FACE AND JAW EMERGENCIES

SUPERFICIAL FACIAL INFECTIONS:

- Factors contributing to depth of involvement include the inciting source, length of time before treatment is initiated, host factors (DM, immune status)

CELLULITIS OF THE FACE:

- PATHOPHYSIOLOGY:
  - A superficial soft tissue infection that lacks anatomic constraints
  - RF ➔ fragile skin, inadequate host defenses, immunosuppression, DM
    - Facial piercings
  - Main organisms Staphylococcus aureus and Streptococcus pyogenes
- CLINICAL FEATURES:
  - Characterised by erythema, oedema, warmth, pain and diminished function
  - Ask about chronic illness, trauma, allergen exposure, radiation exposure, surgical history
  - Check for prostheses, nasal drainage, changes in vision or phonation
  - These patients are generally NOT ILL
  - Diagnosis is clinical
  - Bedside US useful in identifying abscess
  - CT may identify deep-seated abscess
- DIFFERENTIAL DIAGNOSIS:

Table 238-1 Differential Diagnosis of Superficial Facial Infection

	Historical Features		Physical Findings
	Onset/Timing	Risk/Inciting Factors	
Infectious			
Cellulitis	Gradual	Skin breaks, foreign bodies, prostheses, immunosuppression	Diffuse erythema without clear borders, pain
Impetigo	Acute	Infants, children	Discrete vesicles or bullae; patches of crusty skin
Erysipelas	Gradual	Elderly, infants and children, immune deficiency, diabetes, alcoholism, skin ulceration, impaired lymphatic drainage	Well defined, raised area of erythema, pain
Viral exanthem	Often acute	Preceding or concurrent viral illness, fever	Variable
Parotitis	Gradual	Dehydration, diabetes, immunosuppression	Swollen angle of mandible, potentially visible sialolith
Necrotizing fasciitis	Rapid	Trauma, may be minor or nonapparent	Crepitus, skin necrosis, may be subtle
Cutaneousanthrax	Gradual	Animal contact	Black eschar with surrounding erythema
Herpes zoster	Acute	Elderly, immunosuppression	Exquisitely tender erythematous or vesicular rash following a dermatome
Malignant otitis externa	Gradual	Diabetes, water exposure	Ear pain with drainage, facial swelling, tragal tenderness
Trauma			
Soft tissue contusion	Acute	Associated trauma	Tender swelling
Burn	Acute	Occupational, recreational exposure	May be difficult to distinguish from cellulitis
Inflammatory			
Insect envenomation	Acute	Environment supporting insect life	Diffuse, red, puffy
Apical abscess with secondary buccal swelling	Gradual	Usually associated dental pain/caries	Similar to cellulitis; may have intraoral/gingival findings
Contact dermatitis	Gradual or acute	Often identifiable exposure	Variable; maculopapular, itchy rash
Immunologic			
Systemic lupus erythematosus	Gradual	Female-to-male ratio, 9:1	Erythema in classic "malar" distribution
Angioneurotic edema	Acute	Exposure to angiotensin-converting enzyme inhibitor, allergen	Lip, oral mucosal swelling, sometimes facial
Vancomycin flushing reaction	Acute	Recent exposure to vancomycin	Facial erythema, warmth

