CHAPTER 6

SURGICAL MANAGEMENT OF PATIENTS WITH OROFACIAL INFECTIONS
6.1 Introduction

Patients presenting with an orofacial infection may have signs and symptoms ranging from the trivial to the extremely serious. A rapid initial assessment of the patient's status is necessary to determine if simple treatment will alleviate the problem or if the patient is showing signs of toxicity or airway compromise which require immediate hospitalisation and aggressive medical and surgical intervention.

6.2 Pathways of Dental Infection

Serious dental infection spreading beyond the tooth socket may result from pulpal necrosis, periodontal disease or pericoronal infection (Table 3). The pathophysiology of a given infectious process varies depending on the number and virulence of the organism, host resistance and anatomy of the involved region (Topazian and Goldberg, 1987).

Table 3: Pathways of Dental Infection

- fistula
- bacteremia-septicaemia
- cellulitis
- deep fascial space infection
- intraoral soft tissue abscess
- osteomyelitis
- ascending facial-cerebral infection
- dental infection

Infection spreads buccally or lingually from infected teeth and may remain subperiosteal or break through above or below muscle attachments to enter the fascial spaces (Figure 20). The clinical significance of fascial spaces is that when infection develops and extends within these spaces there is little resistance to the spread of infection and it is frequently
FIGURE 20. Some pathways of spread of dental infection
necessary to establish surgical drainage (Waite 1960).

Any infection of dental origin requires definitive treatment of the offending tooth if the source of the infection is to be eliminated, and extraction of the involved tooth is the most rapid method of establishing drainage. The question of whether or not an abscessed tooth should be extracted in the presence of acute infection has been a controversial one. Now, with the utilisation of antibiotic therapy, a tooth may be extracted during the acute stage of diffuse or deep infections (Goldberg and Topazian 1987, Krogh 1951) together the drainage of the dentoalveolar abscess. Incision and drainage represent one of the oldest surgical procedures and may be performed either intra- or extra- orally depending on the extent of the orofacial infection.

6.3 Principles of Incision and Drainage

Flynn and Topazian (1987) advised that the following principles should be utilised when employing incision and drainage of orofacial infections:

1. Incise in healthy skin and mucosa when possible. An incision placed at the site of maximum fluctuancy where the tissues are necrotic or beginning to perforate may result in a puckered, unaesthetic scar.

2. The incision should be placed in an aesthetically acceptable area in a natural skin crease.

3. The incision should be placed in a dependent position to encourage drainage by gravity.

4. The abscess cavity should be widely explored following blunt dissection through deeper tissues so that any locules of pus are opened.

5. The drain should be stabilised with sutures.

6. In extra-oral cases through-and-through drains can be used.

7. The drains should be removed when drainage becomes minimal.

8. The wound margins should be cleaned daily under sterile conditions to
remove clots and debris (Plates 4, 5, 6, 7).

6.4 Anatomical Considerations in Dentoalveolar Infections

The position of a dentoalveolar abscess is related to the anatomic position of the dental root from which it originated and to muscle attachments especially the buccinator and mylohyoid muscles. Infections usually follow the path of least resistance and it is unusual for a dental abscess to appear distant from its site of origin (Figure 21).

Infections associated with Mandibular Teeth

Infections of mandibular incisors and cuspids usually present as bulging erythematous masses in the labial sulcus, which are readily accessible for drainage. Infection at a lingual site is less common and is usually treated by drainage requiring a simple gingival incision at the lingual surface. The mandibular bicuspids may also exhibit buccal infection but care is required in making an incision because of the presence of the mental nerve and its foramen. If the infection extends inferior to the buccinator muscle laterally, or inferior to the mylohyoid muscle linguually, deep infections of the buccal and submandibular spaces may occur. Infection of mandibular molars may involve the buccal vestibule, buccal fascial space, masticator, (Figure 22), pharyngeal, sublingual and submandibular spaces, which may require both intra- and extra-oral drainage.

Infections associated with Maxillary Teeth

The maxillary incisor and canine teeth roots lie closer to the thin labial plate of bone and infections are seen as bulging vestibular abscesses in the labial sulcus. The muscles of the upper lip are quite thin and may allow a generalised cellulitis of the upper lip or midface to occur. Drainage is achieved by sharp incision in the labial sulcus. When palatal infection occurs from an anterior tooth it varies from minimal swelling to massive bulging of the anterior palate. Incision of the palatal mucosa will
PLATE 4. Left Submandibular Space Abscess.

PLATE 5. Aspiration of left submandibular space abscess.
A. mouth, B. left lower border of mandible, C. left angle of mandible, D. line of incision for drainage.
PLATE 6. Incision and drainage of left submandibular space abscess.

PLATE 7. Maintenance of drainage with the placement of a Penrose drain.
FIGURE 21. Frontal section of the head showing routes of infections originating from the teeth
FIGURE 22. Anatomic scheme of the normal (right) and diseased (left) masticator space in axial and coronal projections
provide drainage and the palatal vessels are avoided by making the incision parallel to them. Maxillary bicuspid and molar infection may extend buccally or palatally. Drainage of a buccally situated abscess is obtained by incision high in the buccal vestibule (Figure 23). Posterior spread of infection may involve the masticator and pharyngeal spaces while superior extension into the infratemporal space may also occur (Figure 24).

6.5 Surgical management of fascial space infections

When dental infection spreads deeply into soft tissue, rather than exiting superficially through oral or cutaneous routes, fascial spaces may become involved. The fasciae and fascial planes offer an anatomically comprehensible highway for the spread of infection from superficial to deep parts of the face and neck (Figure 25 and Table 4).

In the surgical treatment of fascial space infections the principles stated earlier (p.144 6.3) apply, together with the following points (Topazian and Goldberg 1987):

1. Diffusion of antibiotics into close fascial spaces is limited because of poor vascularity. Penetration of antibiotics through thick-walled abscesses is minimal. "Average" doses may be inadequate.

2. Therapy of fascial space infections is dependent upon adequate, open, dependent drainage.

3. Large surgical incisions are necessary to obtain adequate surgical exposure to deep compartments.

4. Fascial spaces are contiguous, and infection spreads readily from one space to another.

5. Secondary spaces as well as the primary one must be drained.

6. The anatomy of the face or neck may be grossly distorted by the swelling of the infectious process.

7. Repeat surgical drainage may be necessary". 
FIGURE 23. Usual intraoral incision sites for drainage

a. Infratemporal space
b. Pterygoid space
c. Temporalis muscle
d. Temporal fascia
e. Masseter muscle
f. Medial pterygoid muscle
g. Lateral pterygoid muscle (dotted outline)

FIGURE 24. A diagram of the pterygoid and infratemporal spaces in coronal section
FIGURE 25. A mid-sagittal section of the head, neck and thorax. The arrows show the spread of infection from the floor of the mouth to the subphrenic area.
Table 4: FASCIAL COMPARTMENTS OF CLINICAL SIGNIFICANCE

(Topazian & Goldberg 1987)

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Face
- Canine
- Buccal
- Masticator
  - masseter
  - pterygoid
  - temporal
- Parotid

Suprathyroid
- Sublingual
- Submandibular
  - submaxillary
  - submental
- Pharyngomaxillary (lateral pharyngeal)
- Peritonsillar

Infrahyoid
- Anterovisceral (pretracheal)

Spaces of the Total Neck
- Retropharyngeal
- Danger space
- Space of carotid sheath
6.5.1 Canine Space

This fossa contains considerable amounts of connective tissue and fat that allow the accumulation of tissue fluids and pus. The fossa is bounded:
- superiorly by the levator labii superioris, zygomaticus minor and levator labii superioris alaeque nasi muscles
- anteriorly by the orbicularis oris muscle
- posteriorly by the buccinator muscle

Involvement of this space may develop from infected maxillary anterior or bicuspid teeth (Figure 26, Plates 8 and 9). When the space has been invaded, the patient has facial swelling that obliterates the nasolabial fold, oedema of the upper and lower eyelids that closes the eye and swelling of the upper lip that frequently causes drooping of the corner of the mouth. The affected side of the face is invariably tender and painful and the skin taut and red. Ascending infection from the maxillary teeth through the valveless anterior and posterior facial veins may lead onto cavernous sinus thrombosis.

Drainage of the canine fossa infection is best accomplished through an intra-oral approach, high in the maxillary labial vestibule, by sharp and blunt dissection.

6.5.2 Buccal Space

The relation of the root apices of the mandibular and maxillary bicuspid and molar teeth to the origins of the buccinator muscle (the outer surfaces of the alveolar process of the maxilla and mandible) determines whether infection will exit intra-orally in the buccal vestibule or deep into the buccal space. The buccal space is situated between the buccinator muscle and the masseter muscle bounded:
- medially by the buccinator muscle and buccopharyngeal fascia
- laterally by the skin of the cheek
- anteriorly by the labial musculature
FIGURE 26. The Canine Fossa Space.
The arrows show the direction the pus takes from the apices of the canine and the premolars. (a) Levator labii superioris alaeque nasi; (b) Levator labii superioris; (c) Levator anguli oris; (d) Zygomaticus minor; (e) Zygomaticus major; (f) Buccinator; (g) Orbicularis oris.

PLATE 8. Left Canine Fossa Space Infection.
PLATE 9. Left Canine Fossa Space Infection showing swelling of the upper lip and nasolabial fold with closure of the eye.
- superiorly by the zygomatic arch
- inferiorly by the lower border of the mandible
- posteriorly by the pterygomandibular raphe

The buccal space contains the buccal fat pad, Stenson's (parotid) duct, and the facial artery (Figure 27 and 28). Infection of this space is diagnosed easily because of marked cheek swelling associated with a diseased molar or bicuspid tooth (Plate 10). Fluctuance, when it occurs, is cutaneous and drainage is performed inferior to the point of fluctuance.

6.5.3 Masticator Spaces

These spaces - masseteric, pterygoid and temporal - are well differentiated but communicate with each other as well as with the buccal, submandibular and parapharyngeal spaces (Figures 29, 30 and 31). Infection may be confined to one of these compartments or spread to involve them all. The masticator space lies lateral and anterior to the lateral pharyngeal space and contains the masseter muscle, mandibular ramus, pterygoid muscles, the tendon of insertion of the temporalis muscle and the inferior alveolar vessels and nerves. The two distinct compartments are the medial or deep space known as the pterygoid space and the lateral or superficial space known as the masseteric space. Anteriorly and posteriorly they come together around the border of the mandible to which they are attached. Superiorly, deep to the zygoma the spaces are in communication with the space superficial and deep to the temporalis muscle and tendon - the temporal space. This space is limited superficially by the attachment of the thick outer temporal fascia to the temporal ridge and zygoma. There is a communication between the superficial temporal space and the masseteric space, while the deep temporal space communicates with the pterygoid space.

Infection of the masticator space occurs most frequently from molar teeth especially the wisdom teeth, though non-odontogenic pathology should
FIGURE 27. A diagram showing the buccal space in horizontal section and the spread of infection from an impacted lower third molar tooth.

a. Medial pterygoid muscle  
b. Mandible  
c. Masseter muscle  
d. Buccal space  
e. Parotid gland

FIGURE 28. Buccal space abscess
PLATE 10. Right Buccal Space Infection.
FIGURE 29. A diagram showing the relations of the peritonsillar, lateral pharyngeal, and pterygoid spaces

a. Pterygoid space  b. Peritonsillar space
c. Lateral pharyngeal  d. Superior constrictor
e. Carotid artery  f. Vagus nerve
g. Internal jugular vein  h. Stylopharyngeus muscle
i. Styloglossus muscle  j. Styloid process
k. Stylohyoid muscle  l. Mastoid process
m. Digastric muscle  n. Parotid gland
o. External carotid artery  p. Mandible
q. Facial nerve  r. Inferior dental artery
s. Masseter muscle  t. Lingual nerve
u. Temporalis tendon  v. Buccinator muscle
FIGURE 30. Masseteric, pterygoid and temporal compartments of the masticator space.

FIGURE 31. Extraoral surgical approaches to the submental space A, the submandibular space B, and the masticator compartments C and D.
also be considered (Calcaterra 1984). Clinically, the most noticeable features of an acute infection in the masseteric space are:

- sudden onset
- severe, deep-seated throbbing pain over the ascending ramus of the mandible
- swelling of the soft tissues of the face
- rapid development of severe trismus
- high temperature
- tense, shiny appearance of the tissues over the posterior part of the ramus
- excruciating pain

Clinical features associated with involvement of the pterygoid space are:

- minimal facial swelling
- constant pain
- severe trismus
- soreness of the throat
- difficulty in swallowing
- discomfort in moving tongue
- intra-oral swelling in the retromolar region

If the infection extends to involve the temporal space then external swelling of the temporal region above the zygomatic arch is seen (Plates 11, 12, 13 and 14).

Surgical access to the various compartments of the masticator space is complicated by the containment of the infectious process by the muscle masses. The masseteric and pterygoid spaces can be drained intra-orally by an incision along the external oblique ridge and extra-orally via an incision below the angle of the mandible, avoiding the mandibular branch of the facial nerve. The extra-oral approach allows for dependent drainage of both spaces
PLATE 11. This lateral facial view demonstrates the spread of infection from an impacted lower right third molar tooth which has involved the masseteric and submandibular spaces.
PLATE 12. This intra-oral view demonstrates an oral infection associated with the impacted lower right third molar tooth. The evidence of trismus and swelling of the lingual tissues and soft palate is seen.
PLATE 13. This full facial view demonstrates the spread of infection to the right temporal fascial spaces from the right masseteric and pterygoid spaces from an infected right mandibular fracture.
PLATE 14. This lateral facial view demonstrates massive facial infection involving multiple fascial spaces which include submandibular, masticator, buccal, canine and temporal spaces.
at the insertion of the muscle sling on the inferior border at the mandibular angle. The temporal space, although accessible through the intra-oral incision, may also be drained percutaneously through an incision slightly superior to the zygomatic arch. The incision should be made parallel to the zygomatic arch and therefore parallel to the zygomatic branch of the facial nerve rather than perpendicular to it (Plates 15 and 16).

6.5.4 Parotid Space

The parotid gland is surrounded by a dense fibrous capsule so that fluctuance is rarely observed and spontaneous drainage uncommon. The course of the disease and the patient's response to antibiotic therapy and hydration will determine whether or not surgical drainage is necessary. Modern antimicrobial therapy has decreased the frequency of surgical intervention but the patient whose infection progresses rapidly will still need surgery. Drainage is accomplished by surgical exposure of the gland and penetration of the capsule by blunt probing, thus opening many deep loculations within the glandular parenchyma. A small clamp is an ideal instrument for the penetration procedure and when opened and spread in the same direction as the course of the facial nerve, minimizes the danger to these fibres. Gross purulent flow may not be obtained but serous drainage and decompression of the gland are often adequate to provide relief. The wound should be packed open with moistened saline packs that are changed daily and reduced in size as discharge diminishes.

