesthetics take a few minutes to kick in. In an acid environment, the nerve fibers look to the anesthesia molecules like they are coated with wax and thus diffusion into the fibers is very slow

Local Anesthetic in the Presence of Infection

Infection represents an additional factor in anesthetic performance. Lower tissue pH at the site of infection makes it extremely difficult for the typical LA injection to provide adequate pulpal anesthesia. Infected tissue is more acidic, which makes it more difficult for the RN conversion to occur.

However, LAs suffer a number of drawbacks:

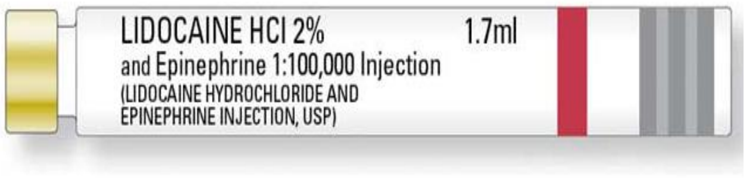
- LAs containing a vasopressor sting on injection
- LAs are associated with a degree of post-injection tissue injury
- LAs have relatively slow onset
- LAs do not work as reliably in the presence of infection and inflammation

All of these drawbacks can be addressed by anesthetic buffering which:

- Eliminates the sting
- Reduces tissue injury
- Reduces latency
- Introduces the independent anesthetic effect of carbon dioxide
- Introduces the catalytic effect of carbon dioxide

Reducing Stinging and Post-Injection Tissue Injury. The burning and stinging of acidic injections represent one of the most common complaints in dentistry.

The mean pH of pus from periapical abcesses was generally acidic and traditional local anesthetics are acidic, which makes them soft-tissue irritants.



Composition of local traditional anaesthetics

Local anaesthetic agent: like ; Lignocaine Hydrochloride- 2%(20mg/ml)

Reducing agent: Sodium meta-bisulphite-(0.5mg)

Preservative: Methylparaben-0.1%(1mg)

Diluting agent: Distilled water

https://www.youtube.com/watch?v=rJMD09iE_N4

Fungicide: Thymol

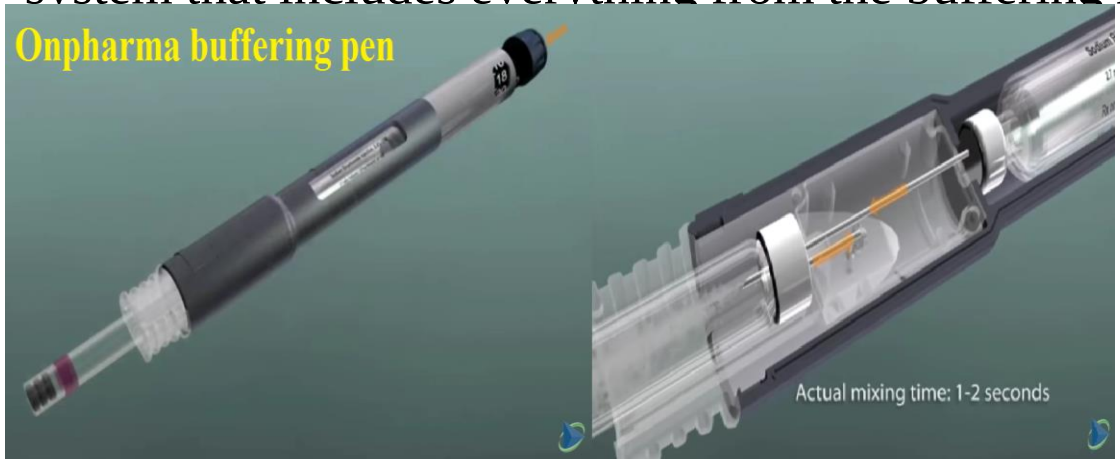
Isotonic solution: Sodium chloride or Ringer's Solution-6mg

Vasoconstrictor: Adrenaline-1

LA IS ACIDIC CAUSING IRRITATION TO THE TISSUE STRUCTURE,WHICH CAUSING DISCOMFORT TO THE PATIENT BECAUSE OF THE TISSUE’S IRRITATION

Buffering of anesthesia has been attempted for years.