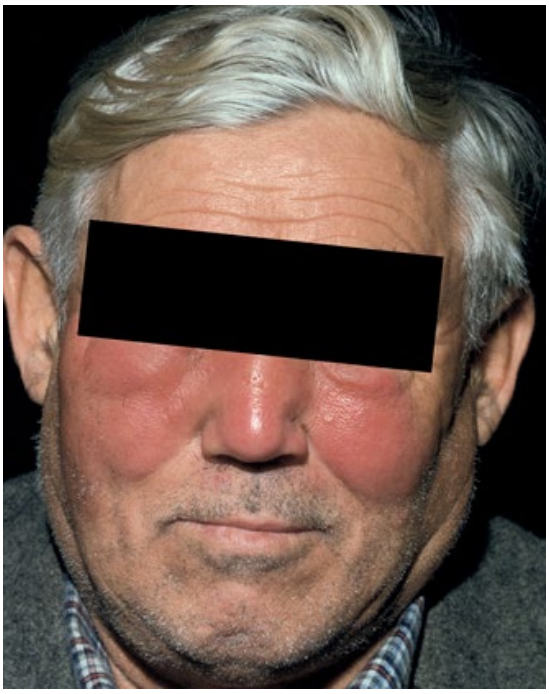
- TREATMENT AND DISPOSITION:
    - Remove foreign bodies and medical devices from the affected area
    - Most patients can be treated with PO antibiotics
    - Fever, difficulty swallowing or breathing, inability to take or tolerate oral antibiotics or clinical deterioration are indications for return to ED
    - Consider hospitalisation for systemic signs of sepsis, antibiotic intolerance, immunosuppression, extensive erythema or induration or if devices/FB cannot be removed in ED
    - Abscesses should be drained
    - Antibiotic should continue for 7-14 days
        - Treatment failure rates of ~15-20% for beta-lactams
    - Treatment overview for superficial infections shown below:

Table 238-2 Antibiotic Therapy for Facial Infections	
Cellulitis	Oral therapy: dicloxacillin, cephalexin, clindamycin; vancomycin and cephalosporins are alternatives
	Suspected MRSA: trimethoprim-sulfamethoxazole, clindamycin, doxycycline, or minocycline
	Parenteral therapy: nafcillin, vancomycin, clindamycin
	Total duration 7–14 d
Erysipelas	Oral therapy: penicillin
	Methicillin-sensitive *Staphylococcus aureus* suspected: amoxicillin/clavulanate, cephalexin, dicloxacillin
	Bullous erysipelas: trimethoprim-sulfamethoxazole, clindamycin, doxycycline, or minocycline
	Parenteral therapy: vancomycin, nafcillin, clindamycin
	Total duration 7–14 d
Impetigo	Topical: mupirocin or retapamulin ointment alone or with oral therapy
	Oral therapy: dicloxacillin, amoxicillin/clavulanate, cephalexin
	Alternative: azithromycin
	MRSA suspected: clindamycin or trimethoprim-sulfamethoxazole
	Total duration 7 d[2]
Suppurative parotitis	Parental therapy: nafcillin, amoxicillin-clavulanate or ampicillin-sulbactam; if penicillin allergic, clindamycin or the combination of cephalexin with metronidazole, or vancomycin with metronidazole
	Hospital acquired or nursing home patients: consider vancomycin
	Total duration: 10–14 d
Masticator space infection	Parenteral therapy: IV clindamycin is recommended; alternatives include ampicillin-sulbactam, cefoxitin, or the combination of penicillin with metronidazole
	Oral therapy: clindamycin or amoxicillin-clavulanate
	Total duration: 10–14 d

ERYSIPELAS:

- PATHOPHYSIOLOGY:
    - A specific form of cellulitis involving the epidermis, upper dermis and lymphatics
    - Caused by Strep pyogenes in the majority of cases
- CLINICAL FEATURES:
    - Red, raised, puffy appearance with a sharply defined or shiny, palpable borer that advances rapidly

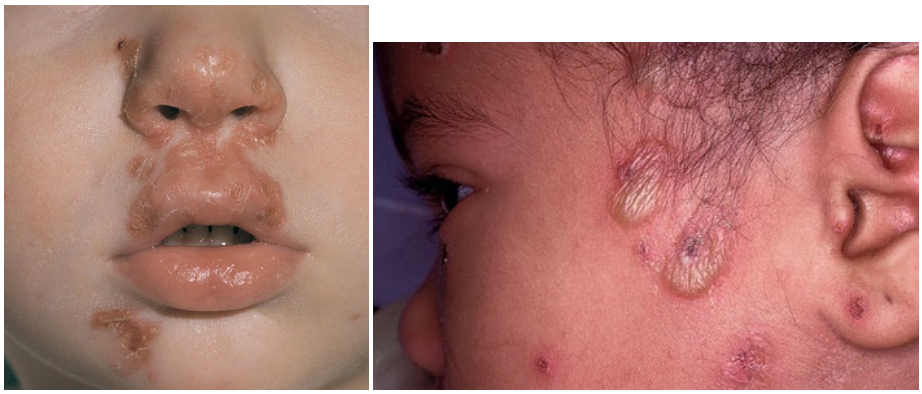
- Differential is the same as for cellulitis