6.5.5 Submandibular and Sublingual Spaces

These spaces are considered as a surgical unit because of their proximity and because of their frequent dual involvement in odontogenic infection. The mylohyoid muscle, forming the floor of the oral cavity, is the key to the diagnosis and surgical management of these space infections. The mylohyoid which attaches to the lingual surface of the mandible in an
PLATE 15. The extra-oral placement of a Penrose drain into the right temporal space.
PLATE 16. The intra-oral placement of a Penrose drain into the right masticator space (temporal, masseteric and pterygoid spaces).
obliquely downward line from posterior to anterior separates the submandibular space below from the sublingual space above.

The sublingual space (Figure 32 and Plate 17) contains the sublingual gland and Wharton's duct and is bounded:
- anteriorly and laterally by the lingual surface of the mandible
- superiorly by the oral mucosa of the floor of the mouth
- inferiorly by the mylohyoid muscle
- posteriorly by the hyoid bone
- medially by the genioglossus, geniohyoid and styloglossus muscles

Only loose connective tissue actually separates one side of the floor of the mouth from the other, allowing infection to spread bilaterally with ease. Anteriorly, the sublingual space communicates with the submental space and posteriorly with the lateral pharyngeal spaces. Clinically, infection of the sublingual space presents as brawny, erythematous tender swelling of the floor of the mouth, beginning close to the mandible and spreading toward the midline or beyond. Surgical drainage of the sublingual space should be performed intra-orally by an incision through the mucosa parallel to Wharton's duct (Plate 18). If the submandibular space is also to be drained, both spaces can be reached through a submandibular approach.

The submandibular space (Figure 33, Plates 19, 20, 21, 22, 23 and 24) contains the submandibular salivary gland and its lymph nodes, the facial artery, the proximal portion of Wharton’s duct and the lingual and hypoglossal nerves. Odontogenic infections of this space are commonly caused by the second and third mandibular molar teeth. The space is bounded:
- laterally by the submandibular skin, superficial fascia, platsyma muscle, superficial layer of the deep cervical fascia and the lower border of the mandible
- medially by the mylohyoid, hyoglossus and styloglossus muscles
FIGURE 32A. Sublingual space, anatomical boundaries

FIGURE 32B. Sublingual space, pathological changes
PLATE 17. This facial view demonstrates a left sublingual space infection with mild facial swelling arising from a non-vital lower left second incisor tooth.
PLATE 18. This intra-oral view demonstrates the drainage of a left sublingual space infection by means of a mucosal incision which is parallel to Wharton’s duct.
FIGURE 33A. A diagram of the submandibular space
a. Submandibular salivary gland and lymph nodes
b. Hyoglossus muscle
c. Mylohyoid muscle
d. Anterior belly of the digastric muscle
e. Posterior belly of the digastric and stylohyoid muscles
f. Middle constrictor
g. Styloid process and stylohyoid ligament

SUBLINGUAL GLAND

FIGURE 33B. Submandibular space infection
PLATE 19. This full frontal view demonstrates a left submandibular space infection.
PLATE 20. This facial view, with the head extended, demonstrates a spreading left submandibular space infection.
PLATES 21 and 22. These facial views demonstrate right submandibular and masseteric space infections which have arisen from impacted and carious teeth.
PLATE 23. The surgical incision and drainage with the placement of a Penrose drain into the right submandibular space (lateral view).
PLATE 24. The surgical incision and drainage with the placement of a Penrose drain into the right submandibular space (frontal view).
- inferiorly by the anterior and posterior bellies of the digastric muscle

- posteriorly by the hyoid bone

Clinical diagnosis of submandibular space infection is made by finding the typical swelling of the space, either brawny or soft. Infection may be secondary to sepsis in an adjoining space such as the sublingual, submental or masticator. Conversely, infection may spread from the submandibular space into any contiguous space including the pharyngeal spaces and can cross the midline into the contralateral submandibular space. If spread is bilateral and involves all submandibular and sublingual spaces the result is Ludwig's Angina (Figure 34, 35 and Plates 25, 26, 27 and 28). Surgical drainage of the space is performed through the skin below the mandible and parallel to it. Blunt dissection is carried to the depths of the space as well as to its anterior and posterior margins.

The submental space (Figure 36) is a potential fascial space existing in the chin which occasionally becomes infected either directly from a mandibular incisor or indirectly from the submandibular space. The submental space is bounded:

- above by the skin and mentalis muscles
- laterally by the anterior bellies of the digastric muscles
- deeply by the mylohyoid muscle
- superiorly by the deep cervical fascia, the platysma muscle, the superficial fascia and the skin

If infection from the incisors exits labially through the mandibular bone, inferior to the muscle attachments, there is a distinct, firm swelling in the submental region. The swollen region is board-like and stiff until suppuration occurs and fluctuation is palpable. The skin over the swelling is taut and red and the patient experiences considerable pain and discomfort
FIGURE 34. Diagrammatic representation of Ludwig's Angina

On the left the normal side, on the right the situation (bilaterally) in Ludwig's Angina. The tongue:

a. is raised by the volume of exudate
b. the cleft between hyoglossus and genioglossus for the lingual and sublingual arteries and veins
c. the sublingual space
d. the submandibular space

Infection in cleft b has direct access to the laryngeal regions and in the sublingual space through to the pterygoid and lateral pharyngeal spaces
FIGURE 35. Diagrammatic sketch of a sagittal section through the tongue and related structures to illustrate the anatomical pathology occurring in Ludwig's Angina.

The progressive cellulitis of the floor of the mouth and oedema of the tongue produces pressure in the directions indicated by the arrows. The oral and pharyngeal cavities are thus greatly encroached upon or even entirely obliterated. The epiglottis may be so tilted posteriorly which makes intubation of the larynx and trachea very difficult.
PLATE 25. This facial view demonstrates Ludwig's Angina arising from a carious lower left third molar tooth.
PLATE 26. This facial view of Ludwig's Angina demonstrates the displacement of the tongue due to swelling of both sublingual spaces.
PLATE 27. Ludwig's Angina (lateral view).
PLATE 28. Ludwig's Angina (frontal view).
FIGURE 36A. A diagram of the submental space from below
a. mandible; b. anterior belly of the digastric;
c. submental lymph nodes; d. mylohyoid muscle;
e. hyoid bone

FIGURE 36B. Submental space infection
when swallowing. As the infection progresses the patient may have difficulty breathing because of the route of infection between the genioglossus and geniohyoid muscle posteroinferiorly to the region of the epiglottis where a mass appears submucosally. Surgical drainage of the space is best accomplished percutaneously via a horizontal incision in the most inferior portion of the chin providing dependent drainage.

6.5.6 Pharyngeal Spaces

The lateral pharyngeal space is a lateral neck space whose shape is that of an inverted cone with its base at the skull and its apex at the hyoid bone. Infections of the lateral pharyngeal space may result from pharyngitis, tonsillitis, parotitis, otitis, mastoiditis and odontogenic infection, especially if the masticator space is primarily infected. This space occupies a unique position among the fascial spaces in the head and neck in that it is directly adjacent to seven potential spaces. These include the sublingual space anteriorly, the parotid compartment posterolaterally, the carotid sheath contents posterocentrally, the retropharyngeal cleft posteromedially, the masticator space laterally, the space of the palatal muscles medially and the submandibular space inferiorly. The significance of the involvement of the lateral pharyngeal space in deep neck infections was described earlier by authors including Mosher (1929), Shapiro (1930), Beck (1933), Hall (1939), and Coller (1937). More recently, clinical reviews and case presentations have been described by Danforth (1963), Spilka (1936), Laskin (1964), Langenbrunner (1971), Levitt (1971), Matucci (1974), Paonessa (1976), Johnson (1976) and Dzyak (1984).

The lateral pharyngeal space, also known as the pharyngomaxillary or parapharyngeal space (Figure 37), has the following boundaries:
- the anterior limit is the pterygomandibular raphe
- the posterior limit extends to the prevertebral fascia and communicates
FIGURE 37. Diagrammatic oblique section through the neck showing boundaries, compartments and important structures of the lateral pharyngeal space.
with the retropharyngeal space
- the medial wall is the buccopharyngeal fascia on the lateral surface of the superior constrictor muscle
- the lateral wall includes the fascia covering the medial pterygoid muscle (superficial layer of the deep cervical fascia), the parotid gland and the mandible

The styloid process divides the space into an anterior muscular compartment related to the tonsillar fossa which contains fat, muscle, lymph nodes and connective tissue, and a posterior vascular compartment which contains the cranial nerves IX through XII and the carotid sheath and its contents. The symptoms of lateral pharyngeal infections will vary according to whether the anterior or posterior or both compartments are involved. There are four classic signs of involvement of the anterior lateral pharyngeal compartment:
- trismus and pain
- induration and swelling at the angle of the jaw
- fever and chills
- pharyngeal bulging with associated dysphagia

Trismus is the most important of these signs. Pharyngeal bulging is due to the thin fasciomuscular wall between the pharynx and the compartment. Because of the relation to the tonsillar wall, there is usually bulging of the posterolateral pharyngeal wall and tonsillar region as well. There may be neurologic involvement of cranial nerves IX through XII and of the cervical sympathetic chain with ipsilateral Horner’s syndrome. In infections limited to the posterior compartment the muscles of mastication are not involved and there is an absence of facial swelling and trismus. However, intra-oral examination will reveal swelling in the tonsillar region.

The treatment of parapharyngeal infection requires immediate hospitalisation, maintenance of airway, incision and drainage, removal
of the causative agent and antibiotic therapy. Incision and drainage is most important to decompress the lateral pharyngeal spaces and to avoid the dreaded complications of airway embarrassment, sepsis and major vessel erosion. The approaches to the lateral pharyngeal space are:
- intra-oral either transpharyngeal or lateral
- extra-oral
- combination of both

The transpharyngeal approach is made through the tonsillar fossa while the lateral approach is most easily performed by making an incision lateral to the pterygomandibular raphe. However, the extra-oral submandibular incision is the safest approach. In the combined intra-oral and extra-oral approach a large curved clamp is passed medial to the medial pterygoid muscle and posteroinferiorly below the angle of the mandible. When the tip of the instrument is palpated extra-orally anterior to the sternomastoid, a cutaneous incision is made over the tip.

The retropharyngeal space is the posterior space lying behind the oesophagus and pharynx and extending from the base of the skull to the mediastinum. Posteriorly lies the "danger space" which communicates with the posterior mediastinum and deeper still lies the prevertebral space which extends the entire length of the vertebral column.

Clinically, retropharyngeal space infections may result from nasal and pharyngeal infections, dental infection diffusing through contiguous spaces, oesophageal trauma or foreign bodies and tuberculosis. Infections of the retropharyngeal space are characterised by dysphagia, dyspnoea, maxillary rigidity, oesophageal regurgitation and fever. If the pharynx can be visualised, a bulging of the posterior pharyngeal wall may be observed.

Surgical drainage of retropharyngeal infection is required promptly, usually with tracheostomy in the first instance to avoid aspiration or airway
obstruction. In the transoral approach, an incision is made through the posterior pharyngeal mucosa and the abscess is opened by blunt dissection. The extra-oral approach is via an incision parallel to the anterior border of the sternomastoid muscle inferior to the hyoid bone. The muscle and the carotid sheath are retracted laterally and blunt dissection is carried deeply to the level of the hypopharynx. Dissection deep to the carotid sheath between it and the inferior constrictor muscles opens the retropharyngeal space abscess.

6.6 Conclusion

Though the morbidity and mortality of odontogenic infections has declined since the advent of antibiotic therapy they remain potentially harmful or lethal if not recognised and treated aggressively. Clinicians should not dismiss odontogenic infections as simple abscesses and should remember that any oral or opportunistic organism may be the responsible pathogen. When deep fascial space infections develop, maintenance of a patent airway, intense antibiotic therapy and vigorous surgical intervention are imperative.
CHAPTER 7

AIRWAY MANAGEMENT
7.1 Introduction

Airway management in patients with potential respiratory obstruction due to orofacial infections is of prime importance. When the patient must sit bolt upright to preserve the airway, is unable to extend the neck, and has severe trismus with orofacial and cervical tissues grossly distorted by swelling, conservative methods of airway control have severe limitations. Intubation either conventionally or with the aid of a fibreoptic bronchoscope or by blind endotracheal intubation may be difficult. Cricothyroidotomy and tracheostomy both necessitate access to the upper part of the trachea and are normally undertaken in a supine position with the neck extended. This position and access to the trachea in such patients can only be obtained if a decision is taken to sacrifice the remaining airway by forcing the patient into a supine position. Emergency tracheostomy would then be performed without adequate anaesthesia on an anxious patient in a state of hypoxia. The cervical swelling also distorts the surgical anatomy and the chances of a successful outcome are very poor. Transtracheal ventilation has been described, although its use in the management of orofacial infection has not been reported.

The patency of the airway is of primary concern and if inadequate, requires immediate attention, preceding all other diagnostic and therapeutic considerations. The signs of airway function should be monitored closely. The degree of stridor should be noted. Noisy respirations indicate airway obstruction. Snoring, which may be considered a nuisance in a healthy person, signifies in an unconscious person a serious degree of airway obstruction by the soft tissues above the larynx. Observation of the patient’s chest may reveal a forceful diaphragmatic action attempting to overcome the obstruction. The patient’s ability to swallow and to cough should be observed, as an inability to cough may lead to an accumulation of
mucus in the trachea. Frequent chest auscultation will reveal the presence of stagnating mucus in the tracheobronchial tree and detect bronchial obstruction. The patient's tidal volume and respiratory rate should be recorded. An assessment of the inspiratory force against an obstruction i.e. the work of breathing, will provide important information and will complement an evaluation of the degree of fatigue or physical exhaustion. It should be remembered that the patient can be in considerable distress with an upper airway obstruction and yet maintain normal blood gases. Oedema of the larynx may further imperil the patency of the respiratory passage and the patient may experience great difficulty in talking. Respirations become shallow, their tone becomes flat and the rate is accelerated. Cyanosis, if noted, is a late sign. Initially, the patient may not complain of respiratory difficulty, particularly if the onset of respiratory obstruction is gradual, that is, over a period of hours. In this case, the patient may not be aware of respiratory obstruction until it becomes severe. Alternatively the patient may be too tired to complain. Therefore, meticulous attention to clinical signs of airway problems makes it possible to anticipate complications and taking steps towards their prevention should be the main aim in management (Patterson 1982, Loughman 1985).