Two percent lidocaine and 8.4% sodium bicarbonate are mixed in a very stylish and ergonomic dispenser in a system that includes everything from the buffering materials to the syringe and needles



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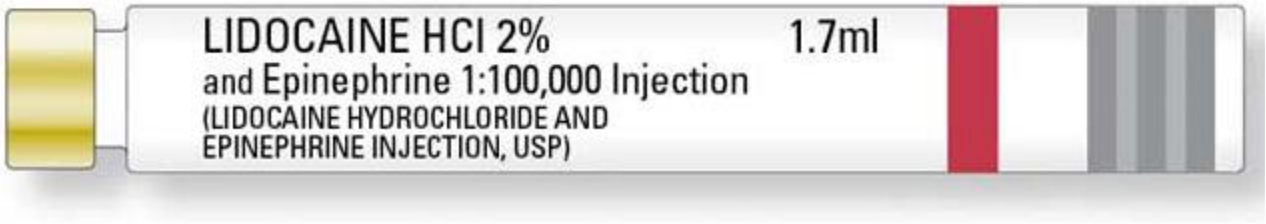
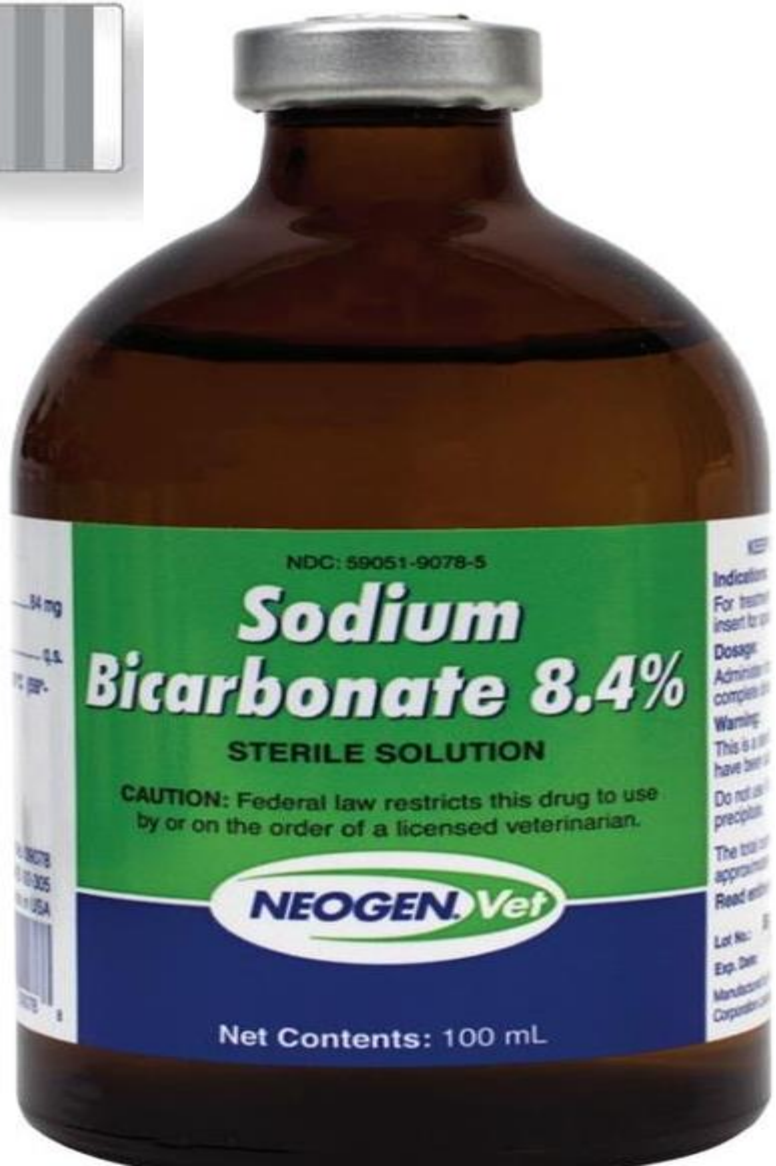




10 principles for an appropriate antibiotic therapy in surgery

Antibiotics can be **lifesaving** when treating patients with **bacterial infections** but are often **used inappropriately**, specifically **when unnecessary** or when **administered for excessive durations or without consideration of pharmacokinetic** principles.