- TREATMENT AND DISPOSITION:
  - TYPICALLY TREATED WITH ORAL ANTIBIOTICS AS OUTATIENTS
  - Consider hospitalisation for those who are immunocompromised, or have evidence of systemic illness
  - Penicillin remains the antibiotic of choice

IMPETIGO:

- PATHOPHYSIOLOGY:
  - A discrete, superficial epidermal infection, characterised by amber crusts (non-bullous form) or by fluid-filled vesicles (bullous form)
  - Staph aureus and Strep pyogenes are the two most common causes
- CLINICAL FEATURES:
  - Well-localised lesions



Non-bullous impetigo (left), bullous (right)

- TREATMENT:

Until culture results are available, in nonremote community settings suspect ***Staphylococcus aureus*** as the pathogen. Drugs that are active against *S. aureus* will also cover *S. pyogenes*.

For **mild or localised infections**, use:

soap and water topically, 8-hourly to soften crusts

PLUS

mupirocin 2% ointment or cream topically to any crusted areas, 8-hourly for 7 days.

For **widespread or recurrent infections**, use:

di/flucloxacillin 500 mg (child: 12.5 mg/kg up to 500 mg) orally, 6-hourly for 10 days.

Cephalexin can be used for patients with penicillin hypersensitivity (excluding immediate hypersensitivity, see Table 2.2) and is a useful alternative to di/flucloxacillin in children due to better tolerability, and palatability of the liquid formulation. Use:

cephalexin 1 g (child: 25 mg/kg up to 1 g) orally, 12-hourly for 10 days.

For patients with immediate penicillin hypersensitivity (see Table 2.2), use:

roxithromycin 300 mg orally, once daily (child: 4 mg/kg up to 150 mg orally, 12-hourly) for 10 days.

In situations where ***Streptococcus pyogenes*** is confirmed, or suspected (eg in Indigenous communities in central and northern Australia), use:

soap and water topically, 12-hourly to soften crusts

PLUS EITHER

1 **benzathine penicillin 900 mg (child 3 kg to less than 6 kg: 225 mg; 6 kg to less than 10 kg: 337.5 mg; 10 kg to less than 15 kg: 450 mg; 15 kg to less than 20 kg: 675 mg; 20 kg or more: 900 mg) IM, as a single dose [Note 1]**

OR

1 **phenoxymethylpenicillin 500 mg (child: 12.5 mg/kg up to 500 mg) orally, 6-hourly for 10 days.**

For patients with penicillin hypersensitivity (see Table 2.2), use:

roxithromycin 300 mg orally, once daily (child: 4 mg/kg up to 150 mg orally, 12-hourly) for 10 days.

SALIVARY GLAND INFECTIONS:

- There are three groups of salivary glands ➔ the parotid, submandibular and sublingual
- CLINICAL FEATURES:
  - Signs are unilateral or bilateral facial swelling
  - Recurrent symptoms, dry eyes or mouth, or joint symptoms suggest immunologic or collagen vascular disorders
  - Differential diagnosis provided below, followed by discussion of most common causes of salivary gland enlargement as well as discussion on sialolithiasis