7.2 **Mechanisms to protect the airway (Spoerel 1972)**

a. **Mechanisms that maintain patency of the supralaryngeal airway**

The tone of the group of muscles attached to the hyoid bone and supporting the base of the tongue is largely responsible for the patency of this portion of the airway. The mildest form of depression may occur during natural sleep in a person with a tendency to snore, when the reduced muscle tone allows vibration of the soft palate. In the supine position the bulk of the tongue will slide backward and occlude the pharyngeal portion of the airway. If there is a large soft tissue swelling involving the oropharyngeal
passage then it is difficult to establish and maintain a patent airway. Airway support or pharyngeal airways may prove ineffective and emergency intubation may also be difficult in these circumstances.

b. **Pharyngeal and laryngeal reflexes protecting the entrance to the trachea**

The pharynx is the common pathway for air, fluid and solid material. The respiratory passage is maintained continuously while the oro-cesophageal passage is established intermittently by the swallowing reflex and, in the reverse direction, by the mechanism of retching. During these intervals the airway below the larynx is protected by reflex closure of the glottis. Swallowing is a very complex reflex involving the muscles of the tongue, soft palate, pharynx and oesophagus. The presence of infection in the orofacial region may inhibit the swallowing reflex and the patient may attempt to expel saliva from the mouth by spitting or drooling. Accumulation of saliva in the oral cavity will allow the bacterial flora in the mouth to rapidly multiply and some of this accumulated material can find access to the trachea and bronchi, thus providing a source of bacterial contamination of the airway.

c. **Cleansing mechanisms of the upper airway**

In normal health the trachea and bronchi are sterile. In the nasal cavity and tracheobronchial tree the ciliary apparatus provides a continuous cleansing mechanism which removes inhaled material and prevents stagnation of secretions that could provide a nidus for bacterial growth. In addition, there is intermittent reflex clearing of the passages in the form of sneezing and coughing. If swallowing is impaired, mucus discharged into the pharynx will accumulate allowing stagnation and infection, including pulmonary infection to occur.

d. **Mechanisms that condition the inspired air**

The major exchange of heat and water vapour takes place in the nose, ensuring that the air entering the trachea is near body temperature and
almost completely saturated with water vapour. When the supralaryngeal airway is by-passed by an endotracheal tube or a tracheostomy, there is a need for efficient humidification to prevent drying of the tracheal mucous membrane.

7.3 Literature Review of the Anaesthetic Management of Ludwig’s Angina

Early reports of orofacial infections described tracheostomy being performed under local anaesthesia, often with great difficulty. The advent of endotracheal intubation in the 1930s and the development of intravenous anaesthetic agents were significant factors in the advance of general anaesthesia and management of the airway (Magill 1936).

Beck (1934) considered that when there was actual dyspnoea a general anaesthetic should not be administered until the airway was under control. When the encroachment was limited to the tongue and pharyngeal region, it could be corrected by the insertion of a pharyngeal breathing tube, at which time the administration of a general anaesthetic was considered safe. However, where the dyspnoea was caused by oedema of the larynx or by pathology in the lower respiratory tract, Beck considered that a general anaesthetic should not be administered until a tracheostomy had been performed.

Beck used a local anaesthetic in deep neck infections when he considered that a general anaesthetic was hazardous. He believed that a tracheostomy should always be performed under a local anaesthetic or even without anaesthetic. When there was extensive inflammation present, it was difficult to obtain anything but anaesthesia of the skin since deep insertion of the injecting needle into the swollen, infiltrated areas could carry infection into uninvolved tissues. Ideal anaesthesia could be achieved by paravertebral injection into the cervical nerves at their points of exit from the cervical column. This required a posterior approach through thick muscle
layers and a high degree of skill.

Williams and Marcus (1941) reviewed the anaesthetic management of 25 patients with Ludwig's Angina, noting that if the patient exhibited any of the warning signs of imminent obstruction then the surgeon should have the tracheostomy set ready. He should even prepare for the operation by dividing all structures so as to expose the tracheal cartilages. The anaesthetic methods used included:

- local anaesthesia in 2 patients
- inhalation anaesthesia in 13 patients
- rectal anaesthesia in 1 patient
- intravenous anaesthesia in 8 patients
- combined local and inhalation anaesthesia in 1 patient

It was concluded that the proper operative procedure in Ludwig's Angina was too extensive to be attempted under local anaesthetic. The two patients in this series in whom local anaesthetic was used both died because only small incisions were made and inadequate drainage obtained. The authors found that all of the inhalation agents produced some degree of spasm of the airway, as evidenced by coughing and gagging during induction. In Ludwig's Angina, the airway which is greatly encroached upon, is more susceptible to spasm and, therefore, respiratory obstruction of sufficient degree to require tracheostomy may occur. In this study of the 13 patients receiving inhalation anaesthetics, the outcome for 7 was uneventful, 5 required emergency tracheostomy and in 1 patient, partial obstruction was relieved by surgical drainage of the suprahyoid region. Intratracheal intubation was attempted in 2 patients without success. The authors believed that intubation was difficult in the great majority of patients with Ludwig's Angina because of the anatomical distortion peculiar to this disease. Rectal anaesthesia with Avertin was used successfully in one patient who had no
respiratory embarrassment pre-operatively. The combination of local infiltration and nitrous oxide had the advantage of postponing the use of nitrous oxide until the deepest structures were being incised but the danger of spastic obstruction was not avoided by this method. The intravenous barbiturates, Evipal and Pentothal, administered fractionally were considered to offer the best advantages. Induction could be smooth and the depth of anaesthesia could be easily controlled. Should tracheostomy become essential, an even level of anaesthesia could be maintained throughout the procedure. The disadvantages were that they depressed the respiratory centre, produced shallower respirations and could produce spasm of the airway. The authors felt that intravenous anaesthesia was safest for the patient and most convenient for the surgeon and anaesthetist. However, preparation for tracheostomy was stressed as a life saving measure, regardless of the type of anaesthesia employed.

Bennett (1943) introduced the technique of blind nasotracheal intubation under topical anaesthesia using a 10% aqueous solution of cocaine. This was considered particularly useful for the management of drainage of abscesses of the submandibular space. He presented 6 patients in which respiratory obstruction was severe.

The rapidity with which orofacial infection may be manifested was noted by Kalman et al (1953). Their patient showed toxic symptoms 12 hours after the extraction of a tooth associated with pericoronitis, and within 24 hours required a tracheostomy. It was thought that tracheostomy, if not unduly delayed, could be achieved through essentially uninvolved tissue planes, since the swelling would be limited initially to the suprahyoid region.

There is no doubt that management of the airway by either endotracheal intubation or tracheostomy for patients with severe orofacial infections is potentially life-saving. However, death from unknown causes may occur during
the anaesthetic phase before any surgical intervention has been carried out (Kelly et al, 1957). Numerous theories have been offered but the causes most commonly considered were:

1) carotid body stimulation
2) vasovagal reflex
3) trauma to the larynx during the introduction of an endotracheal tube
4) laryngeal obstruction due to the infectious disease.

Though an endotracheal tube would seem to be the answer, laryngeal oedema with the often accompanying trismus of the jaw can prevent insertion of the endotracheal tube into the larynx, and repeated unsuccessful attempts to do so may precipitate a fatal laryngospasm. The infectious process may also have impinged on the laryngeal vestibule and glottis. The swollen raised tongue which is then pushed upward and backward by the disease process tends in time to force the already oedematous epiglottis further back, thus narrowing the airway to an even more dangerous degree. In fact the mere manipulation of the head during laryngoscopy attempts has been known to lead to sudden deaths in patients with deep neck infections.

Kelly et al considered that early tracheostomy was the key to proper management of Ludwig's Angina and was the answer to avoiding deaths from acute laryngeal obstruction. Recognising that patients were becoming sensitised to antibiotics and that bacterial resistance to antibiotics was becoming more common, the danger of waiting for spontaneous rupture of such an infection was increased if surgical drainage was not employed early in conjunction with antibiotics. Thus the safest and best method of treating Ludwig's Angina was considered to be a combination of:

a) early tracheostomy with local anaesthesia

b) complete drainage with release of tension of the fascial planes under general anaesthesia
c) adjunctive employment of antibiotics

Intubation with a nasoendotracheal tube for a patient with gross orofacial and cervical swelling can be accomplished with the patient awake (Marks 1975). In this case, a surgical team was on standby prepared to do an emergency tracheostomy if the intubation failed. General anaesthesia was then administered and the surgery performed. Marks noted that most deaths from Ludwig's Angina were the result of mechanical obstruction. The restoration of the occluded airway takes priority over all other considerations (Meyers 1972). Respiratory distress can develop rapidly and abruptly and may lead to sudden respiratory arrest.

Holland (1975) presented a patient with Ludwig's Angina who was managed by early surgical intervention in an attempt to relieve the imminent respiratory obstruction. However, the surgery was inadequate and did not produce resolution of the facial infection nor decompression of the airway. The patient remained intubated post-operatively and the development of a chest infection was attributed to the inhalation of infected material from the mouth which then necessitated a tracheostomy. It was realised that tracheostomy was a more logical approach to the problem of respiratory obstruction in Ludwig's Angina and in addition laryngeal reflexes could be maintained as a barrier to inhalation of infected material. In retrospect, the tracheostomy should have been part of the primary surgical procedure. Holland advocated the use of local analgesia for the tracheostomy, but in this report, the patient experienced total respiratory obstruction unless sitting in the upright position. Therefore, in the severe form of Ludwig's Angina it is desirable to pass an endotracheal tube initially to establish an airway in the supine position. This should be done with the patient conscious because induction of general anaesthesia abolishes voluntary control of the airway which may be difficult to re-establish. Once the
airway is secure, attention can be focussed on the second aim of management, the control of infection. Holland concluded that in less severe infections where there is no immediate respiratory problem and the infection is not localised, the initial management should be intensive antibiotic therapy and careful observation to recognize developing respiratory obstruction.

The prevention of supraglottic airway obstruction is critical (Burtner et al 1978). With certain pathological conditions, the unobstructed airway in the conscious patient becomes impossible to secure and to maintain once general anaesthesia commences. If a potential or supraglottic obstruction is evident then a safe technique should be selected to secure and maintain the airway. Burtner et al presented four patients of which two had neck infections and in whom potential or actual airway obstruction became critical at the time of administration of general anaesthesia. One patient underwent awake nasal endotracheal intubation with local anaesthesia, and once the airway was secure, surgery was undertaken without precipitating a hasty tracheostomy. The decision to use a general anaesthetic on the second patient appeared to precipitate an airway obstruction. This patient exhibited airway obstruction that was not evident on initial examination, but became apparent only when the patient lost control of his airway on induction with relaxation of the voluntary muscles of the pharynx and larynx. Thus, the danger of paralysing a patient with potential upper airway obstruction during anaesthesia is evident.

Anaesthesia introduces several intrinsic factors that lead to upper airway obstruction which include:
- the relaxation of the voluntary pharyngeal-laryngeal muscles during induction of anaesthesia with the use of muscle relaxants;
- manipulation required for exposure of the larynx and placement of the endotracheal tube which can produce tissue oedema, haematoma, bleeding and
increased secretions. Therefore, the potential for complete airway obstruction becomes a reality if intubation is prolonged and repeated attempts at intubation are made;
- oesophageal reflux secondary to muscle relaxation which can block the airway and can be aspirated. This may limit the ability to see the larynx and make intubation difficult and hazardous and may lead to Mendlesohn’s syndrome;
- impaction of the epiglottis into the glottis, especially with a long epiglottis causing complete obstruction of the airway and the glottis. The insertion of an oral airway to improve a partial obstruction can push the epiglottis into the superior aperture of the glottis, thereby precipitating laryngospasm.

Burtner et al concluded that there were 5 options available in the anaesthetic management of patients with potential upper airway obstruction which depended on:

1. the assessment of the patient’s preoperative ability to maintain the patency of his or her airway
2. the patient’s suitability for intubation and general anaesthesia
3. the nature of the obstruction as seen by radiography and indirect laryngoscopy

The options available are:

1. **Awake oral-tracheal intubation**
   Proper mobility of the neck and good oral opening in a cooperative patient is required together with an ability to overcome the gag reflex.

2. **Awake nasotracheal intubation**
   Blind, awake nasotracheal intubation with the patient under local anaesthesia can be performed by listening to breathing sounds through the tube as it is passed from the nose to the supraglottic area and
guided into the trachea. A flexible fibreoptic bronchoscope is useful in this technique.

3. **General anaesthesia with ventilation**

Inhalation induction of general anaesthetic with spontaneous ventilation of the patient, using halothane and 100% oxygen without muscle relaxants but with the use of topical local anaesthesia on the vocal cords. The patient retains muscle tone and the airway remains patent prior to intubation. This method can only be used when the patient has normal neck mobility and reasonable jaw opening.

4. **Direct laryngoscopy and intubation**

Preoxygenation with 100% oxygen followed by rapid induction with thiopentone and suxamethonium for direct laryngoscopy and intubation. If intubation is not successful there are grave consequences if no alternative airway can be established.

5. **Tracheostomy**

This can be performed with the patient under local anaesthetic or general anaesthetic. There is a reluctance to perform tracheostomy with the patient under local anaesthesia when endotracheal intubation can be accomplished. This reluctance stems from a forbidding list of complications from a rapidly performed tracheostomy which includes:

- major haemorrhage
- injury to the cricothyroid complex
- penetration of the posterior tracheal wall
- pneumothorax if the lung apices ride high
- varying degrees of subcutaneous and mediastinal emphysema which may develop once the pretracheal fascia is cut
- problems of supraglottic and tracheal collapse

However, the primary advantage of tracheostomy is immediate control of
upper airway obstruction in a patient who cannot be safely intubated.

Burtner et al concluded that the administration of general anaesthesia worsens and, in many cases, precipitates upper airway obstruction. If this is not corrected either by termination of the induction process or by intubation or by tracheostomy, it will lead to anoxia and cardiac arrest. The anaesthetist must be certain of intubation in a patient with potential obstruction before giving a muscle relaxant. Ideally, the potentially obstructed patient should have his or her airway established while he or she still maintains control of the voluntary muscles of the pharynx and larynx.

Allen et al (1985) reviewed 10 patients with Ludwig's Angina and reported that only one patient required a tracheostomy. They found that deep anaesthesia without narcotic premedication and without muscle relaxants would relieve pain and muscle spasm allowing the mouth to be opened and the vocal cords to be visualised with a laryngoscope. Intubation could then be performed under direct vision. If, during this technique airway obstruction occurred, a nasopharyngeal airway was inserted since it was thought that the most likely cause of obstruction was superoposterior displacement of the tongue. However, the tracheostomy set was always in readiness. The authors believed that awake blind nasal intubation was a difficult technique best suited to an experienced anaesthetist. However, it could precipitate obstruction in patients with Ludwig's Angina or inadvertently rupture lateral pharyngeal or retropharyngeal abscesses. Fibreoptic bronchoscopy was considered a technique that also required an experienced and skilled anaesthetist and a cooperative, stable, premedicated patient. Barsamian (1987) reported on a patient who presented with Ludwig's Angina and a right pneumothorax. Attempts to intubate the patient with the fibreoptic unit were unsuccessful so an emergency cricothyrotomy was performed. This maintained an adequate airway while the tracheostomy was performed.
Assessment of the soft tissue swelling and the airway can be made with the use of the lateral radiograph of the neck. This is a useful guide in evaluating the bony and soft tissue relationships of the neck. The radiographs are taken with the patient sitting erect and with the neck in the neutral position. Wholey et al (1958) reviewed 600 lateral cervical radiographs taken at the Mayo Clinic and established normal values and variations for sagittal measurements of the retropharyngeal and retrotracheal spaces. The retropharyngeal space was measured from the antero-inferior aspect of the second cervical vertebra to the posterior wall of the pharynx, while the retrotracheal space was measured from the antero-inferior aspect of the sixth cervical vertebra to the posterior aspect of the trachea. They suggested that measurements of the retropharyngeal space greater than 7 mm in both children and adults and measurements of the retrotracheal space greater than 14 mm in children and 22 mm in adults, warranted further investigation to exclude the possibility of a pathologic process. Any localised irregularity of the posterior tracheal wall should be recorded as significant until proven otherwise. Such localised bulges may be due to benign or malignant neoplasms, haematomas or inflammation.

