The head and neck contain a number of spaces that can be invaded by organisms of the mouth or by spread of cervical osteomyelitis. Infection in these spaces may progress from superficial infection to cellulitis to the formation of an abscess requiring immediate drainage. Spread of infection between spaces depends on anatomic location. Most patients require hospitalization and intravenous antibiotic therapy. Because a deep space infection may be occult, a high index of suspicion is required for diagnosis. Early recognition is necessary to avoid tissue damage, bacteremia or airway compromise. The possibility of deep space infection should be considered in any patient who does not respond to the usual treatment of an abscessed tooth or tonsillitis. This type of infection also should be considered in a toxic patient who has a fever of unknown origin, with or without blood cultures that show anaerobic organisms. Computed tomography or magnetic resonance imaging is usually necessary to locate the infection and to detect suppuration that will be amenable to surgical exploration and drainage.
Major soft tissue infections of the head and neck are uncommon, but they can be catastrophic. These infections can spread rapidly through soft tissue planes and can threaten airway patency. Each of the major soft tissue infections has a characteristic location (Figure 1) and clinical presentation (Table 1). Although all of these infections can be treated with antibiotics, referral for surgery is sometimes required. This article reviews the diagnosis and treatment of serious soft tissue infections of the head and neck (Table 2).

![Peritonsillar abscess](image)

**Figure 2.** Peritonsillar abscess, with suppuration evident within the areolar tissue between the tonsil and the superior pharyngeal constrictor muscle. Note the characteristic deviation of the uvula to the opposite side. Circles show possible sites of drainage.

**Table 1. Etiology and Clinical Presentation of Head and Neck Infections**

<table>
<thead>
<tr>
<th>Infection</th>
<th>Etiology</th>
<th>Clinical presentation</th>
</tr>
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<tbody>
<tr>
<td>Peritonsillar abscess</td>
<td>Mixed oral aerobes and anaerobes; follows tonsillitis</td>
<td>Young adult with fever, dysphagia, sore throat; muffled &quot;hot potato&quot; voice, otalgia and tender cervical adenopathy</td>
</tr>
</tbody>
</table>
Submandibular cellulitis
   Mixed oral aerobes and anaerobes; follows tooth abscess
   Drooling, dyspnea, stridor and hyperextended neck; tongue
   may be elevated; usually no adenopathy

Lateral pharyngeal space infection
   Mixed oral organisms
   Fever, induration of carotid sheath; diagnosis often
delayed, with patient presenting as toxic with obscure
septicemia and metastatic abscess

Retropharyngeal space infection
   Mixed oral organisms; due to trauma or direct spread of other
   head or neck infection
   Dysphagia, dyspnea, head tilted to unaffected side and
cervical lordosis

Cervical prevertebral space infection
   Staphylococci or *Mycobacterium tuberculosis*; occurs as direct
   spread from cervical osteomyelitis
   Fever, tenderness over cervical spine with little
   swelling of external neck

Malignant external otitis
   *Pseudomonas aeruginosa*
   Dull, boring ear pain; granulation tissue; neuropathy of
   the seventh, ninth and twelfth cranial nerves.

Table 2. Diagnosis and Treatment of Head and Neck Infections

<table>
<thead>
<tr>
<th>Infection</th>
<th>Diagnosis</th>
<th>Treatment</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peritonsillar abscess</td>
<td>Throat examination showing tender peritonsillar mass displacing uvula</td>
<td>Administer IV penicillin; perform surgical drainage by needle aspiration or by incision and drainage</td>
<td>Beware of airway obstruction with bilateral involvement, laryngeal edema or retropharyngeal extension</td>
</tr>
</tbody>
</table>
Submandibular cellulitis
Fever; induration of floor of the mouth
Secure airway with tracheostomy if needed; administer IV penicillin; consider tooth extraction and surgical drainage
Surgery is indicated for airway compromise, pus, gas on lateral neck radiograph or failure of IV antibiotics

Lateral pharyngeal space infection
Neck CT, MRI or gallium scan
Administer IV penicillin; perform incision and drainage of abscess
Blood cultures may reveal anaerobes

Retropharyngeal space infection
Neck examination and lateral neck radiograph showing retropharyngeal swelling
Administer broad-spectrum antibiotics; drain suppuration
Beware of airway obstruction from laryngeal edema

Cervical prevertebral space infection
Soft tissue neck radiograph showing swelling; neck CT or MRI
Administer antistaphylococcal antibiotics; drain suppuration
Beware of spinal cord or root compression, meningitis

Malignant external otitis
Head CT or MRI
Administer antipseudomonal antibiotics, such as gentamicin (Garamycin), pipericillin (Pipracil) or ciprofloxacin (Cipro)
Maintain high index of suspicion in diabetic patients and in other immunosuppressed patients.