Table 238-4 Differential Diagnosis of Salivary Gland Swelling

	Historical Features		Clinical Features
	Onset	Risks/Inciting Factors	
Infectious			
Viral parotitis (mumps)	Gradual	Nonimmunized	Prodromal illness, unilateral tense swelling, absent warmth/erythema
Buccal cellulitis		*Haemophilus influenzae* infection in nonimmunized	Erythematous, tender
Suppurative parotitis	Rapid	Dehydration, immunosuppression, chronic illness, recent anesthesia	Painful buccal swelling, fever, pus expression from Stensen's duct
Masseter space abscess	Gradual	—	—
Tuberculosis	Gradual	Exposure, immunosuppression	—
Immunologic			
Sjögren syndrome	Gradual	—	Dry mouth, eyes, sclerosis
Systemic lupus	Gradual	Female sex, Asian, or African American race	No signs of infection
Sarcoidosis	Gradual	Female sex, African American race	No signs of infection
Other			
Neoplasm	Gradual	—	No erythema, warmth
Sialolithiasis	Gradual	Dehydration, chronic illness	Swelling, tenderness, no signs of infection

- **VIRAL PAROTITIS (MUMPS):**
  - PATHOPHYSIOLOGY:
    - An acute infection of the parotid glands, most often caused by PARAMYXOVIRUS but less commonly by influenza, parainfluenza, coxsackievirus, HIV
    - Most common in children <15
    - Vaccination has reduced its incidence significantly
  - CLINICAL FEATURES:
    - After prodrome of fever, malaise, headache, myalgias and arthralgias, the classic salivary gland swelling then follows (typically bilateral involvement)
    - Diagnosis is clinical
  - TREATMENT:
    - Supportive
    - The patient is contagious for 9 days after onset of parotid swelling
    - Usually benign in children but can be severe in adults
    - Unilateral ORCHITIS can affect 20-30% of males, whereas oophoritis affects only 5% females
    - Other complications to be mindful of → mastitis, pancreatitis, aseptic meningitis, sensorineural hearing loss, myocarditis, haemolytic anaemia, polyarthritis
- **SUPPURATIVE PAROTITIS:**
  - PATHOPHYSIOLOGY:
    - A serious bacterial infection of the parotid gland that occurs in patients with COMPROMISED SALIVARY FLOW
    - Caused by retrograde migration of oral bacteria in to the salivary ducts and parenchyma
    - Predisposing factors to suppurative parotitis include recent anaesthesia, dehydration, prematurity or advanced age, sialolithiasis, oral neoplasms, salivary duct strictures, tracheostomy and ductal foreign bodies
    - Medications that cause either systemic dehydration or decrease salivary flow specifically can cause parotitis (TCA, diurietics, antihistamines, β-blockers)
    - Several chronic illnesses predispose ➔ Sjogren, HIV, hepatic/renal failure, DM, hypothyroidism, CF, depression.
  - CLINICAL FEATURES:
    - The onset IS RAPID
    - Skin overlying parotid gland is red and tender
    - PUS MAY BE EXPRESSED FROM STENSEN'S DUCT
    - Often there is fever and trismus
  - DIAGNOSIS:
    - Diagnosis is CLINICAL
    - Imaging is not helpful unless an abscess is suspected, in which case US or CT scanning are diagnostic
  - TREATMENT/DISPOSITION:

- Treatment should OPTIMISE SALIVARY FLOW ➔ hydration of the volume-depleted patient. Massage and apply heat to the affected gland. STIMULATE SALIVATION USING SIALOGOGUES SUCH AS LEMON DROPS
- When possible, discontinue drugs that cause dry mouth.
- Same approach for other glands/sialadenitis
- Antimicrobial therapy for coverage of Staph and Strep an dsurgical input if there is a fluctuant collection

Management of acute suppurative sialadenitis includes surgical review, intraductal or surgical drainage if fluctuant, rehydration and antibiotics.