Below you can find 10 principles of antibiotic therapy across the surgical pathway.

1-Enhancing infection prevention and control

"Prevention is better than cure". It is important that all clinicians depend on **evidence-based infection prevention** and **control interventions to reduce demand for antibiotics** by **preventing healthcare associated infections** from **occurring in the first place**, and **making every effort to prevent transmission when they occur**.

2-Controlling source control

Appropriate source control is of outmost importance in the management of surgical infections. Source control encompasses all measures undertaken to **eliminate the source of infection**, **reduce the bacterial inoculum and correct or control anatomic derangements to restore normal physiologic function**.
In critically ill patients with severe sepsis these principles can be applied at different times in the same patient. Source control generally **involves drainage** of abscesses or **infected fluid collections**, **debridement of necrotic** or **infected tissues** and **definitive control of the source of contamination**.
Early control of the septic source can be achieved **using both operative and non-operative techniques**. An operative intervention remains the most viable therapeutic strategy for managing surgical infections in critical ill patients.

As a general principle, every verified source of infection should be controlled as soon as possible. The level of urgency of treatment is determined by the affected organ(s), the relative speed at which clinical symptoms progress and worsen, and the underlying physiological stability of the patient. **Non-operative interventional** procedures include percutaneous drainages of abscesses.

3-Prescribing antibiotics when they are truly needed

The overuse of antibiotics is widely accepted as a major driver of some emerging infections (such as *Clostridium difficile*) and for the continued development of antimicrobial resistance (AMR). Antibiotics can be life-saving when treating bacterial infections but are often used inappropriately, specifically when unnecessary. Antibiotics should be used after a treatable infection has been recognized or when there is a high degree of suspicion for infection.

4-Prescribing appropriate antibiotics

Initial antimicrobial therapy in patients with surgical infections is **typically empirical** in nature because especially critically ill patients need immediate treatment, and microbiological data (culture and susceptibility results) usually requires ≥24 h for the identification of pathogens and antibiotic susceptibility patterns.

Empirical antimicrobial therapy should be based on **local epidemiology**, **individual patient risk factors** for difficult to treat pathogens, **clinical severity of infection**, and **infection source**.

Knowledge of local rates of resistance should be an essential component of the clinical decision-making process when deciding on which antimicrobial regimen to use for empiric treatment of infection.

Every clinician starting empiric therapy should know the local epidemiology. **Surveillance initiatives are important, both in a local and in a global context**.

Previous antibiotic therapy is one of the most important risk factors for resistant pathogens.
Inappropriate therapy in critically ill patients may have a strong negative impact on outcome. An ineffective or inadequate antibiotic regimen is one of the variables more strongly associated with unfavorable outcomes in critically ill patients. **Broad empiric antimicrobial therapy should be started as soon as possible in patients with organ dysfunction (sepsis) and septic shock**.
Finally, selection of **empirical therapy** should take into account the infection source because etiological distribution varies according to the source site.

5-Prescribing antibiotics with appropriate dosage

Antibiotic dosing regimen should be established depending on **host factors** and **properties of antibiotic agents**.
Antibiotic pharmacodynamics integrates the **complex relationship** between **organism susceptibility** and **patient pharmacokinetics.**
Pharmacokinetics describes **the fundamental processes of absorption**, **distribution**, metabolism, and **elimination** and the resulting **concentration-versus-time profile of an agent administered in vivo**.
The achievement of appropriate target site concentrations of antibiotics is essential to eradicate the relevant pathogen. **Suboptimal target site concentrations may have important clinical implications, and may explain therapeutic failures, in particular, for bacteria for which in vitro MICs are high**.

6-Reassessing treatment when culture results are available

The patient should be reassessed when the results of microbiological testing are available.
The results of microbiological testing may have great importance for the choice of therapeutic strategy of every patient, in particular in the adaptation of targeted antimicrobial treatment.