Assael et al (1986) presented three patients with impending airway obstruction subsequent to head and neck infection and described the usefulness of soft tissue radiographs of the region in determining the location of the problem. Airway obstruction may occur by enlargement of the lingual, parapharyngeal or retropharyngeal spaces. It may also be a consequence of epiglottic, endotracheal or lower airway obstruction.

Knowledge of the site of respiratory obstruction is essential if the airway is to be restored effectively and without complication. If airway obstruction is limited to the upper airway then it can be managed by oro- or naso-pharyngeal airway and subsequent endotracheal intubation. However,
airway obstruction due to endotracheal or laryngeal oedema is managed by tracheostomy.

In conclusion, the techniques for establishing an airway include:
- positioning of the patient
- oro- and naso-pharyngeal airways
- trans-tracheal ventilation
- endotracheal intubation with or without the use of the fibreoptic bronchoscope under local or general anaesthesia
- tracheostomy

7.4 Trans-Tracheal Ventilation

A technique which may be of use in an emergency situation is Trans-Tracheal Resuscitation which involves the intermittent administration of oxygen at 60 lbs p.s.i. through a 14 gauge intravenous cannula inserted at an angle of 45° into the trachea (Jacoby et al 1956). When, after respiratory obstruction, all commonly applied methods of providing a clear airway have not been successful, or are unable to be accomplished, the insertion of an oxygen carrying needle into the tracheal lumen may prevent a respiratory death. Oxygen saturation of the arterial blood decreased to the point of cyanosis, may be reversed to normal or above within minutes after application of this technique and can be maintained for 30 minutes or more until the obstruction is alleviated. The materials necessary for transtracheal resuscitation consist of a 14 gauge needle, a 10 cc syringe, a source of oxygen and an adaptor to attach the oxygen supply to the needle.

The insertion of the transtracheal needle is accomplished simply and rapidly. The patient's neck is extended. The location for the needle insertion is the cricothyroid membrane. The trachea is steadied between the index finger and thumb of one hand; the needle, attached to an empty syringe, is thrust through the skin at the midline, in a posterior and inferior
direction. Traction is made on the plunger of the syringe. When the needle enters the trachea the operator obtains a sensation of release of resistance. At the same time, air is aspirated freely into the syringe. The plunger of the syringe is then moved up and down several times to be certain that air moves without resistance in both directions. The syringe is detached, and the oxygen supply is then connected to the needle.

If the point of the needle is not located within the lumen of the trachea, subcutaneous emphysema could occur and if the point of the needle was to enter a blood vessel, the possibility of air embolism also exists. The administration of oxygen should not be commenced unless the operator is certain that the point of the needle is within the lumen of the trachea. An additional complication might be the perforation of other structures by the needle if it were improperly placed.

It is not recommended that transtracheal resuscitation be used as a substitute for endotracheal intubation or tracheostomy, but it can be of value for a relatively brief period for the prevention of death from anoxia.

The technique has also been described by Spoerel et al (1971) who used it for procedures and operations on the upper airway and larynx. Smith et al (1975) also reported on the technique for both elective patients who had carcinoma of the larynx or tongue and for resuscitation of patients with acute respiratory distress. Layman (1983) used the technique in Nigeria as a routine method of ventilation for patients undergoing surgery with gross facial pathology following cancer of the oral cavity.

7.5 Endotracheal Intubation with and without the use of the Fibreoptic Bronchoscope under Local or General Anaesthesia

Endotracheal intubation in the presence of swelling of the neck, glottic oedema, elevation of the tongue, trismus and pharyngeal oedema is a formidable anaesthetic problem. The trismus constitutes a technical problem
for the anaesthetist. Acute trismus of recent origin will usually relax under general anaesthetic, but limitation of jaw opening of longer standing may fail to relax with a general anaesthetic, even when a muscle relaxant is administered.

Spread of inflammatory swelling into the floor of the mouth and base of tongue, or into the region of the glottic aperture or soft palate, may limit access and obliterate normal anatomy, making laryngoscopy and endotracheal intubation impossible. Laryngoscopy may also cause rupture of the abscess into the mouth or pharynx with serious risk of aspiration of pus into the air passages. In the management of these patients the anaesthetist must be aware of the inherent dangers and determine the history and extent of the infective process. A decision should be made whether the intubation will be carried out with the patient awake or anaesthetised. In either situation, premedication with narcotics should be avoided. If the patient is to be anaesthetised, induction can be accomplished with an intravenous agent or with an inhalational technique. The danger in using an intravenous technique is loss of motor tone with aggravation of the compromised airway and resultant inability to ventilate or intubate the patient.

The advantage of deep inhalational anaesthesia is that trismus may be relieved and the mouth opened sufficiently to permit passage of a laryngoscope and assessment of the possibility of intubation. Intubation can then be performed under direct vision with or without the use of muscle relaxants. Alternatively, vision of the larynx may not be achieved but blind nasal intubation may be skilfully and successfully carried out by an anaesthetist experienced in this technique (Gordon 1972). Blind awake nasal intubation with a co-operative patient under local anaesthesia is much easier and much more likely to be successful than blind nasal intubation under general anaesthesia for two main reasons:
1. When asleep there is a loss of motor tone which a) would normally have guided the endotracheal tube along the airway and into the larynx and b) might result in loss of the patency of the airway, possibly resulting in obstruction.

2. When awake the patient can co-operate and can be asked to "breath the tube into his windpipe" as the anaesthetist advances the tube. The patient's active co-operation and control of motor tone makes this a highly successful manoeuvre.

In 1973 Davis described a fiberoptic laryngoscope for nasal intubation, which allowed intubation to be performed under local anaesthesia. Shwartz et al (1974) reported on the use of fiberoptic laryngoscopy to avoid tracheostomy in a patient with Ludwig's Angina. The patient was maintained in the seated position so that total obstruction did not occur and following the application of local anaesthetic to his nose and nasopharynx the fiberoptic laryngoscope was placed through an endotracheal tube and then passed easily under vision into the trachea. The endotracheal tube was then slid over the laryngoscope and into the trachea. The laryngoscope was then withdrawn. With the nasoendotracheal tube in place and the airway secure the patient could be anaesthetised. The authors considered that intubation could be performed with minimal stress and discomfort in the awake patient who had adequate topical anaesthesia.

Sutherland et al (1986) presented three patients with oropharyngeal sepsis where, after application of nebulised lidocaine, awake fiberoptic intubation using the Williams oral airway intubator and fiberoptic bronchoscope was used. Both the fiberoptic laryngoscope and bronchoscope can be used through the mouth or nose. However, the fiberoptic bronchoscope gives much better vision and suction than does the fiberoptic laryngoscope and so is the preferred instrument.
7.6 Tracheostomy

This technique is now not considered the method of choice to gain control of the airway in patients with orofacial infections, though it may be required in the continuing management of such patients.

"Quick tracheotomy" (Oppenheimer 1966) is a simple and reliable method of establishing a temporary airway until an orderly tracheostomy can be carried out. This procedure of coniotomy or cricothyrotomy (Figure 38) is the cutting of the cricothyroid membrane (Greenway 1972, Brantigan 1976, Feinberg 1987). It requires no special instruments and little knowledge of anatomy, and it can be done anywhere under almost any conditions. The cricothyroid membrane lies near the surface and can be easily palpated irrespective of how fat the neck may be, and there are no vital structures superficial to it. The only instrument necessary for cricothyrotomy is a knife, though a scalpel is preferable.

An elective tracheostomy under either local or general anaesthesia, is done via a 4 to 5 cm incision transversely or vertically positioned about 2 cm above the suprasternal notch. There is some reluctance to perform tracheostomy now that the safety and efficacy of prolonged nasotracheal intubation has been established. The term prolonged intubation has been used to designate durations of eight hours to several months. Although one to two weeks is generally considered safe, no discrete time limit has been defined beyond which complications seem to increase significantly. Whether a tracheostomy or a nasoendotracheal tube is the better choice remains controversial. With the use of high-volume, low-pressure cuffs, both routes of intubation have similar low rates of cuff induced complications (Bishop et al 1984). Suggestions to decrease laryngeal injury include the use of nasal intubation as nasotracheal tubes are generally smaller and therefore cause less injury, and the use of swivel connectors that allow movement of
By moving the finger, pressing slightly, from the prominentia laryngea downward to the dip marking the thyroid-cricoid interspace, the point for insertion of the knife into the trachea can be easily identified.

The knife enters a relatively safe area well above the thyroid gland and the blood vessels that make the usual site of tracheotomy hazardous. The posterior aspect of the cricoid cartilage prevents thrusting the tip of the knife too deep.

Front view showing proper insertion of knife blade and the position of thyroid gland and major blood vessels well below incision site.
the patient and the tube as one unit. It is thought that motion of the tube in the larynx may contribute to injury. However, the final decision in patients with orofacial infections is dependent on the clinical status of the patient and the findings of direct laryngoscopy.

7.7 Conclusion

Patients with severe orofacial infections can experience rapid swelling of the facial and cervical tissues with consequent respiratory obstruction. Initial management must include an assessment of the airway and a decision as to the manner in which it can be best maintained in the pre-operative, intra-operative and post-operative periods.
CHAPTER 8

COMPLICATIONS OF OROFACIAL INFECTIONS
8.1 **Introduction**

It is a cardinal surgical principle that an abscess be drained as soon as possible and the drainage be maintained until the abscess becomes dry. In dentistry, this principle has been set aside because of the fear of complications. However, studies have shown that extensive infections and complications are more likely to be due to delayed surgical intervention. It should be emphasised that the extraction of teeth in patients with acute suppurative infection treats the primary dentoalveolar infection and, consequently, prevents the development of fascial space abscesses. In the late stage of infection the extraction of the offending tooth does not effect a cure because the acute infection has already migrated to an adjacent fascial space.

Major complications of orofacial infections include:

- Cavernous Sinus Thrombosis
- Brain Abscess
- Orbital Spread of Odontogenic Infections
- Mediastinitis
- Empyema
- Vascular Complications
- Necrotising Fasciitis
- Ludwig’s Angina as a Complication
- Complications of Orofacial Infection in Children

8.2 **Cavernous Sinus Thrombosis**

In the years prior to the discovery of antibiotics, cavernous sinus thrombosis was a complication of odontogenic infections with almost a 100% mortality rate. Penicillin and other antibiotics have successfully resolved this very serious infection and now the development of cavernous sinus thrombosis from an odontogenic infection is a rarity (Clune 1963, Palmersheim
The cavernous sinuses lie between the layers of the dura mater, one on each side of the sella turcica and extend from the superior orbital fissure to the apex of the petrous temporal bone. Among its tributaries the cavernous sinus receives the superior opthalmic vein and a branch from the inferior opthalmic vein. A feature of the veins draining into the sinus is that they are devoid of valves (Figure 39 and 40).

The term "cavernous sinus thrombosis" includes cases of phlebitis, thrombophlebitis and phlebothrombosis as well as true aseptic thrombosis. By far the majority of case reports are of septic origin and would therefore be classified as phlebitic or thrombophlebitic. The commonest route of spread of infection results from suppurations of the upper lip, ala nasi, vestibule of the nose and eyelids by way of the angular, supraorbital and supratrochlear veins to the opthalmic veins. The organisms most frequently associated with cavernous sinus thrombosis are streptococci and staphylococci species. The spread of the disease from the primary focus may be by a process of infective thrombophlebitis so that a cord of thrombus extends from the lesion to the sinus, or by a 'discontinuous' form in which the primary site and the sinus are not connected by a continuous clot. A third possibility is that the infection reaches the sinus by a process of septic venous embolism. Whichever the mode of spread, when the condition has become established on one side, it almost invariably spreads along the intercavernous sinuses to involve the opposite cavernous sinus. Septicaemia and meningitis may also follow cavernous sinus thrombosis while brain abscesses and pulmonary embolism are frequent complications (Shaw 1952).

Septic thrombosis of the cavernous sinus was first described with autopsy findings by Duncan (1821) and the first clinical reports were made by Bright (1831) and Vigla (1839). Yarrington (1961) reviewed the
FIGURE 39. Transverse section of the "classical" cavernous sinus

CS Cavernous Sinus          PP Pterygoid Plexus
1. inferior cerebral vein   2. superior petrosal sinus
3. inferior petrosal sinus  4. emissary veins to PP
5. emissary veins to PP     6. inferior ophthalmic vein
7. superior ophthalmic vein 8. sphenoparietal sinus

FIGURE 40. Venous connections of the cavernous sinus

II Nerve

V Nerve

V Nerve Ophthalmic

VI Nerve

V Nerve Maxillary
literature which documented 878 patients with cavernous sinus thrombosis. This series showed a mortality rate of 73% and a morbidity rate among the survivors of 65%. However, Yarrington concluded that any figure arrived at was conservative as many fatalities and undiagnosed patients were probably not reported.

The clinical symptoms of cavernous thrombosis may be classified as:

1. the early or systemic symptoms
2. the intermediate or regional (largely orbital) symptoms
3. terminal or toxicomeningeal symptoms (Childs 1942)

**Systemic symptoms**, which signal invasion of the general circulation once the thrombosis has extended into the venous system, include chills, recurrent spikelike rises in temperature, profuse perspiration, rapid pulse, headache, nausea and vomiting, transitory euphoria followed by mental clouding or delirium, leucocytosis and positive blood cultures.

**Orbital symptoms**, which follow obstruction of the afferent venous channels and involve the adjacent extraocular nerves and the optic nerve, include:

- oedema of the eyelids and conjunctiva
- protusion and fixation of the eyeball
- induration and swelling of the adjacent forehead, nose and cheeks
- proptosis of the eyeball
- distention of the superficial veins of the forehead and face
- hyperaemia of the retina
- papilloedema
- occasionally epistaxis
- pain in the distribution of the ophthalmic and maxillary divisions of the trigeminal nerve
- ophthalmoplegia
- dilation of the pupil
- impairment or loss of vision
- photophobia with lachrymation
- regional pain and tenderness over the eyeball

Terminal picture which is advanced toxaemia in addition to symptoms and signs of meningeal irritation such as neck stiffness and unconsciousness. Death is usually caused by meningitis, brain abscess or generalised sepsis.

The most common pathway of extension into the sinus from dental infections is via the ophthalmic veins and their anastomoses with the facial, angular, infraorbital and inferior palpebral veins. The first reported dental infection being the cause of such a serious disease is thought to be by Lancial (1888). Other references include Lauret (1921), Eagleton (1926), Dixon (1929), Morgenstern (1935), Hyde (1938), Jacobs (1940), Childs (1941), Haymaker (1945), Grant (1952) and Shaw (1952).

The mainstay of treatment of cavernous sinus thrombosis nowadays is a combination of antibiotics effective against gram positive, gram negative and anaerobic organisms (Taylor 1957). In earlier years various methods of surgical treatment were described. Ligation of the carotid artery of the affected side to "put the inflamed sinus at rest" was recommended. Bailey (1928) proposed ligation of the angular vein to prevent the spread of infection through the ophthalmic vein to the cavernous sinus. However, the consensus of opinion was that surgical intervention was largely ineffective.