1 **di/flucloxacillin 2 g (child: 50 mg/kg up to 2 g) IV, 6-hourly**
then di/flucloxacillin 500 mg (child: 12.5 mg/kg up to 500 mg) orally, 6-hourly for a total of 10 days

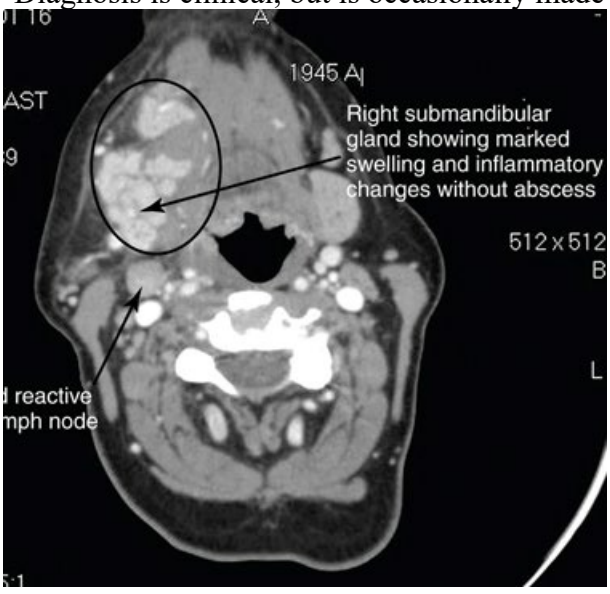
OR

2 **clindamycin 450 mg (child: 10 mg/kg up to 450 mg) IV or orally, 8-hourly for a total of 10 days**

OR

2 **lincomycin 600 mg (child: 15 mg/kg up to 600 mg) IV, 8-hourly**
then clindamycin 450 mg (child: 10 mg/kg up to 450 mg) orally, 8-hourly for a total of 10 days.

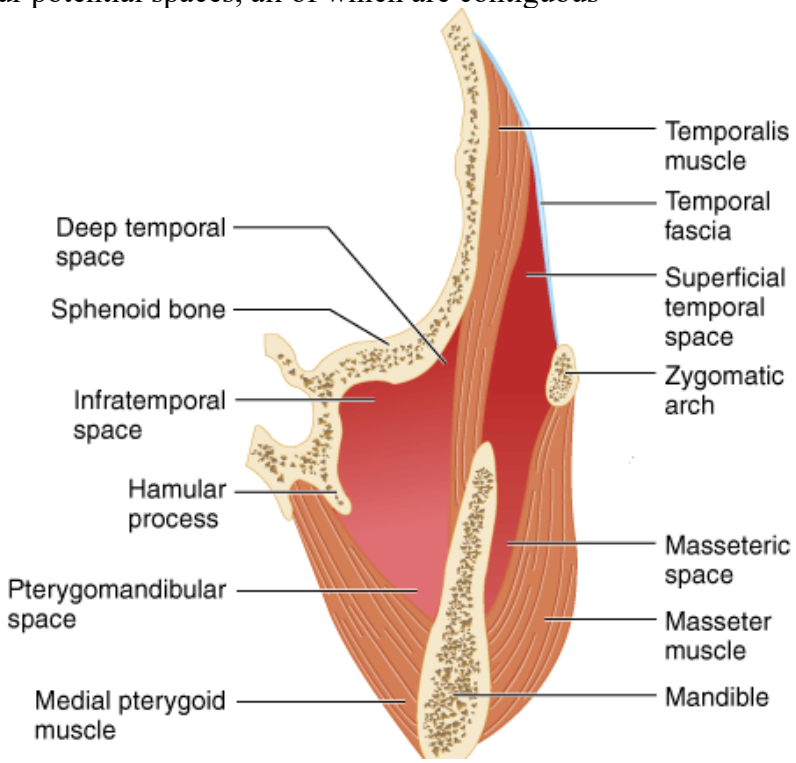
- **SIALOLITHIASIS:**
  - PATHOPHYSIOLOGY:
    - Development of a calcium stone in a stagnant salivary duct
    - Salivary calculi usually develop in men between the third and sixth decades
    - More than 80% of stones occur in the SUBMANDIBULAR GLAND as the secretions are more viscous and its ascending course
  - CLINICAL FEATURES:
    - The symptoms of pain, swelling and tenderness resemble parotitis
      - However, the pain and swelling of sialolithiasis is exacerbated by meals and may develop over the course of minutes when eating ➔ typically unilateral
  - DIAGNOSIS:
    - Diagnosis is clinical, but is occasionally made on CT