They provide an opportunity to expand antimicrobial regimen if the initial choice was too narrow but also allow de-escalation of antimicrobial therapy if the **empirical regimen was too broad.**

7-Using the shortest duration of antibiotics based on evidence

Duration of therapy **should be shortened as much as possible** unless there are special circumstances that require prolonging antimicrobial therapy **such as immunosuppression**, or **ongoing infections**.
Oral antimicrobials, can **substitute IV agents as soon as the patient is tolerating an oral diet so as to minimize the adverse effects which are associated with intravenous access devices**.
Patients who have **signs of sepsis** beyond **5 to 7 days of treatment warrant aggressive diagnostic** investigation to determine if an **ongoing uncontrolled source of infection or antimicrobial treatment failure is present**.
In the management of **critically ill patients with sepsis** and **septic shock clinical signs and symptoms as well as inflammatory response markers such as procalcitonin**, although debatable, may assist in guiding antibiotic treatment.

8-Educating staff

A range of factors such as **diagnostic uncertainty**, **fear of clinical failture**, **time pressure** or **organisational contexts can complicate prescribing decisions**. However, due to cognitive dissonance (recognising that an action is necessary but not implementing it), **changing prescribing behaviour is extremely challenging**. Efforts to improve educational programs are thus required and this should preferably be complemented by **active interventions such as** prospective audits and feedback to clinicians to stimulate further change.
It is also crucial to incorporate fundamental principles of appropriate prescribing prctice in under- and post graduate training at medical faculties to equip young doctors and other healthcare professionals with the required confidence, skills and expertise in the field of antibiotic management.

9-Supporting surveillance of AMR and HAIs and monitoring of antibiotic consumption

Monitoring of antibiotic consumption should be implemented and feedback provided regularly along with **AMR** or **healthcare-associated infections (HAIs) surveillance data**.

10-Supporting an interdisciplinary approach

An antimicrobial stewardship program (ASP) is a focused effort by a **healthcare organization** or **a portion of an organization** (i.e., an intensive care unit) to **optimize antimicrobial use for the purposes of improving patient outcomes**, **reducing adverse consequences (toxicity, selection of pathogenic organisms**

recognized in **guidelines for implementing** and operating ASPs they perform numerous functions that are integral to successful antimicrobial stewardship.

Nurses are antibiotic first responders, central communicators, as well as 24-hour monitors of patient status.

It's important to use antibiotics only when they are needed **to protect your patient from harms caused by unnecessary antibiotic use and combat antibiotic resistance.**

Sinus formation :

An odontogenic cutaneous sinus tract is a pathologic channel that initiates in the oral cavity and **exits at the cutaneous surface of the face or neck**. It might **resemble** an **ulcer**, **cyst**, **furuncle**, or **retracted, sunken skin.**