Peritonsillar abscess
Peritonsillar abscess, or quinsy, is a complication of tonsillitis. Infection begins in the tonsils and then moves posteriorly into the loose areolar tissue between the tonsillar capsule and the superior pharyngeal constrictor muscle. Pus forms in this space, giving rise to the distinctive clinical features of the infection.
Peritonsillar abscess most commonly occurs in persons 15 to 30 years of age, with the average age at occurrence being 22 years. Seventy percent of cases occur during the winter months (October through February). In one study, 20 percent of patients with peritonsillar abscess were taking penicillin at the time of diagnosis.

Most patients with peritonsillar abscess present with a history of gradually increasing temperature, dysphagia and severe sore throat. A muffled "hot potato" voice and ipsilateral ear fullness and otalgia are characteristic features caused by swelling.
of the soft palate and occlusion of the eustachian tube. Examination of the pharynx reveals a tender peritonsillar mass displacing the uvula and the soft palate (Figure 2). Tender cervical adenopathy is common. Trismus, which results from irritation of the internal pterygoid muscle, is present in patients with anterior abscesses. Because trismus does not accompany posterior tonsillar abscesses, it may be assumed that only cellulitis is present.

Complications of peritonsillar abscess include airway obstruction, usually in patients with bilateral abscesses or laryngeal edema, and spread of infection through the superior constrictor muscle to the lateral pharyngeal space. Before the introduction of antibiotics, the mortality rate associated with peritonsillar abscess was 50 percent, primarily due to the spread of infection.

Therapy for peritonsillar abscess includes antibiotic coverage of alpha- and beta-hemolytic streptococci and anaerobes, most commonly *Fusobacterium nucleatum* and *Bacteroides melaninogenicus*. Penicillin is the drug of choice, followed by amoxicillin / clavulanate (Augmentin).

Traditional surgical treatment of peritonsillar abscess involves incision and drainage of the abscess. The mucosa is incised sharply, and the abscess cavity is entered with a blunt hemostat to avoid damaging the carotid artery.

Drainage by means of immediate needle aspiration is becoming popular as an alternative to incision and drainage (Figure 2). One study compared 18-gauge needle aspiration at three points with incision and drainage, both followed by oral penicillin therapy. No differences were found in the failure rates for aspiration as compared with incision and drainage. Patients in both groups were able to eat comfortably after an average of 3.7 days.

Tonsillectomy is appropriate for patients with recurrent peritonsillar abscess. However, recurrence is rare in patients over age 40 and in patients with no past history of tonsillitis.

**Submandibular Cellulitis**

Submandibular cellulitis was described by von Ludwig in 1836 as a "gangrenous induration of the connective tissues of the neck which advances to involve the tissues which cover the small muscles between the larynx and the floor of the mouth". Subsequently known
as Ludwig's angina, this rapidly spreading cellulitis occurs almost exclusively after a dental infection. Submandibular cellulitis often follows dental extraction for periapical infection of the mandibular molars. If the abscess has spread into bone, no drainage of the abscess cavity occurs from the extraction alone. Taking the path of least resistance, the infection penetrates the thin alveolar bone of the medial mandible and enters the submaxillary space (Figure 3). Cellulitis in the submaxillary space can spread to the sublingual space, leading to the classic clinical presentation (Figure 4).

Patients with submandibular cellulitis typically are febrile, with tachycardia, odynophagia and trismus. Dentition is often poor, with recent extraction, signs of periodontal disease or percussion tenderness of lower molars. The floor of the mouth is indurated, and the tongue is often elevated superiorly against the palate and posteriorly into the hypopharynx.

The clinical features of submandibular cellulitis mimic those of epiglottitis and include drooling, dyspnea, stridor and a tendency for the patient to sit in a semi-erect position with the neck hyperextended. Because the cellulitis spreads so rapidly, there is no time for cervical adenopathy or fluctuance to develop. However, gas from gas-forming organisms may be seen on a lateral soft tissue radiograph.

Progression to complete airway obstruction is common. Historically, the condition has been described as "morbus strangulatorius", and the 60 percent mortality rate reported by von Ludwig was the result of complete airway obstruction.

The first step in the treatment of submandibular cellulitis is ensuring airway patency. In one series, 35 percent of patients required intubation. Because emergency intubation is difficult, early recognition of impending airway compromise is critical; orderly tracheostomy under local anesthesia can then follow. However, in a recent series of 14 cases, tracheostomy was avoided in all but one patient with the use of aggressive antimicrobial therapy, early surgical intervention and careful monitoring of respiratory system.