- TREATMENT AND DISPOSITION:
    - Outpatient therapy with analgesia, oral antibiotics if there is superimposed infection, massage and sialogogues such as lemon drops
    - Complications ➔ recurrent or persistent obstruction, strictures, infection, gland atrophy

MASTICATOR SPACE INFECTION:

- PATHOPHYSIOLOGY:
    - Four potential spaces, all of which are contiguous



    - Bacterial agents invade all of these spaces and may gain entry from dental infections, trauma, surgery or injections
    - POLYMICROBIAL INFECTIONS ARE CHARACTERISTIC
- CLINICAL FEATURES:
    - The most frequent findings are facial swelling, pain, erythema and trismus
    - Constitutional signs may include fever, malaise, dehydration, dysphagia, nausea or vomiting
- DIAGNOSIS:
    - Contrast-enhanced CT is the preferred tool for deep space infections
    - CT scan can define the extent of the abscess
- TREATMENT AND DISPOSITION:
    - The patient's condition determines therapy
    - Be aware that these tissue planes EXTEND DOWN THE NECK TO THE MEDIASTINUM, AND THE EXTENT OF INFECITON SHOULD BE DEFINED EFFICIENTLY AND TREATMENT BEGUN PROMPTLY
    - Airway compromise is rare but should be considered

- The role of ED is to control the airway, stabilise the patient, and treat for sepsis
- COMBINATION OF PENICILLIN AND METRONIDAZOLE

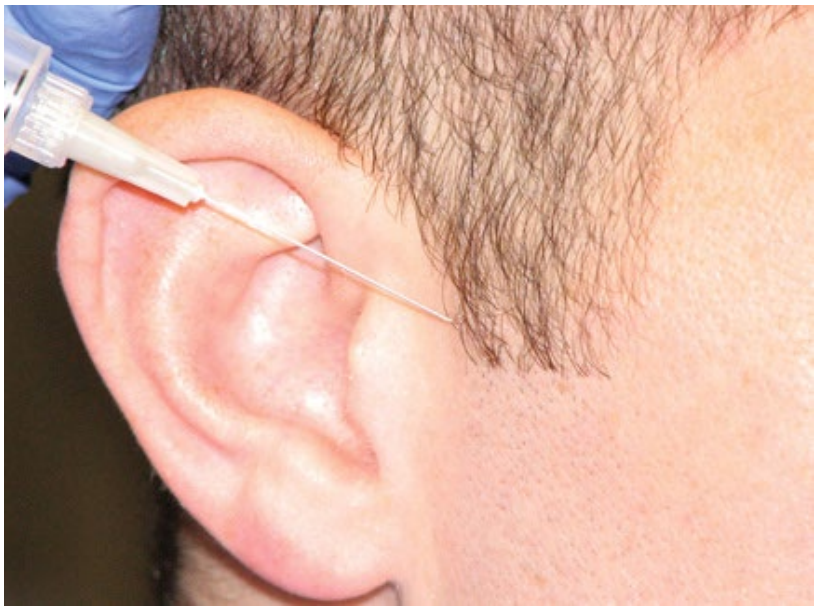
TEMPOROMANDIBULAR JOINT DISORDERS:

TEMPORMANDIBULAR JOINT DYSFUNCTION:

- PATHOPHYSIOLOGY:
  - TMJ dysfunction causes pain of the joint and its surrounding structures
  - Variety of causes ➔ neuromuscular, anatomic deviations as result of congenital defect or trauma, dental abnormalities
- CLINICAL FEATURES:
  - Chief complaint is USUALLY PAIN LOCLISED TO ONE OF THE MUSCLES OF MASTICATION OR PAIN WITH CHEWING
  - Physical findings ➔ limitation in the range of motion of the mandible
  - Differential diagnosis ➔ jaw trauma (fracture and dislocation), odontogenic pain, otologic referred pain, temporal arteritis
- TREATMENT AND DISPOSITION:
  - Oral maxillofacial surgeon manages fractures and should be consulted for trismus and significant mandible displacement
    - Otherwise, simple fractures → analgesia, soft diet and referral less urgently
    - Chronic TMJ dysfunction may require referral to dentist, maxillofacial surgeon or pain specialist