More recently, Taicher et al (1978) presented a patient with acute neurological symptoms of a cavernous sinus thrombosis due to a minor dental infection. Extraction of the involved tooth and antibiotic therapy led to a rapid resolution of the neurological signs.

Goteiner et al (1982) described a patient who developed cavernous sinus thrombosis and a brain abscess following the removal of periodontally-
involved teeth. The organisms cultured from the brain and the blood were
typical of those found in the mouth. Resolution occurred following intensive
antibiotic therapy, drainage of the cerebral abscess and removal of the
remaining involved teeth.

Palmersheim et al (1982) reported on the development of a cavernous
sinus thrombosis following the removal of third molar teeth. The extensive
nature of the postoperative infection and the development of a fatal disease
was attributed to the patient's lack of compliance to treatment.

Harbour et al (1984) also reported on a previously healthy 29 year old
male patient who died of spread of infection from gingivitis to both
parapharyngeal spaces and to the cavernous sinus. Initial failure to
distinguish the subtle signs of retro-orbital disease from orbital cellulitis
and failure to recognise the possibility of spread of infection from the oral
cavity led to delayed treatment and the ultimate fatal outcome. This
emphasises that odontogenic disease, even of a relatively trivial nature,
should not be overlooked as a potential cause of a lethal infection.

Though a rarity following odontogenic infections, cavernous sinus
thrombosis, as depicted in Plates 29 and 30, needs to be recognised and
managed early to avoid a fatal outcome.

8.3 Brain Abscess

Brain abscesses following dental infections are rare although serious
complications may be found in the frontal or temporal lobes. In the
pre-antibiotic era, infections of the middle ear and mastoid accounted for
the majority of brain abscesses (Kaplan 1976). Infection extends into the
cerebral tissues either via septic thrombophlebitis of the emissary veins
communicating with the cortical veins or by direct extension from osteitis
of the adjacent bone. In 80% of patients the clinical course is usually less
than 2 months, with a range as short as one week or as long as five months.
PLATE 29. This facial view demonstrates the orbital manifestations of a right cheek infection which has the complication of Cavernous Sinus Thrombosis.
PLATE 30. Surgical decompression and drainage of the right orbit of the patient presented in Plate 29.
The patient complains of headache, which may be variable at first but rapidly becomes constant and progressive. Focal neurological deficits are seen, depending on the location of the lesion, presenting in the form of hemiparesis, sensory disturbance of a cortical nature, visual field defects or dysphasias. Lesions of the anterior frontal lobes or the non-dominant temporal lobe may remain "silent" for prolonged periods and only present with evidence of increased intracranial pressure. Seizures may occur in 10%-20% of patients with frontal lobe abscesses. The signs of the primary focus of infection combined with the neurological picture, contribute to the establishment of a diagnosis of brain abscess. Computerised axial tomography (CAT) of the brain is helpful in visualising the abscess and in demonstrating the distortion of the ventricular system. The treatment of brain abscess is surgical. Craniotomy is preferred with excision of the abscess under intensive, high dose, intravenous antibiotic coverage.


Of particular interest are the following reports:

Haymaker (1945) reviewed 28 patients in which fatal intracranial or spinal lesions occurred following dental extractions. Analysis of this group suggested direct extension of the infection to the intracranial cavity as the essential cause of death in 17 patients and spread of infection by way of the general circulation in the remaining 11. In some of the patients there was both direct and haematogenous spread. In this study most of the teeth extracted were molar teeth, particularly third molar teeth.
Henig et al (1978) presented a patient who developed a brain abscess which grew *Streptococcus viridans* in pure culture. The patient had been receiving endodontic treatment for a six month period and this persistent tooth infection was considered the cause of the cerebral abscess.

Ingham et al (1978) discussed two patients whose frontal lobe abscesses were secondary to dental sepsis. Only when clinical and radiological evidence eliminated sinusitus was a dental focus suspected. It was also found that the bacterial flora of the abscesses in both patients were typical of dental infections.

Baddour et al (1979) presented a 53 year old man who was admitted with obscure signs of disease. Oral examination revealed carious and periodontally involved teeth associated with periapical infections. Following removal of all the infected teeth recovery was uneventful until two weeks later, when a CAT scan revealed a lesion in the left frontal lobe. The diagnosis of brain abscess as a sequel to odontogenic infection was confirmed at surgery, after which the patient made a full recovery.

Gallagher et al (1981) presented a patient with a fatal brain abscess following periodontal treatment. It was believed that curettage of the periodontally involved mandibular premolar teeth caused a bacteraemia that localised in the right frontal lobe of the brain.

In conclusion, the intracranial spread of an odontogenic infection, although rare in the antibiotic era, still exists. Most of the venous system of the face communicates in some way with the cavernous sinus and with the whole venous system of the brain so that an infected venous embolus may find its way there. Even more complicated than the venous system is the arterial blood supply to the head and the neck with its many communications between the internal and external carotid arteries, which together with the significant collateral circulation of the head and neck area can move an
arterial infected embolus from the face towards the brain (Zachariades et al 1986). An infection can also extend to the brain through the foramina of the skull, independent of the passing blood vessels as well as through the infra-orbital fissure and the retrobulbar tissues (Plates 31 and 32).

8.4 Orbital Spread of Odontogenic Infections

Orbital cellulitis following dental extractions has been described by a number of authors, including Yates (1978), Gold (1979), Stone (1979), Kaban (1980), Middleton (1980), Pelligrino (1980), Simpson et al (1981), Janakarajah (1985) and Bullock (1985). Organisms from an odontogenic cause may gain entrance to the orbit:

- through local tissue planes
- by haematogenous spread
- by involvement of the paranasal sinuses

Nowadays, with the widespread use of antibiotics the rapid spread of a dental infection to the orbit is rarely seen, but other factors such as the virulence of the organism, the general health of the patient, the lack of recognition of underlying pathology or a poor choice in initial antimicrobial therapy may dispose certain patients to a rapid spread of the infection. This may result in loss of vision and even death. Therefore, prompt diagnosis is imperative so that the appropriate medical and surgical therapy can be instituted (Figure 41).

Odontogenic infections usually travel by the path of least resistance; pus from maxillary teeth may spread to the soft tissues of the cheek then, either by direct spread or by an ascending facial thrombophlebitis, to involve the orbit. Infections of the maxillary molars may also spread posteriorly into the infratemporal and pterygopalatine fossae. From there the infection can spread along the tuberosity of the maxilla and gain access to the orbit through the inferior orbital fissure.
PLATES 31 and 32. These facial and oral views demonstrate a patient following intra-cranial drainage of a right frontal abscess which was associated, microbiologically, with multiple maxillary carious teeth.
FIGURE 41. Diagrammatic representation of path of spread of odontogenic infection from maxillary premolar and molar teeth
It should be remembered, however, that pansinusitis is the most common cause of orbital cellulitis. In the adult, the orbit is surrounded by 50% of sinus cavities. Odontogenic infections can reach the orbit through the paranasal sinuses and it has been stated that as many as 20% of all cases of sinusitis may have a dental origin.

Orbital cellulitis is the only condition likely to be confused with cavernous sinus thrombosis, but there is a lesser degree of toxaemia and the signs are strictly unilateral. Movements of the eye may be limited but true cranial palsies are not found (Guindi 1983).

Price (1971) discussed the differential diagnosis between orbital cellulitis and cavernous sinus thrombosis considering it most difficult in patients with unilateral ocular movement. The authors found in their study that patients with orbital cellulitis did not have visual loss, retinal changes or adverse neurological findings. As the initial therapy for both conditions is the same, i.e. with antibiotics treatment should be started as soon as either condition is suspected.

Limongelli (1977) discussed the differential diagnosis of orbital cellulitis stating that most of the signs were due to orbital congestion and oedema, loss of muscle tone and retrobulbar pressure from infection. Excess pressure on the optic nerve and its blood supply could result in optic neuritis, optic atrophy and blindness. Resolution of the infection should bring remission of all clinical signs without residual neurological deficit except for blindness. Limongelli discussed a patient who presented with panfacial cellulitis following dental extractions which was unusual as there was contralateral orbital cellulitis and blindness with sparing of the ipsilateral eye. This was considered to be a result of haematogenous spread rather than of direct extension.

Kaban (1980) reported on the usefulness of CT scanning to assess the
spread of an orbital cellulitis associated with odontogenic infection. The CT scan can identify the muscle core, optic nerve and the globe which can be contrasted with the less dense surrounding fat. Inflammatory exudate can be localised to the anterior or posterior aspect of the orbit and the relationship of the fluid to the optic nerve can be demonstrated. In this particular report the CT scan showed fluid in the infratemporal fossa, the ethmoid sinus and the posterior aspect of the orbit surrounding the optic nerve. Surgical management allowed indirect drainage of the orbit by decompressing the infratemporal fossa. If the purulent exudate had been more anteriorly located in the orbit then drainage would have been carried out through an incision in the orbital septum.

Middleton (1980) stressed that periorbital and facial cellulitis were two infections that were easily misdiagnosed because their initial presentations could appear innocuous. Periorbital cellulitis is anterior to the septum of the eyelids with diagnostic signs of circumferential warmth, oedema, erythema and moderate chemosis. Sensation, orbital pressure, pupillary responses and fundi are normal with pain on eye motion generally lacking. Facial cellulitis involves the orbit behind the septum and is associated with proptosis, ophthalmoplegia, impaired vision, marked chemosis, high fever and restricted or painful eye movement. Both periorbital and facial cellulitis require a careful search for underlying infection and should initially be treated with broad spectrum antibiotics because of the wide variety of infecting organisms.

Pellegrino (1980) in discussing the extension of a dental abscess to the orbit stressed the importance of establishing drainage after dental treatment so that potentially fatal sequelae could be avoided. He reminded clinicians that they should not rely on antibiotics to resolve dentoalveolar abscesses as delay in treatment contributed greatly to the progress of the dental
abscess.

*Haemophilus influenzae* type b infections should be included in the differential diagnosis of orbital cellulitis (Simpson et al, 1981). *H. influenzae* strains are gram negative, aerobic, non-motile, non-spore forming strictly parasitic bacteria which live predominantly in the upper aerodigestive tract. It is the type b strain, which in general, is associated with significant morbidity. Most commonly, *H. influenzae* type b infections occur in children aged six months to five years and can produce a variety of pathological manifestations in the head and neck, including periorbital cellulitis. Effective management includes correct diagnosis, prevention of complications and high doses of appropriate antibiotics. Such infections are associated with a detectable bacteraemia so that identification by blood cultures is possible. Alternatively aspiration of the cellulitic area can be diagnostic. Ampicillin was the drug of choice but ampicillin resistance has now emerged. Because of its effectiveness against Staphylococcus, Streptococcus and other pathogens including *H. influenzae* type b, cefamandole is suggested as the initial antibiotic of choice in treating facial and orbital cellulitis. In severe life-threatening infections, therapy is usually begun with both ampicillin and chloramphenicol. Surgical treatment includes drainage of abscesses developing in facial and orbital cellulitis if there is inadequate response to antibiotics or deteriorating vision.

Janakarajah (1985) presented a 14 year old patient with dental infection which proceeded to maxillary sinusitis and orbital cellulitis due to delay in seeking treatment. The author emphasised the close anatomical relationship of the orbit to the oral cavity and the need for early and vigorous treatment of dental infections.

Bullock (1985) reported on four patients with orbital cellulitis
following the extraction of maxillary molar teeth. It was noted that the
time interval between the dental extractions and the development of orbital
symptoms ranged from two hours to thirteen days.

Though the spread of odontogenic infection to the orbit is rare, the
importance of appropriate antimicrobial therapy and the implementation of
adequate and timely surgical drainage cannot be underestimated.

8.5 Mediastinitis

Acute mediastinitis has been reported following oropharyngeal
infections, though it is most commonly seen as a complication of oesophageal
perforation. [Cogan (1973), McCurdy (1975), Howell (1976), Moncada (1978),
(1987)]. The 'mediastinum' refers to the extrapleural portion of the
thoracic cavity intervening between the two pleural sacs. Infections
involving the pre-tracheal space, the lateral pharyngeal space and pyriform
fossa may extend inferiorly into the mediastinum. Respiratory dynamics also
influence the spread of infection within the mediastinum and fluctuations in
the negative intra-thoracic pressures tend to suck the contents of fascial
spaces and the oesophagus into the mediastinum. Mediastinitis need not be
extensive or impressive to produce severe local and systemic reaction. The
symptoms of acute mediastinitis are pain, fever, respiratory distress, and,
since oesophageal perforations account for more than 90% of patients,
dysphagia. On radiography, mediastinal widening is frequently seen as a
primary sign of mediastinitis (Payne 1969).

Since mediastinitis is a secondary condition, every effort should be
made to determine and treat the primary cause of the mediastinal
complication. However, in acute mediastinitis adequate drainage and
prevention of continued contamination are the major considerations in the
surgical management of these patients. In some patients the control of septic and hypovolaemic shock or the maintainance of an airway and ventilation may be of major initial consideration. The identification of the micro-organisms and their sensitivities is important, but initially broad spectrum antibiotics are administered together with correction of fluid and electrolyte imbalances. External drainage can usually be achieved satisfactorily via the cervico-mediastinal route. Early references to this procedure were made by Glogau (1922) and by Sautter (1926). Glogau described an operative method to simultaneously reach from the outside the primary focus within the oral, pharyngeal or laryngeal regions together with the opening and sealing of the collar mediastinum. Glogau believed that the opening and sealing of the collar mediastinum offered the only chance of saving the patient from sepsis (Figure 42).

Sautter (1926) described a patient who developed Ludwig's Angina and
FIGURE 42. Technique of cervical mediastinotomy (Glogau 1922)

Access to the retrovisceral-prevertebral space is obtained through a low cervical incision retracting the sternomastoid muscle laterally and trachea and thyroid medially. Posterior collections as low as the fourth thoracic vertebra are adequately drained through such an incision.
mediastinal abscess following tonsillectomy. He made a fairly thorough search of the literature from 1890 to 1922 and was convinced that mediastinal abscess was a very rare terminal condition following tonsillectomy. He believed that when a patient was not recovering from primary or secondary inflammations of the neck frequent radiographs should be made to anticipate any mediastinal infiltration. He suggested that the drainage incision for Ludwig's Angina should be made at the lowest level of the cellulitis, and whenever possible, parallel with and anterior to the border of the sternomastoid muscles so that the lower end of the wound could be packed off to minimise the possibility of a descending infection.

Furstenberg and Yglesias (1937) reviewed the anatomy of the neck and mediastinum in order to explain the mode of spread of infection from the orofacial region to the mediastinum, and then discussed the surgical management of mediastinitis. Pearse (1938), in the pre-antibiotic era, collected a series of 110 patients with mediastinitis following cervical suppuration. Those treated surgically showed a mortality rate of approximately 50% while those treated medically had a mortality rate of 86%.

Moncada (1978) presented five patients with mediastinitis from odontogenic and deep cervical infections noting that potentially lethal consequences can occur very quickly. In his patients, awareness of such complications and early radiographic diagnosis led to prompt surgical drainage, proper antibiotic therapy and survival after stormy clinical courses. The earliest radiographic sign of invasion of the mediastinum is widening. As the infection progresses there is progressive septicaemia and vascular collapse. Compromise of pulmonary function can occur when fluid accumulates in the extrapleural space.