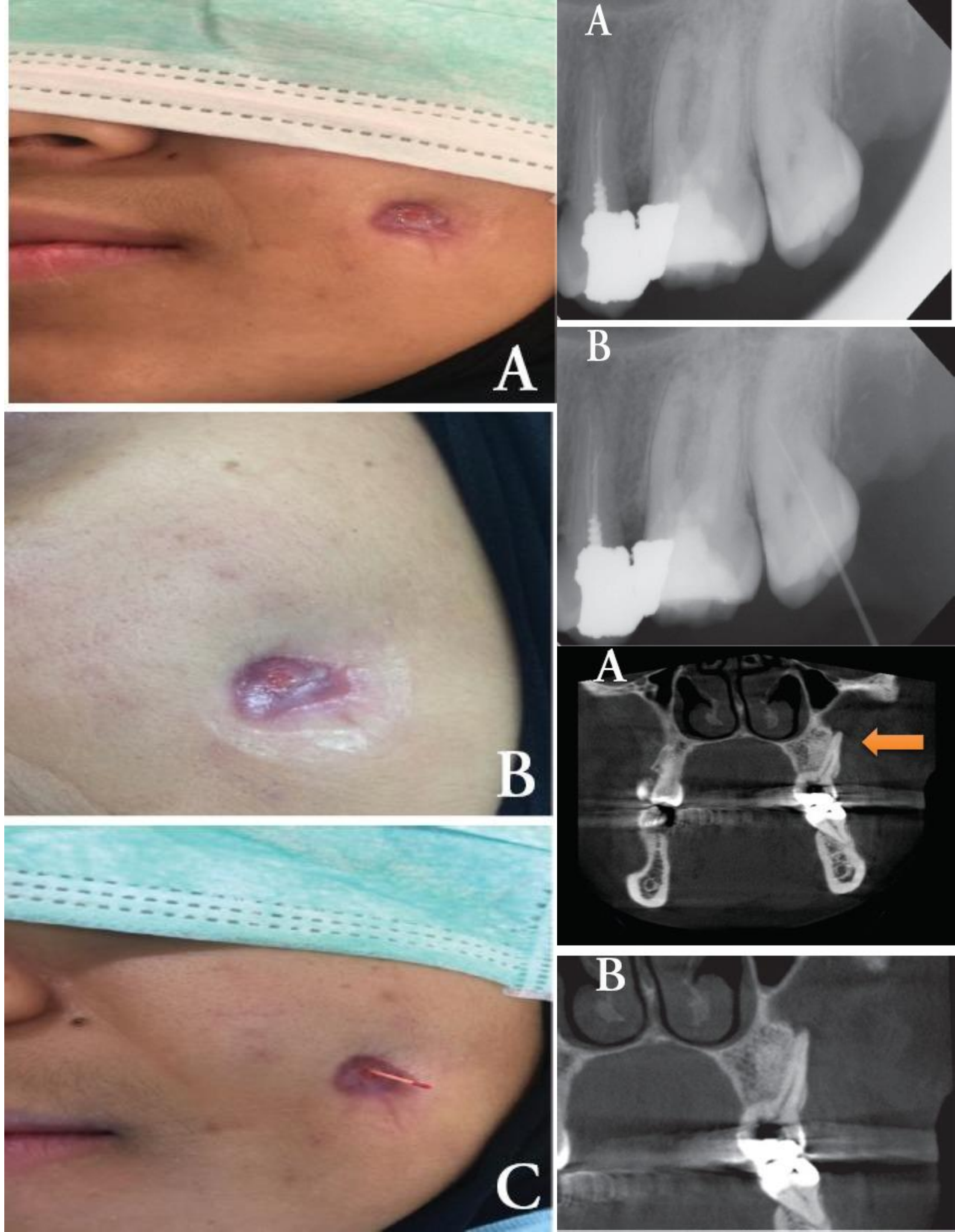
Notably, it is commonly **misdiagnosed**, due to the rarity of its occurrence and the **lack of associated symptoms**.2

This typically results in unsuitable treatment (namely, surgical excision, **antibiotics**, **biopsy**, or **radiotherapy)** and **subsequent relapse of the cutaneous sinus tract**.**Patients require multiple visits to the physician to receive a proper diagnosis**.

Approximately 50% of affected patients may undergo multiple ineffective attempts at **incision, drainage**, and **long-term use of antibiotics**, **due to improper diagnosis and lack of treatment of the infectious dental etiology**.

Approximately **80%** of cutaneous dental sinus tracts originate from **mandibular teeth** and nearly 50% of these lesions are associated with anterior mandibular teeth.

However, **eradication of the cause of the infection**, either through **endodontic treatment (if the tooth can be restored),** or **extraction (if the tooth cannot be restored),**



provides simple and effective resolution of the sinus **tract.**

Odontogenic cutaneous sinus tracts arise due to **pulp infection**, **root fracture**, **chemical irritation**, chronic apical periodontitis, or **dental trauma**.

Affected teeth become necrotic and **exhibit apical periodontitis due to spread of infection** into the **periradicular area**.

The infected by-products of infection will follow the path of **least resistance**, **desiccate** and **breakthrough the skin** to **form draining sinus tracts**.

Access opening was carried out for tooth #26, initial instrumentation and irrigation included an irrigating regimen of sodium hypochlorite (NaOCl) (5.25%), followed by saline, and then chlorohexidine (2%).

The canals were dried and $Ca(OH)_2$ (Multi-Cal, Pulpdent Corporation, USA) was applied. **The patient was examined one week later and showed discontinuation of the external discharge** .There were obvious signs of initial healing of the fistulae. The prior erythematous color of the external scar had reduced. During the second visit, widened the canals with full irrigation . In 6 weak root canal filling sealed the canals , patient improved significantly .

The treatment of odontogenic cutaneous sinus tracts requires the treatment of the ***cause*** of infection, either by **extraction** in the case of a non-restorable tooth, or **root canal** treatment if the tooth is restorable.

Recurrence will most likely occur if excision of the lesion is carried out surgically, without suitable treatment of the affected teeth,


THANK YOU