DISLOCATION OF THE MANDIBLE:

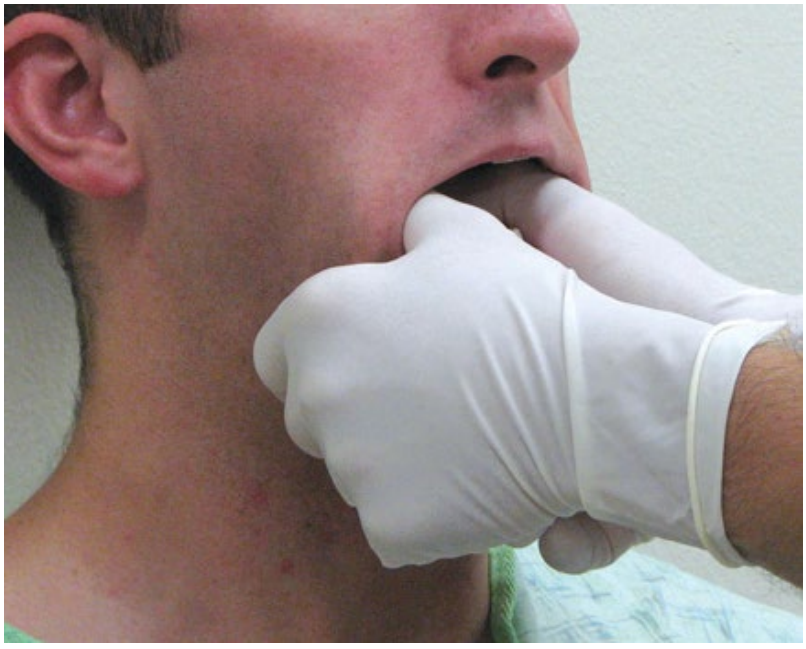
- PATHOPHYSIOLOGY:
  - The mandible can be dislocated in an anterior, posterior, lateral or superior direction
  - ANTERIOR DISLOCATION IS MOST COMMON and occurs when the manibular condyle is forced in front of the articular eminence. Muscular spasm then traps the mandible in anterior dislocation
    - RF ➔ shallow glenoid fossa, seizure, prior trauma to capsule
  - Dislocations are usually bilateral
  - In POSTERIOR DISLOCATION ➔ the articular condyle is thrust backward against the mastoid and the condylar head may prolapse into the external auditory canal
  - LATERAL DISLOCATION ➔ Occurs with mandibular fracture
  - Posterior, superior and lateral dislocations all occur with severe trauma
- DIAGNOSIS:
  - Diagnosis is clinical in the case of spontaneous atraumatic anterior dislocation
  - Otherwise, OPG will confirm diagnosis
- TREATMENT:
  - Reduction may be attempted in closed anterior dislocation without fracture
  - Can give local anaesthesia to aid reduction as shown below



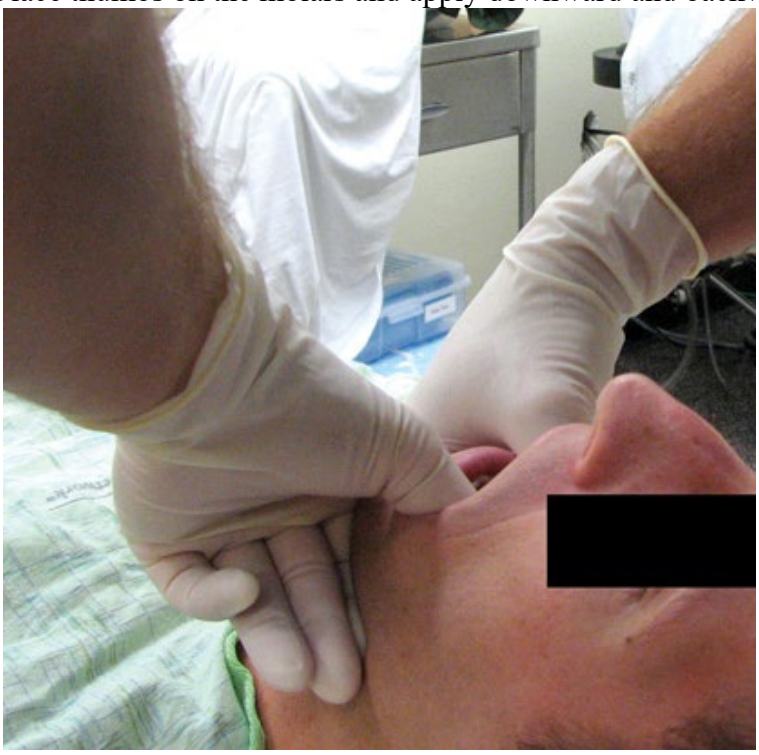
Give local anaesthetic in the preauricular depression just anterior to the tragus