Murray and Finegold (1984) concluded that anaerobic bacteria play an important role in the pathogenesis of mediastinitis and that the need for
adequate anaerobic culture and appropriate selection of antimicrobial agents along with proper surgical management is crucial in these severe and often fatal infections.

Bounds (1985) described a patient with cervical cellulitis in which mediastinal and subphrenic abscesses formed. In contrast to Moncada (1978) he found that in this patient, despite careful examination of the chest films, both during life and post mortem, no mediastinal widening or gas was detectable.

Rubin (1987) noted the high morbidity and mortality that may be associated with mediastinitis. He described a patient with a one week history of toothache and progressive neck swelling which developed into the most lethal form of mediastinitis, the diffuse necrotising variety. Despite massive antibiotic therapy, tracheostomy and two attempts at cervicomediastinal drainage, the patient died.

It can only be emphasised again that early recognition and aggressive surgical and medical management provide the only chance of survival for patients with these very severe infections.

8.6 Empyema Thoracis

This is the term used to describe the presence of pus in the pleural space, which may be as thin as serous fluid or so thick that it is difficult to aspirate through even a wide-bore needle. The causative organism may or may not be isolated from the pus. An empyema may involve the whole pleural space (total empyema) or only part of it (loculated or encysted empyema). Empyema is always secondary to infection in a neighbouring structure, usually the lung.

Spread of oral infection to the chest may occur via the parapharyngeal space which leads indirectly to the mediastinum or by aspiration of infected material into the bronchial tree or by spread of infection by septic emboli
to the pulmonary circulation. Whichever route of infection occurs, the anatomical source of the infection should be recognised so that appropriate antibiotics are selected which are specific for the putative organisms involved.

Strauss (1980) reported on a patient who developed Ludwig’s Angina, empyema, pulmonary infiltration and pericarditis following the simple removal of an impacted wisdom tooth. Prompt surgical therapy was thought to have saved the patient’s life and a realistic postulation to the infectious aetiology allowed an appropriate antibiotic to be chosen so that empirical therapy was commenced pending availability of culture and sensitivity reports. Other reports in the literature include: Young and Sampson (1980) and Zachariades et al (1988).

These case reports demonstrate that odontogenic infection can result in difficult to control, life-threatening situations, which may be among the most challenging problems faced by oral and maxillo-facial surgeons.

8.7 Vascular Complications

Liston (1843) was the first clinician to show that a chronic abscess of the cervical region could directly communicate with the carotid artery and cause its erosion. Even if ligation of the common or internal carotid arteries is performed, it is frequently accompanied by death or severe morbidity which may be permanent. The major hazard of carotid ligation is the occurrence of cerebral damage (Moore 1955). Venous thrombosis of major neck vessels is somewhat less of a problem when sepsis is treated vigorously with antibiotics. If, however, early recognition of this complication is not accomplished, it, too, can be fatal (Alexander, 1968).

Lifschutz (1931) stated that erosion and perforation of the important blood vessels of the neck, as a result of suppuration from surrounding tissues, was comparatively rare and that haemorrhage was more common from the
internal carotid artery than from any other vessel. Ligation of the common carotid artery appears to be the only treatment that assures recovery when haemorrhage takes place. It is associated with less morbidity than ligation of the internal carotid artery because of retrograde collateral flow through the external carotid artery system. However, if the origin of the bleeding is definitely from the external carotid artery or one of its tributaries, then selective ligation of the involved branch is ideal. If bleeding still persists after ligation of the appropriate vessels, the oral cavity, nasopharynx and abscess cavity can be packed to provide further means of control.

Alexander (1968) reported on four deep neck abscesses with major vascular complications. He suggested that the rupture of a major artery of the neck secondary to an abscess is frequently accompanied by recurrent small haemorrhages, a protracted clinical course, haematoma of the surrounding tissues and shock.

Langenbrunner (1971) presented two patients with pharyngomaxillary space abscesses complicated by carotid artery erosion. The history of tonsillitis or oropharyngeal infection followed by an enlarged neck mass with neurological involvement of cranial nerves IX through XII and the cervical sympathetic chain should alert the clinician to the diagnosis of pharyngomaxillary space abscess. The author advocated aggressive management of such patients with commencement of high dosage empirical antibiotic therapy and maintenance of the airway by tracheostomy if necessary. Surgical approaches may be through the posterior triangle which only gives limited exposure, or via the transpharyngeal route through the bed of the tonsil from which it is extremely difficult to drain the area, or via the Mosher type incision in the neck at the anterior border of the sternomastoid muscle which provides excellent exposure. This allows an abscess to be
drained and if severe haemorrhage occurs then the operation can proceed to the ligation of the artery.

8.8 Necrotising Fasciitis

Necrotising fasciitis of the head and neck is a rare condition which has been reported following odontogenic infections. The disease is characterised by a severe gangrene of the skin and superficial and deep fascia, occasionally with the formation of subcutaneous gas, and is produced by a variety of microorganisms including haemolytic Streptococci, Staphylococci and gram negative bacilli. Synergistic necrotising cellulitis is a variant in which muscle is also involved. The first recording of necrotising fasciitis is believed to be in 1871 by Joseph Jones, a Confederate Surgeon, who described "hospital gangrene". He stated that the skin of the affected parts turned blue, gray or black and virtually melted away in a 24 hour period (Andrews 1969).

This serious infection has a high mortality. It usually occurs as a complication of traumatic or surgical wounds and its highest incidence is seen in patients with ischaemic small-vessel disease, particularly alcoholics and diabetics. The course of the infection may be so rapid that it involves an entire extremity within 24 hours, or it may remain dormant for weeks and then suddenly spread. It may involve any part of the body with the most common location being the lower extremities (Stone et al 1972).

The key to successful management is early diagnosis accompanied by prompt and thorough surgical debridement through wide incisions and fasciotomy. Generally, multiple debridement procedures are necessary, because of progressive gangrene and the need for drainage of loculations of pus and gas. The mixed nature of the infecting flora requires high dose intravenous antimicrobial therapy to cover gram positive and gram negative forms. It is essential to obtain both aerobic and anaerobic cultures and
tissue biopsy, as the definitive antibiotic therapy is dependent on these results. The mortality rate has remained high, despite the introduction of antibiotics and other forms of supportive management. This has been attributed to the severity if associated diseases or the failure to recognise the disease process.

Reports of necrotising fasciitis following dental infections include Crowson (1973). He described a 45 year old man who presented with an extensive neck infection following the removal of an infected wisdom tooth. A diagnosis of necrotising fasciitis was made at surgery. It was thought that the presence of undiagnosed, uncontrolled diabetes and an unfortunate delay in operation were factors in the fatal outcome.

Richardson (1975) described a 51 year old man who developed necrotising fasciitis of the neck following the removal of a carious upper right second molar tooth. The authors showed that prompt treatment of the deep cervical fascial spaces with the excision of necrotic deep fascia and placement of multiple drains were important factors in the limitation of spread of the disease and the survival of the patient. It was emphasised that the carotid sheath should be left intact to serve as a barrier to infections of the carotid artery.

Roser (1977) presented a patient with necrotising fasciitis arising from a periapical abscess. The importance of the surgical drainage was well illustrated as initially the infection was only treated with high dosage antibiotic therapy to which all the causative organisms were susceptible. After extensive and repeated incision and drainage procedures the infection was finally brought under control.

Mruthyunjaya (1981) reported on a patient with a longstanding infection associated with a carious tooth, who presented with a large area of ulceration near the left angle of the mandible and a discharge of foul-
smelling fluid. The area was covered by necrotic tissue and there was
destruction of most of the subcutaneous tissue and fascia. Following local
debridement, high dosage antibiotics and the removal of the offending tooth,
the external wound slowly granulated.

Krespi et al (1981) presented two patients with necrotising fasciitis,
one of which was associated with multiple infected carious teeth. Both
patients were successfully treated with massive debridement, drainage of the
upper mediastinum, intravenous antibiotics and hyperbaric oxygenation. The
authors believed that the use of hyperbaric oxygenation in conjunction with
surgery and antibiotics had a favourable effect on the prognosis of the
disease. In addition a closed bag was placed around the wound so that it was
purged continuously with 100% oxygen for 90 minute periods, 3 to 4 times a
day for 5 days. It was felt that the local application of oxygen to the
wound was beneficial as long as the wound was frequently cleansed and all
debritus removed so that the oxygen had access to all involved areas.

Stock (1987) described a patient with severe dysphagia and rapid
development of swelling and pain in the neck. The patient had been treated
for acute pharyngitis but also had a periapical abscess associated with the
first left mandibular molar tooth. Within 24 hours of admission diffuse
brawny oedema involved the neck from the submandibular area to the
supraclavicular fossa. A CT scan showed marked soft tissue oedema extending
from the base of the skull to the clavicle. There were also several areas
of low density consistent with fluid and their irregular occurrence suggested
extensive undermining and multiple arborizations. The patient underwent
immediate surgery when widespread fascial necrosis and myonecrosis involving
the suprathyroid musculature and platysma was found. The necrotic process had
dissected into the submandibular, lateral pharyngeal, masticator and
retropharyngeal spaces. The CT findings accurately identified the fluid
density produced by the infection and alerted the clinician to the extension of infections beyond the planned field of surgical procedure.

McAndrew (1987) presented a patient with necrotising fasciitis caused by dental infection, which was unusual in the extensive nature of the spread of the disease. Though the patient survived there was severe mutilation. The necessity for early diagnosis and the total debridement of all necrotic tissue until healthy bleating tissue is encountered was re-emphasised. When the disease is controlled then consideration towards skin grafts and reconstruction can be made.

Steel (1987) described a patient with subcutaneous necrotising ulceration present on the chest wall following a dental abscess, which was controlled with high dosage intravenous antibiotics without excision of overlying skin. The author did conclude, however, that the traditional methods of surgical treatment should normally be adhered to, unless rapid remission of the disease process attends the initial antibiotic therapy.

Balcerak et al (1988) reviewed three patients with cervicofacial necrotising fasciitis, two of dental aetiology and one the result of blunt and abrasive facial trauma. All the patients responded well to aggressive surgical treatment together with broad spectrum antibiotic coverage and supportive medical therapy. From their experience the authors concluded that surgical exposure and maintainance of such exposure with gauze packs and irrigation was most important in arresting the spread of the lesion. They believed this to be due to the alteration of an anaerobic fascial environment by oxygenation through exposure and hydrogen peroxide irrigation.

In March 1982, a 64 year old factory worker was transferred to the Westmead Hospital under the care of an associated surgical discipline. He had a one week history of increasing cervicofacial swelling following the removal of a left mandibular molar tooth. On his admission to hospital, he
had apparent dysphagia and a brawny, brown coloured swelling of his submental and right submandibular regions. His floor of mouth was distended. He weighed 101 kilograms and had a daily intake of five beers and smoked at least 10 cigarettes. He was taken to the operating theatre where submental and right submandibular incisions were performed with some drainage of pus. He was returned to the ward where his airway obstruction persisted and during his return to the operating theatre for tracheostomy he suffered respiratory and cardiac arrests. Following his resuscitation, he was transferred to the Intensive Care Unit in a deeply unconscious condition. The following day, multiple carious teeth were removed from the right mandible and radical exposure of the neck revealed necrotic fascia of the right submandibular and cervical regions, extending superiorly to the base of the skull, posteriorly into the retropharynx and down to the thyroid gland. The patient did not regain consciousness and was diagnosed as brain dead 4 days later (Plates 33, 34 and 35).

Post mortem examination showed that infection involved the structures in the right side of the neck and extended to the mediastinum to surround the trachea, oesophagus and other mediastinal structures to the level of the the diaphragm. Microbiological cultures at the time of operation had shown a moderate growth of normal oral flora with anaerobic cultures eventually isolating B. melaninogenicus and B. fragilis. Blood cultures had been negative.

The progress of this patient demonstrates well the rapidity with which respiratory obstruction may occur, significantly related to the pharyngeal and laryngeal swelling which is difficult to appreciate on extra-oral examination. It emphasises the discussion in Chapter 7 that priority for such patients is the establishment and maintenance of a patent airway, and that the care of these patients is best undertaken with appropriately
PLATE 33. This facial view demonstrates the initial submental and submandibular drainage incisions of a patient with necrotising fasciitis.
PLATE 34. This intra-oral view shows multiple carious maxillary and mandibular teeth which were associated with the orofacial infection and necrotising fasciitis.
PLATE 35. This view shows the wide exposure of the necrotic fascial tissues in the right neck.
experienced medical and nursing personnel so that subtle changes in breathing are recognised. It should be appreciated that endotracheal intubation may be virtually impossible when oral and pharyngeal swelling increases rapidly. It should be remembered that the ability to perform intubation can change within a few hours, therefore, it is important that the anaesthetist has a good understanding of the disease process.

It appears that, unfortunately, for this patient the nature of the infection involving the right side of his face and neck was not understood. A diagnosis of cervical haematoma secondary to the recent dental extraction in the left mandible was made, and delay in obtaining the correct diagnosis and management contributed to the extensive spread of the infection. It is interesting to note that the original microbiological cultures grew normal oral flora and identified Bacteroides species as the anaerobic organisms. It is well established that the presence of pus requires surgical drainage and, though infection of this virulence and spread is now unusual in the orofacial region, it is essential that the surgeon has a thorough understanding of the fascial spaces. Otherwise adequate drainage will not be obtained and, as shown in this patient at post-mortem, spread to the mediastinum and diaphragm may occur.

The history of this patient's demise demonstrates very succintly the unfortunate nature of events that can occur in a very short period of time and more than adequately emphasises the two main objectives of management—maintenance of a patent airway and surgical drainage of the infection.

8.9 Ludwig's Angina as a Complication of:

(I) Diabetes mellitus
(II) Renal failure
(III) Multiple systemic involvement
(IV) Myocarditis
(V) Haemophilia

(VI) Dermatomyositis

(VII) Fractured mandible

(VIII) Infection of an oral malignancy

(IX) Infection of an Ameloblastoma

(I) Diabetes Mellitus

This is a metabolic syndrome characterised by an inappropriate elevation of the plasma glucose level associated with changes in lipid, protein and carbohydrate metabolism for which a relative or absolute lack of insulin is responsible. It is associated with degenerative complications and there is increasing evidence to support the concept that poorly controlled diabetes mellitus enhances the likelihood of virulent bacterial and fungal infections.

Wadsworth (1954) reported on a 73-year old woman who, following a vague soreness of the throat, rapidly developed a classical Ludwig's Angina. There was no preceding history of local or systemic infection but there was an acute onset of diabetes and it was therefore considered that the diabetes was a fundamental factor in the spread of the infection and the speed at which it developed. Treatment of the diabetic condition and antibiotic therapy rapidly controlled and cured a serious infection.