Intravenous penicillin or oxacillin (12 to 20 million units per day in six doses) is the treatment of choice, with efficacy against the mostly mixed anaerobes and alpha-hemolytic streptococci that cause the infection. Neonates and patients with diabetes or systemic lupus erythematosus should also be treated for gram-
negative aerobic infections. Tooth extraction with surgical drainage is a requisite for those with airway compromise, soft tissue gas, evidence of pus collection or failure to respond to intravenous antibiotic therapy. Because the cellulitis resolves slowly, patients usually require hospitalization for about eight days.

Deep Space Infections

Deep space infections of the head and neck are polymicrobial in origin. If untreated, these infections can produce extensive tissue necrosis of fascial and muscle compartments, with little obvious damage to overlying skin and subcutaneous tissue. Sites of spreading are defined by the anatomic location of the lateral pharyngeal space, the retropharyngeal space and the prevertebral space (Figure 1).

Lateral Pharyngeal Space Infection

The lateral pharyngeal space lies lateral to the superior pharyngeal constrictors and the oral cavity. The space is bounded laterally by the internal pterygoid muscle, the mandible and the parotid gland. Infection in this space usually occurs as a complication of dental infection, submandibular cellulitis, pharyngitis or peritonsillar abscess.

The lateral pharyngeal space is divided into anterior muscular and posterior neurovascular compartments by the styloid process and the stylopharyngeus muscle. Since posterior infection threatens the carotid sheath, infection in the lateral pharyngeal space carries the risk of suppurative jugular thrombophlebitis, which is detectable as an indurated swelling along and behind the sternocleidomastoid arch. Neuropathy of the 11th and 12th cranial nerves may also occur. However, no one sign is very sensitive, and delays in diagnosis are common.

Lateral pharyngeal space infection associated with pharyngitis can present up to three weeks after the initial infection. Patients may be acutely ill, or they may have an obscure septicemia with fever of unknown origin. Anaerobic endocarditis or metastatic bone or lung abscess should prompt a search of the neck. Gallium scanning or magnetic resonance imaging (MRI) of the neck is usually indicated.

Infection of the lateral pharyngeal space is treated with
prolonged intravenous antibiotics, and incision and drainage of abscesses. In one series of eight patients, needle aspiration guided by computed tomography (CT) provided effective non-surgical drainage.

**Retropharyngeal Space Infection**

The retropharyngeal space lies behind the pharyngeal constrictors but anterior to the prevertebral fascia, extending from the base of the skull to the level of the T1 or T2 vertebra. Retropharyngeal space infection results from direct trauma, direct spread of a dental or peritonsillar abscess, or medial extension of a lateral pharyngeal space infection.

The clinical presentation of retropharyngeal space infection includes difficulty swallowing or breathing, anterolateral neck swelling, cervical lordosis and unilateral cervical lymphadenopathy (Figure 5). Examination of the posterior pharynx reveals a tender, asymmetric swelling and, sometimes, the presence of gas.

Radiographs may show characteristics soft tissue swelling between the posterior wall of the pharynx or trachea and the cervical vertebral bodies (Figure 6). The width of soft tissue should not exceed 7 mm at C2 and 22 mm at C6. Because exhalation can produce retropharyngeal bulging in normal children, radiographs made during inspiration are most appropriate for detecting retropharyngeal abscess. CT scanning is useful to gauge the extent of the abscess, which can be quite large (Figure 7).

Retropharyngeal space infection is most common in young children who have had an upper respiratory tract infection, when suppuration extends to retropharyngeal lymph nodes. Spontaneous onset without antecedent detectable infection has also been reported. The classic symptoms of neck stiffness and bulging of the posterior pharyngeal wall are present in less than 50 percent of patients. Stridor or airway obstruction was found in nine of 17 patients in one series; two of these patients had mistakenly been diagnosed as having epiglottitis.

Treatment of retropharyngeal space infection includes broad-spectrum antibiotics and drainage of suppuration. Complications include laryngeal edema with possible airway obstruction. Mediastinitis can develop from direct extension of the infection to the anterior or posterior portions of the superior mediastinum. Aspiration pneumonia develops in one-half of cases and results from either spontaneous rupture of the abscess into the airway or from
direct spread from the mediastinum. The mortality rate for retropharyngeal space infection in adults is 25 percent, even with treatment.

**Cervical Prevertebral Space Infection**

An abscess may develop in the potential space between the vertebral bodies and the prevertebral fascia. This infection usually results from osteomyelitis of the cervical spine. The clinical presentation includes tenderness over the cervical spine, with little swelling of the external neck. The pharynx is not swollen or is symmetrically swollen. Because the diagnosis is often delayed, complications are part of the spectrum of presentation.

Complications of cervical prevertebral space infection include spinal cord and nerve root compression, meningitis and spread of infection into the mediastinum. A lateral neck radiograph may show retropharyngeal swelling. Unlike retropharyngeal infection, cervical prevertebral space infection is most often caused by coagulase-positive staphylococci or *Mycobacterium tuberculosis*.