REDUCTION OF THE ANTERIOR TMJ DISLOCATION:

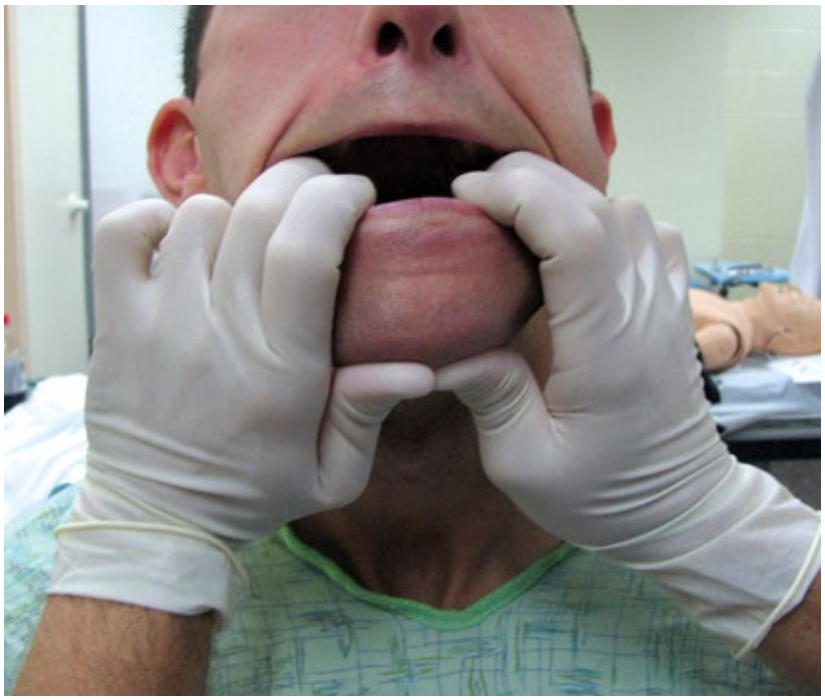
- Most commonly used technique requires the patient to be seated with the head against the wall or chair back
- Apply a few layers of gloves for protection
- Facing the patient, place gloved thumbs in the patient's mouth, over the occlusal surfaces of the mandibular molars
    - Curve your fingers beneath the angle and body of the mandible
    - Using the thumbs apply pressure downward and backward toward the patient
    - Slightly opening the jaw may help disengage the condyle



- OTHER TECHNIQUES:
  - Patient recumbent and supine
  - Stand at head of the bed
  - Place thumbs on the molars and apply downward and backward pressure



- WRIST PIVOT METHOD:
  - Operators thumbs are placed on the mentum, applying upward force, while the fingers apply downward force on the body of the mandible



- Complications of the reduction itself are unusual but can include iatrogenic fracture or avulsion of the articular cartilage

DISPOSITION AND FOLLOW UP:

- Patients with dislocations that are open, superior, associated with fracture, have any nerve injury or are irreducible by closed technique should be referred emergently to a head and neck or oral surgeon
- Once reduced ➔ soft diet and caution against mouth opening beyond >2cm for two weeks