Harrison (1983) described the development of Ludwig's Angina from a periodontal abscess in a patient with poorly controlled diabetes mellitus. In spite of treatment reflecting the customary surgical and medical approach to management of this infection, the patient failed to show the consistent improvement that might be expected in a non-diabetic patient. These factors emphasise the important relationship between diabetes mellitus and the management of infection. If increasing findings of infection are present, it is important to strive for normal blood sugar levels in addition to the usual treatment with adequate drainage and appropriate antibiotics.
(II) **Renal Failure**

Clinical shock due to the presence of circulating bacteria and bacterial products has been known as septic shock, endotoxin shock, gram negative septicaemia or bacteraemic shock syndrome. Septic shock has been defined as "a state of hypotension caused by bacteraemia and accompanied by oliguria, signifying decreased peripheral blood flow". Septic shock and acute renal failure initiated by an infected dental cyst was reported by Matthews (1971) in a 43-year old man who had longstanding dental sepsis and was admitted to hospital with acute renal failure. This was due to acute tubular necrosis precipitated by bacteraemic shock arising from an infected dental cyst. The patient was managed initially with dialysis followed by incision and drainage of the infected cyst and subsequently by removal of the infected teeth and cyst.

(III) **Multiple Systemic Involvement**

Fein (1973) described a 68-year old man who was in severe distress with Ludwig's Angina resulting from an infected lower right third molar tooth. Initially, surgical drainage was carried out with the simultaneous administration of high-dosage antibiotics. As a result of medical consultation and laboratory studies concurrent diagnoses of bilateral basilar pneumonitis, early congestive cardiac failure, cirrhosis of the liver and mild diabetes was made. The patient required bilateral thoracocentesis and careful monitoring of his nutritional, fluid and electrolyte balance together with management of his cardiac and pulmonary diseases to enable him to make a satisfactory recovery.

(IV) **Myocarditis**

Myocarditis presents as inflammatory changes within the myocardium without involvement of the valves, pericardium or other cardiac structures. Acute myocarditis may be secondary to almost any infectious disease or may
occur as a primary isolated disease. However, increasingly, primary myocarditis of unknown aetiology is being identified as viral, often the Coxsackie virus. In most cases the clinical course of myocarditis is benign. In severe forms, and especially in the presence of additional cardiovascular loads or damage, congestive heart failure may occur. Cardiac arrhythmias and disturbance of conduction are frequent and may threaten life. Rarely, sudden death may be the first sign of acute myocarditis (Abelmann 1966)

Palank (1979) reported on a patient who suffered "Fatal acute bacterial myocarditis after dentoalveolar abscess". The patient was a healthy 19-year old female who had a dental extraction for an acute abscess 12 days prior to admission and was prescribed oral erythromycin. Following admission with chest pain, clinical and laboratory findings were consistent with an acute anterior wall myocardial infarction and approximately 20 hours later the patient died. It was interesting to note that, despite the course of antibiotic therapy, bacteria occurred in the bloodstream with isolated involvement of the myocardium. The organisms cultured from the blood of the patient before death could be found as normal flora in the oral cavity. At postmortem there was no evidence of endocarditis and no bacteria in any of the other organs. It appeared that the bacteria originating in the oral cavity resulted only in the isolated involvement of the myocardium. Such a presentation of acute bacterial myocarditis is extremely rare.

In the same year Dickson presented a patient who suffered "Fatal myocarditis associated with peritonsillar abscess". This patient was a 58-year old Chinese male in previous good health who had a four-day history of sore throat and malaise. On admission a diagnosis of peritonsillar abscess with spontaneous drainage was made and he was commenced on intravenous penicillin and quickly improved, but the next day the patient suffered a fatal cardio-respiratory arrest. The diagnosis of myocarditis was never
suspected until after the cardiac arrest. It was thought that the streptococcus was probably the aetiologic agent from the tonsillar focus, and that the method of action could have been due to a bacterial toxin or a hypersensitivity reaction.

(V) **Haemophilia**

Nelson (1985) reported the management of Ludwig’s Angina in a patient with Haemophilia A with a high titre of inhibitors to Factor VIII. Of the major types of haemophilia, Haemophilia A (classical haemophilia) is the most common, affecting 80% of all haemophiliacs, and it is characterized by decreased amounts of Factor VIII. Haemorrhage control in patients with classical haemophilia may require replacement therapy with exogenous Factor VIII.

Classic treatment considerations in this patient with Ludwig’s Angina could not be considered initially, until haemostasis could be assured. Intravenous penicillin was chosen as the antibiotic of choice to which, fortunately, a favourable response occurred. Once the Factor VIII levels were corrected the infected tooth was removed under local anaesthesia mandibular block with good resolution of the facial swelling. Though mandibular block injections in haemophiliacs are controversial, it was preferred to general anaesthesia from a haematological standpoint in this particular patient.

(VI) **Dermatomyositis**

This is an uncommon disease characterised by necrotising inflammation of striated muscle, skin and subcutaneous tissue. The disease has been described following trauma, infection, immunisations and the use of drugs. Although no aetiologic factor is known, viral infection and autoimmune disturbances have been implicated. Muscle weakness is usually the presenting symptom accompanied by oedema and dermatological changes. Classic
manifestation consists of a pinkish violet erythema and puffy swelling of the eyelids, face and neck.

Fridrick (1987) presented an interesting patient with dermatomyositis with a superimposed Ludwig’s Angina. The rapid progression of the clinical course necessitated surgical intervention. In the earlier stage, it was said to be virtually impossible to differentiate between signs of dermatomyositis and an infectious process. No clear cause of the facial swelling was determined but it was presumably from an ulcerative lesion in the floor of the mouth. It was thought that many conditions contributed to the patient’s fatal outcome including the dermatomyositis complicated by Ludwig’s Angina, multiple surgical interventions, general anaesthesia, chronic steroid therapy and overwhelming infection.

(VII) Fractured Mandible

Rosen (1972) reported on an 18-year old male who, having sustained a bilateral undisplaced fractured mandible, developed a Ludwig’s Angina four days later. This responded to extra-oral drainage and high dosage antibiotics. Following resolution of his facial swelling, closed reduction and fixation of his facial fractures were performed. Though teeth are often seen in the line of facial fractures they usually cause no problem. However, it should be remembered that they can be the cause of severe septic infection.

(VIII) Infection of an Oral Malignancy

The pathology of Ludwig’s Angina usually involves some form of oral pathosis. Though an odontogenic source is implicated in up to 70% of cases, it can be precipitated by secondary infection of an oral malignancy.

Fischmann and Graham (1985) presented a 60-year old edentulous lady who had Ludwig’s Angina. On intubation a large squamous cell carcinoma of the base of the tongue was identified and infection of this tumour had
precipitated the episode of Ludwig's Angina. The patient's acute infection responded to drainage and high dosage antibiotics.

Grace (1984) described an unusual presentation in an 88-year old man, who developed a sudden swelling in his tongue and floor of mouth together with submandibular swelling. The clinical picture resembled Ludwig's Angina except that he remained afebrile with his white cell count within the normal limits and showed no response to high-dosage antibiotics. Sputum cytology then confirmed a bronchial neoplasm and, in view of the patients debility, all active treatment was ceased and the patient succumbed within 72 hours. At post-mortem, the patient's real symptoms were found to be due to metastatic lingual carcinoma in the tongue and the sudden swelling was a consequence of necrosis. Metastatic spread to the soft tissues of the tongue is rare, usually late and of poor prognosis. Only one in every thousand metastatic tumours will involve the soft tissues of the tongue, the lungs being by far the most common primary site.

(IX) Infection of an Ameloblastoma

Tsuji (1984) reported on a patient with "Ludwig's Angina as a complication of ameloblastoma of the mandible". A 45-year old man who had been aware of a swelling in his left mandible for three years finally sought care when he could not eat because of the great increase in pain and swelling. Radiological examination showed a polycystic lesion of bone destruction involving the angle and the ramus of the left mandible. A diagnosis of Ludwig's Angina was made, due to probable ameloblastoma of the mandible. Surgical drainage was performed and high-dosage antibiotics given but the patient died the following day due to cardiac insufficiency.

At post mortem, it was found that infection involved the submandibular, sublingual and submental spaces bilaterally and had also spread to the left temporal fossa, subcutaneous tissues of the face, muscles of mastication,
cervical and mediastinal regions, connective tissues near the thyroid gland and the thoracic cavity. The provisional diagnosis of ameloblastoma was confirmed histopathologically. It was concluded that the extent of the infection at the time of admission was the most important factor in the death of the patient (Figure 43).

8.10 Complications of Orofacial Infections in Children

There are a few reports in the literature which are strictly related to Ludwig's Angina in children. Steinhauer (1967) reported on a case of Ludwig's Angina in a 12-day old baby. It appeared the baby sustained a laceration, about 1-cm long, in the floor of the mouth which was the source of the disease. Surgical intervention together with the critical handling of the intravenous fluid therapy, feedings and drug administration allowed full recovery.

Barkin (1975) presented four children with Ludwig's Angina aged between 19 months and 10 years. Each of them presented with an acute onset of massive swelling of the tongue and hypopharyngeal space, giving a typical protruding tongue and "bull neck " appearance. Three of the children had severe underlying deficiencies in host defense, while the fourth child was in good health prior to the onset of the illness, for which no reason could be found.

Gross and Nieburg (1977) described Ludwig's Angina in two young children, one following trauma and one associated with a carious tooth.

Briggs (1979) presented a 15-year old girl who had Ludwig's Angina associated with her carious teeth, which resolved with intravenous penicillin. However, six weeks later she returned with a painful swelling below the right mandible, which increased in size with salivary stimulation. As her symptoms began in the resolution phase of the infection and were progressive, a diagnosis of a fibrosed submandibular duct subsequent to
FIGURE 43. Diagram showing extent of the infection as a complication of ameloblastoma of the mandible
Ludwig's Angina was made. This was confirmed by surgical intervention.

Brook and Friedman (1982) described two children with intracranial complications of periapical abscesses of the upper incisor teeth. The infection spread to the central nervous system through the maxillary, ethmoid and frontal sinuses. Subdural empyema occurred in both children and both patients presented with frontal headache and pansinusitis. CT was instrumental in the early diagnosis of the intracranial infection. Surgical drainage and appropriate antimicrobial therapy resulted in complete eradication of the infection in both children.

Brook (1987) demonstrated the importance of anaerobic bacteria in abscesses of the head and neck. These organisms were usually mixed with aerobic bacteria and were recovered in greater frequency in abscesses of the head than those of the neck. They were also recovered in greater frequency in infections that originated from sites where anaerobes are involved in chronic infection, such as teeth, sinuses and tonsils. Brook's data also demonstrated the presence of beta-lactamase producing organisms in 46% of the head and neck abscesses in children, and these would account for the reports of clinical failure following penicillin therapy. Penicillin resistance should, therefore, be anticipated when using antimicrobial therapy. The importance of appropriate aerobic and anaerobic culturing techniques is also emphasised.

8.11 Conclusion

Without a doubt, serious and often fatal complications of odontogenic infections, could be avoided by early dental treatment. Where extensive local and systemic problems occur, then management needs to be aggressive. One would hope that education will control the fear and ignorance that prevents many individuals from receiving routine dental care.
CHAPTER 9

RETROSPECTIVE STUDY
9.1 Introduction

The community in the Western suburbs of Sydney continues to experience life-threatening orofacial infections, which may pose significant management difficulties. This retrospective study consists of 90 patients who presented to the Westmead Hospital during the period 1981 to 1988. They had ignored the early symptoms of localised orofacial infection and their concern had been aroused by the sudden extent of their facial swelling and accompanying upper aerodigestive tract difficulties. The aim of this study was to evaluate the clinical management of this group of patients.

9.2 Materials and Methods

The Western Metropolitan Health Service of Sydney cares for a population of approximately two million people. The area contains a high proportion of socially disadvantaged persons whose dental care tends to be neglected. The clinical management of these 90 patients was based on systematic examination and investigations.

The clinical investigations included the basic parameters of pulse, temperature, blood pressure and urinalysis. Radiographic examination of the head and neck was undertaken for every patient: this would both show the underlying aetiology of infection and evaluate the encroachment of soft tissue swelling on the airway. Blood tests were performed, when indicated, looking for underlying medical problems such as anaemia, diabetes, hepatic and renal disease. In patients over the age of 45 Chest X-ray and ECG were indicated as base-line studies for individuals undergoing general anaesthesia. They were also performed when there was a clinical indication following physical examination of the patient.

With the establishment of the diagnosis of an orofacial and neck infection the principal considerations of treatment involved:

- airway management and choice of anaesthesia
- surgical drainage of the infection
- general medical care
- antibiotic therapy
- microbiological cultures and sensitivities including both aerobic and anaerobic studies of the infective lesions. (Anaerobic cultures were carried out on approximately 25% of specimens).
- dental care and dental education.

9.3 Results of the Study

<table>
<thead>
<tr>
<th>Patients</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>90</td>
<td>100%</td>
</tr>
</tbody>
</table>

Age Range: 7 yrs to 77 yrs
(Median Age = 30 yrs)

Gender: - Male : 59 66%
- Female : 31 34%

"Socio-economic" Status:
- employed (E) : 44 49%
- unemployed (U) : 18 20%
- pensioners (P) : 17 19%
- students (S) : 11 12%

Presentation of patients from onset of orofacial infection:
- within 3 days : 33 37%
- 4-7 days : 38 42%
- more than 7 days : 19 21%

SYMPTOMS AND SIGNS:
- Swelling of the face and neck : 90 100%
- Elevated temperature >37.5° : 90 100%
- Pain : 90 100%
<table>
<thead>
<tr>
<th>Condition</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Halitosis</td>
<td>90</td>
<td>100%</td>
</tr>
<tr>
<td>Trismus (inter-incisal distance)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nil - &gt;25 mms</td>
<td>15</td>
<td>17%</td>
</tr>
<tr>
<td>Mild - 16-25 mms</td>
<td>21</td>
<td>23%</td>
</tr>
<tr>
<td>Moderate - 6-15 mms</td>
<td>35</td>
<td>39%</td>
</tr>
<tr>
<td>Severe - &lt;5 mms</td>
<td>19</td>
<td>21%</td>
</tr>
<tr>
<td>Dysphagia</td>
<td>79</td>
<td>88%</td>
</tr>
<tr>
<td>Dyspnoea</td>
<td>25</td>
<td>28%</td>
</tr>
<tr>
<td>Dysphonia</td>
<td>15</td>
<td>17%</td>
</tr>
<tr>
<td>Dehydration</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(minimal oral intake in the 24 hours prior to admission)</td>
<td>90</td>
<td>100%</td>
</tr>
</tbody>
</table>

**SITE AND AETIOLOGY OF INFECTION**

<table>
<thead>
<tr>
<th>Category</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Odontogenic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maxillary teeth</td>
<td>20</td>
<td>22%</td>
</tr>
<tr>
<td>Mandibular teeth</td>
<td>65</td>
<td>73%</td>
</tr>
<tr>
<td>Dental caries/Periodontal disease</td>
<td>48</td>
<td>53%</td>
</tr>
<tr>
<td>Impacted mandibular third molar</td>
<td>19</td>
<td>21%</td>
</tr>
<tr>
<td>Post-operative dental infections</td>
<td>18</td>
<td>20%</td>
</tr>
<tr>
<td>Others</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fractured mandibles</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(involving impacted third molar tooth)</td>
<td>2</td>
<td>2%</td>
</tr>
<tr>
<td>Facial lacerations</td>
<td>1</td>
<td>1%</td>
</tr>
<tr>
<td>Acute sialadenitis</td>
<td>1</td>
<td>1%</td>
</tr>
<tr>
<td>Ranula</td>
<td>1</td>
<td>1%</td>
</tr>
</tbody>
</table>
## SYSTEMIC AND SOCIAL FACTORS