Cervical prevertebral space infection is treated with intravenous antibiotics that are effective against staphylococci. If retropharyngeal infection is suspected, treatment consists of intravenous antibiotics effective against the anaerobic and aerobic organisms commonly present in the mouth. Surgical drainage of the abscess if also indicated.

**Malignant External Otitis**

Malignant external otitis is a progressive form of external otitis caused by *Pseudomonas aeruginosa*, which is capable of penetrating the cartilaginous plates of the external canal and invading the soft tissue and bone (Figure 8). First reported in 1968, the infection was termed "malignant" because the mortality rate approached 50 percent. A review of 23 patients with malignant external otitis found that all were diabetic, all were over age 60 and all but one had *P. aeruginosa* as the sole pathogen. (One had both *P. aeruginosa* and a Serratia species as pathogens.) In one series of 13 patients, tap-water irrigation of the ears by medical personnel was found to precede the onset of symptoms in eight of the patients. It is possible that the forcible introduction of water caused a breakdown of the normal tissue defense against the invasion of *P. aeruginosa*.
Although elderly diabetic patients have the highest risk for malignant external otitis, the infection has been documented in other immunosuppressed patients and in poorly nourished infants. Unlike adults, children with malignant external otitis manifest facial neuropathy early in the course and are more likely to have tympanic membrane destruction, highlighting the need for prompt diagnosis and early treatment.

Figure 8. Invasion of Pseudomonas aeruginosa through the cartilaginous plates of the external auditory canal into the surrounding soft tissue.

The classic presentation of malignant external otitis includes a painful ear with purulent drainage from the canal. Granulation tissue forms at the anterior junction of the bony and cartilaginous portions of the canal. The infection does not respond to the usual local therapy for external otitis, because P. aeruginosa has
penetrated the epithelium of the external canal and invaded the underlying cartilage and bone. Prompt detection of malignant external otitis is extremely important. This infection should be considered in any diabetic or immunosuppressed patient who fails to respond to conventional therapy for external otitis.

Malignant external otitis may result in soft tissue or bone destruction. In one series, cranial nerve deficits due to invasion of the parapharyngeal spaces were found in 14 of 23 patients. The most common cranial nerve deficit is facial nerve paralysis, but multiple nerve deficits (usually glossopharyngeal and hypoglossal) are also common (Figure 9). Thus, patients with malignant external otitis typically present with loss of the nasolabial fold upon smiling, ipsilateral paralysis of the soft palate and ipsilateral deviation of the tongue. These deficits are occasionally present before granulation tissue develops.

Posterior invasion of *P. aeruginosa* leads to clouding of the mastoid air cells. With medial invasion, displacement and erosion of ossicles occur, while anterior invasion may produce temporomandibular joint arthritis and mandibular condyle osteomyelitis. Intracranial invasion may result in meningitis, brain abscess or cavernous sinus thrombosis.

Treatment of malignant external otitis includes the use of intravenous antibiotics that are effective against *P. aeruginosa*, control of blood glucose levels and careful debridement of infected tissue. Although most cranial neuropathies resolve with successful treatment of malignant external otitis, patients with progressive neuropathies may require referral. If symptoms of active infection persist after two weeks of treatment, referral for extensive surgical debridement may be necessary.

In one study, 21 of 23 patients with malignant external otitis were treated successfully with ciprofloxacin (Cipro), 750 mg twice daily, and local excision of the aural lesion. Treatment was given on an outpatient basis after a relatively short period of hospitalization (16.8 days), compared with 49 days of hospitalization for patients treated with intravenous gentamicin (Garamycin) and extended-spectrum penicillin.
Figure 9. View, from the base of the skull, of the invasion of malignant external otitis into the parapharyngeal space, with associated deficits in cranial nerves VII, IX and XII.

Diagnostic imaging can assist in detecting the spread of infection and in documenting the response to therapy. Plain radiographic films and tomograms are not sufficiently sensitive for the early detection of malignant external otitis. CT scanning may reveal abnormalities of the external auditory canal, destruction of the mastoid air cells and the presence of soft tissue or fluid in the middle ear and mastoid (Figure 10). Spread of infection to the parapharyngeal spaces can also occur (Figure 11). MRI may reveal more extensive soft tissue involvement than was suspected from the results of CT scans of the head. However, neither study is as effective as technetium scanning in revealing the spread of malignant external otitis to the base of the skull (Figure 12).

Since it is difficult to be certain about the presence of residual infection, determining the point at which treatment can be stopped is often a problem. Gallium scanning has been used to document eradication of infection, signaling a point at which treatment may be stopped.