<table>
<thead>
<tr>
<th>General</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Diabetes</td>
<td>4</td>
<td>4%</td>
</tr>
<tr>
<td>- Respiratory disease</td>
<td>12</td>
<td>13%</td>
</tr>
<tr>
<td>- Obesity (&gt;25% normal weight for height)</td>
<td>15</td>
<td>17%</td>
</tr>
<tr>
<td>- Cardiovascular disease</td>
<td>9</td>
<td>10%</td>
</tr>
<tr>
<td>- Renal disease</td>
<td>1</td>
<td>1%</td>
</tr>
<tr>
<td>- Pregnancy</td>
<td>1</td>
<td>1%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Social</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>- Alcoholism</td>
<td>21</td>
<td>23%</td>
</tr>
<tr>
<td>- Other drugs of addiction</td>
<td>9</td>
<td>10%</td>
</tr>
</tbody>
</table>

### FORMS OF MANAGEMENT

#### ANAESTHETIC MANAGEMENT

Technique for tracheal intubation

<table>
<thead>
<tr>
<th>Direct</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>- Conventional intubation</td>
<td>49</td>
<td>54%</td>
</tr>
<tr>
<td>- Conscious nasal intubation</td>
<td>17</td>
<td>19%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Indirect</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>- Controlled blind nasal intubation</td>
<td>21</td>
<td>23%</td>
</tr>
<tr>
<td>- Use of the fibreoptic bronchoscope</td>
<td>3</td>
<td>4%</td>
</tr>
<tr>
<td>- Cricothyroid puncture</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>- Tracheostomy - primarily</td>
<td>0</td>
<td>0%</td>
</tr>
</tbody>
</table>

#### Post-operative airway management

| - Self sufficient                           | 75     | 83%  |
| - Endotracheal intubation                   | 10     | 11%  |
- Tracheostomy
  Number: 5  Percentage: 6%

**Surgical Management**

- Drainage - intra-oral
  Number: 90  Percentage: 100%
- Drainage - extra-oral
  Number: 60  Percentage: 67%
- Removal of involved teeth
  Number: 68  Percentage: 75%

**Antibiotic Administration**

- Penicillin
  Number: 76  Percentage: 85%
- Amoxycillin/Ampicillin
  Number: 3  Percentage: 3%
- Flucloxacillin
  Number: 7  Percentage: 8%
- Erythromycin
  Number: 6  Percentage: 7%
- Cephalosporins
  Number: 5  Percentage: 6%
- Co-trimoxazole
  Number: 2  Percentage: 2%
- Metronidazole
  Number: 40  Percentage: 45%
- Gentamycin
  Number: 6  Percentage: 7%
- Clindamycin
  Number: 1  Percentage: 1%
- Tobramycin
  Number: 1  Percentage: 1%

**Range of Microorganisms Cultured**

**From Specimens of Orofacial Infective Lesions**

- Streptococcus (79 patients)
  - Alpha-haemolytic
    Number: 37
  - Beta-haemolytic
    Number: 56
  - Non-haemolytic
    Number: 29
- Staphylococcus
  Number: 15
- Neisseria
  Number: 15
- Haemophilus
  Number: 12
- Bacteroides : 18
- Actinomyces : 4
- Fusobacterium : 10
- Arachnia : 1
- Corynebacterium : 2
- Enterobacterium : 1
- Eubacterium : 3
- Klebsiella : 4
- Lactobacillus : 1
- Micrococcus : 1
- Peptostreptococcus : 3
- Propionobacterium : 3
- Pseudomonas : 2
- Serratia : 1
- Veillonella : 3

GENERAL MEDICAL CARE

- Clinical examination and investigations : 90
- Maintenance of nutrition : 90
- Control of pain and anxiety : 90
- Upgrading of oral hygiene : 90

Length of stay in hospital

- Range : 2 days to 5 weeks
- Median : 5 days

POSTOPERATIVE COMPLICATIONS

- Deep vein thrombosis (DVT) : 1 1%
- Postoperative pancreatitis : 1 1%
- Anaphylaxis (Penicillin) : 1 1%
- Diabetic crisis : 1 1%
- Delirium tremens (DTs) : 2 2%
- Lower airway complications : 9 10%

**POSTOPERATIVE OUTCOME**
- Recovery : 89 99%
- Death (from necrotising fasciitis and cardio-respiratory arrest) : 1 1%

### 9.4 Discussion and Conclusion

This study supported the reports in the literature as discussed in Chapter 1. The individuals most often afflicted with a major orofacial infection were on average, in their third decade but infections can affect both the young and the aged. In this particular study 33% of the group were female. Often early symptoms were ignored and it was only when the individual became debilitated, distressed or anxious that they sought treatment. 66% of the group presented after 3 days of onset of their orofacial infection when symptoms of increasing pain and facial swelling and an inability to eat and drink had become intolerable.

The general clinical signs of elevated temperature, pulse rate, respiration rate and blood pressure were present, together with increasing pain and swelling due to the infection. This had led to a lack of sleep with consequent emotional instability and exhaustion. Rapid clinical assessment was necessary so that their primary care could be commenced. Increasing dysphagia, breathing difficulties and hoarseness of the voice indicated significant swelling within the upper aerodigestive tract. When this occurred priority was airway control.

The source of infection in 95% of these patients was odontogenic,
predominantly involving mandibular teeth. This included postoperative dental infections, pericoronary infections associated with impacted third molar teeth, dental caries and periodontal disease. Postoperative infections followed a history of "difficult" extractions of teeth which were extensively carious or abscessed. While gross dental caries and impacted teeth were relatively easy to diagnose, it was sometimes difficult to locate non-vital teeth in a mouth which had received extensive dental treatment. The place of inadequate dentistry was not forgotten, nor inappropriate advice from both dental and medical clinicians. This was demonstrated by a patient in the study who was known to have a ranula and was advised that no treatment was necessary unless he had any problems. Unfortunately, his first difficulty was acute requiring intubation initially to overcome his respiratory obstruction and subsequently tracheostomy.

The majority of the individuals had no significant past medical history but it was important to ensure that no underlying pathology was present. Blood tests such as full blood examination and serum biochemical analysis were performed, when indicated, to check for anaemia, diabetes, hepatic and renal disease. Patients presenting with underlying problems of alcohol and drug addiction often required management of withdrawal symptoms. For many patients fear of dental extractions had been primary in avoiding dental treatment.

There is no doubt that surgical intervention in establishing both drainage of the infection and removal of the cause is absolutely essential and provides the most rapid recovery. Though radiographic investigations may aid in establishing the diagnosis, it is the clinical examination and the knowledge of the potential spread of the infection from the initial focus that is important in enabling appropriate treatment to be undertaken. I believe this remains the key to the management of these patients. Adjunctive
patients. Adjunctive empirical antibiotic therapy is also commenced but it is the surgical management which brings about the rapid resolution of both the infection and the associated pain. Also of particular importance is the upgrading of oral hygiene and the institution of local measures to combat infection. In this study, rapid resolution of these orofacial infections occurred once definitive surgical management of both the primary and secondary sites had been performed.

In order that surgical treatment could be undertaken control of the airway and the administration of general anaesthesia was essential. It was important that the choice of anaesthetic technique and postoperative airway care was made early as respiratory problems occur rapidly. If the anaesthetic was administered before acute respiratory difficulties occurred then conventional direct intubation techniques were used.

However, this study showed that 46% of the patients were not intubated in a conventional direct technique. Obviously, it was important that the patient was not paralysed unless ventilation could be maintained easily and direct intubation could be performed. In circumstances where severe trismus is present, particularly of a longstanding nature, and it was anticipated that the administration of general anaesthesia would not allow relaxation appropriate for endotracheal intubation or could even precipitate laryngeal obstruction then conscious nasal intubation was considered. With careful explanation and preparation of the patient, this technique was a safe method of securing the airway prior to administration of general anaesthesia. In this series, this technique was successfully used for 19% of the patients. Fibreoptic bronchoscopy, used for 4% of the patients, was not a simple technique and required a skilled anaesthetist who was well-experienced in its use. This procedure was found to be time consuming and difficult to undertake especially in a patient with impending airway problems. Similarly,
controlled blind nasal intubation also requires experience and was a technique with which the anaesthetist was familiar before proceeding to patients with facial and neck swelling. Controlled blind nasal intubation was used for 23% of the patients in this study.

In patients in whom conventional intubation was anticipated, no pre-medication was given and no paralysing agents administered until test ventilation was performed and jaw relaxation ascertained. Laryngoscopy was then performed to view the extent of swelling and oedema present in the upper airway. If the vocal cords could be viewed then conventional paralysing agents were administered and endotracheal intubation performed.

As discussed previously, tracheostomy can be difficult due to distorted anatomy associated with neck infection and swelling and was not recommended for primary management. However, it could well be suitable for the immediate postoperative care. Examination of the airway would reveal the extent of the swelling and consideration of this, together with further anticipated swelling due to surgery, would decide the postoperative management - extubation, intubation or tracheostomy. Assessment of the lower airway was also important so that adequate ventilation was maintained. Patients had to be able to control their secretions and prevent inhalation of infected material.

Postoperative care was undertaken in an appropriate setting - intensive care or high dependency surgical areas. If patients were already fatigued and debilitated they were often unable to communicate any breathing problems and it was therefore important that they were cared for in an environment where any difficulties could be detected early.

Postoperatively, the administration of intravenous antibiotics together with the rehydration of the patient were maintained. The control of pain, using non-narcotic analgesics on a regular basis, avoided the respiratory
depression associated with narcotic drugs. Chest physiotherapy was also commenced in order to avoid lower airway complications.

Mobilisation was essential in order to avoid problems of deep vein thrombosis and pulmonary embolism, and the patients were encouraged to return to normal activities. Re-establishment of nutrition, initially in a liquidised, homogenised form, was commenced. For those patients, who were unable to tolerate even a liquid diet, naso-gastric feeding was undertaken. Any infection no matter how mild, has some measurable adverse effect on nutritional status: sepsis increases the calorie demand 11-13% for each degree increase in body temperature. The increased excretion of nitrogen resulting from both decreased protein synthesis and accelerated catabolism of muscle protein leads to a negative nitrogen balance. In addition, the febrile state may contribute electrolyte and water loss via sweat. Once the catabolic phase is over, the dietary intake must be sufficient to allow for repletion of the peripheral tissues and it should be remembered that the anabolic period is several times longer than the catabolic one (Scrimshaw et al 1976, Falender et al 1987).

Local measures of hygiene were commenced with irrigation of the surgical sites using copious amounts of warm saline. Normal toothbrushing techniques were also used and if necessary upgrading of oral hygiene with scaling of the teeth.

Microbiological studies of specimens from the orofacial infective lesions showed that the most common bacteria isolated were various species of Streptococci. In this study they were isolated in specimens from 88% of the patients. The major anaerobic organisms identified were: Bacteroides, Fusobacterium and Actinomyces species, which parallels the general trend of the increased incidence of anaerobic involvement in orofacial infections reported in the literature. Ideally, anaerobic cultures and sensitivities
of the infective lesions should be performed as there is always a need for recognition that, on some occasions, atypical organisms may cause "classical" infections. This study has shown that the patients made rapid clinical improvement following surgical intervention, however, when the patient's general status did not improve in the first twelve to twenty-four hours in association with other measures then repeated aerobic and anaerobic cultures were obtained. In the future it is also important that the microbiological profile is updated. This study supported the consensus in the literature that the microbiota of orofacial odontogenic infections generally arise from combinations of organisms found in the normal oral flora.

Antibiotic therapy was directed towards likely pathogens in any given clinical infection. For the treatment of such serious infections, parenteral antibiotic therapy was the method of choice. It was important to maintain plasma MIC levels of antibiotic in the blood for an adequate period of time. Based on the fact that many of the organisms cultured from the oral cavity were sensitive to penicillin, this was still the drug of choice. However, due to the increasing number of beta-lactamase producing anaerobes, metronidazole was also recommended, but importantly, the initial management with surgical drainage changed the environment and discouraged the continued proliferation of the anaerobic population. The dosages recommended by this study, were:

- Benzylpenicillin - 600 mgs, 4 hourly (intravenously)
- Metronidazole - 500 mgs, 8 hourly (intravenously)

For patients with a known penicillin allergy, Erythromycin was the alternative drug of choice. In this study, the use of other drugs such as the cephalosporins, ampicillin and co-trimoxazole, had usually been chosen by other attending clinicians and these were continued if the patient showed clinical improvement and the microorganisms proved sensitive. The addition
of an aminoglycoside was thought to be necessary when Staphylococcus was found to be present. The continued sensitivities of the microorganisms within the oral cavity to penicillin and metronidazole eliminated the need to use other drugs with major adverse side-effects. Importantly, the route of administration and dosage had to be suitable to combat these severe infections.

The median length of stay in hospital was 5 days and was evidence that rapid recovery occurred once the correct surgical intervention had been undertaken. Longer stays were related to the management of patients with post-operative complications. An obese patient, weighing 140 kg, with Ludwig’s Angina suffered a deep vein thrombosis in the left leg and was hospitalised for three weeks. A 77-year old man with multiple medical problems including poorly controlled diabetes and alcoholism suffered postoperative pancreatitis together with a diabetic crisis and was hospitalised for the longest period of 5 weeks. One anaphylactic reaction to penicillin was recorded; this occurred while the patient was intubated for his surgical treatment. Lower airway complications were seen in patients who had a history of asthma or who were heavy smokers. Two of the alcoholic patients required management of their delirium tremens. As discussed in section 8.8 one patient whose condition was complicated by necrotising fasciitis, died following a cardio-respiratory arrest. The other 89 patients in the study made a full recovery.

In the long-term, it is essential that other dental problems are treated appropriately. Fear of dentistry needs education and occasionally the assistance of the clinical psychologist to overcome such phobias. Education of both dental and medical personnel is also necessary so that they understand that such afflicted individuals need urgent hospitalised treatment. The primary consideration of interdisciplinary treatment must
involve airway and surgical management, with removal of the aetiological factors, and not just treatment of the symptoms and signs with indiscriminate use of antibiotics.

In conclusion, this retrospective study has shown that early definitive surgical intervention of odontogenic and orofacial fascial space infections, once a patient's airway has been secured, allows for rapid clinical improvement and significant relief of pain with minimal postoperative morbidity. Adjunctive treatment of high dosage intravenous antibiotics, with penicillin and metronidazole as the drugs of choice, is recommended. These should be commenced empirically pending the results of microbiological culture and sensitivity tests. General care of the patient, with particular attention to their nutritional status, and control of their pain and anxiety should be combined with upgrading of their oral hygiene and dental treatment. In the long-term, dental education forms an integral part of the recovery programme, not only for the afflicted patient, but is also a community health preventive measure.


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