Paparella: Volume III: Head and Neck

Section 2: Disorders of the Head and Neck

Part 7: The Neck

Chapter 43: Deep Neck Infections

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Formerly, infections of the deep fascial planes of the head and neck were fairly common and were a source of considerable morbidity and mortality. Mosher said of deep neck infections, "Pus in the neck calls for the surgeon's best judgement, his best skill and often for all of his courage". Although the advent of antibiotics has reduced the overall number of deep neck infections, they still occur in the general population, with a definite potential for significant morbidity, and even mortality, with improper or delayed treatment. Additionally, there are several new groups of patients at risk for deep neck infections, such as immunocompromised individuals recovering from organ transplants or chemotherapy, or those with acquired immunodeficiency syndrome (AIDS) and the increasing numbers of drug addicts who are injecting drugs into various areas of the neck. Prevention of the severe sequelae that may be associated with deep neck infections (such as mediastinitis, airway obstruction, carotid artery hemorrhage, or septicemia) requires a knowledge of the various portals of entry for infection, the presenting signs and symptoms, the possible microbiologic features, appropriate laboratory and radiologic workups, and the various treatment options. Additionally, proper treatment requires knowledge of the various fascial planes that infections travel along to assure complete and adequate drainage.

Relevant Anatomy of Deep Neck Infections

The Cervical Fasciae and Potential Compartments

One of the major considerations in the diagnosis and treatment of deep neck infections is the ability of infections to travel from one anatomic region to another via planes or potential spaces created by the various fasciae of the head and neck. This arrangement of multiple investing fasciae, and the ability of infection to travel along them, is somewhat unique in the body, and a thorough understanding of their anatomy and the potential spaces they create is essential to the effective treatment of these serious infections.

Fasciae of the Head and Neck

Discussion of the fasciae of the head and neck has given rise to controversy and confusion since their original description by Burns in 1811. Most textbooks of anatomy and surgery treat the cervical fasciae cursorily and, often, inaccurately. There are a variety of reasons for this inadequate treatment, including lack of interest, but a major factor is the number of disparate observations reported in the literature. Grodinsky, citing Malgaigne, noted "the cervical fasciae appear in a new form under the pen of each author who attempts to describe them". These disparities, when coupled with variations in terminology, have given rise to a large volume of distracting literature. The following description is an attempt to

instill, in a concise fashion, well-accepted concepts concerning the cervical fasciae.

The neck may be divided into two separate portions, the first and largest being the *cervical spine* with its attached musculature; the smaller portion lies anterior to the spine and has been termed the *visceral segment.* The spine and its musculature provide support for the head and protect the cervical portion of the spinal cord. The visceral portion of the neck contains all of the neck viscera, vasculature, nerves, and lymphatics. The cervical fasciae function to hold the visceral structures in their proper relationship and provide a gliding surface that allows the various segments to move in relation to one another. It is in the visceral portion that virtually all serious deep neck infections occur.

The cervical fasciae are generally described as being composed of a superficial and a deep layer.

The Superficial Cervical Fascia

The superficial cervical fascia is a sheet of fibrous connective tissue that encircles the head and neck with attachments to the fascia of the shoulders, thorax, and axilla. The superficial cervical fascia is relatively well defined over the lower portion of the face and lateral portion of the neck, whereas it becomes somewhat thinner more anteriorly. Posteriorly it is thick and adherent to deep fascia. The major structure contained within the superficial cervical fascia is the platysma muscle, which arises from the fascia overlying the superior portion of the chest musculature, traverses the anterolateral portion of the neck, crosses the mandible (with some fibers inserting to the bone), and ends in the lower face, with fibers inserting to the skin and blending with the lower muscles of facial expression. The innervation of the platysma muscle is through the cervical branch of the facial nerve. The marginal mandibular branch of the facial nerve lies just deep to the superficial cervical fascia, with its enclosed platysma muscle, in the potential space between the superficial fascia and the deep cervical fascia. Thus, simple elevation of the platysma muscle, as in the standard cervical flaps, will not protect the marginal mandibular nerve, and a portion of the superficial layer of the deep cervical fascia must be reflected to keep the marginal nerve out of harm's way. This potential space between the superficial cervical fascia and deep cervical fascia allows free movement of the skin and platysma muscle over the deeper structures of the neck. This space also has the potential to allow for spread of infection along its deep border, although in reality it rarely plays a significant role in serious infections of the neck.

The Deep Cervical Fascia

The deep cervical fascia is the most important determinant in the spread of cervical infections. For purposes of discussion, the deep cervical fascia is generally thought of as being composed of three sections: (1) the superficial or investing layer of the deep cervical fascia, (2) the middle or visceral fascia, and (3) the deep or prevertebral fascia. These various fasciae, with their interconnecting septa, create the potential spaces or compartments that tend to dictate the development and spread of cervical infections.

The Superficial Layer of the Deep Cervical Fascia. This completely envelops or invests the neck from the skull to the chest. It has been called the mother of the cervical fasciae, since all other major deep cervical fasciae develop as septum from it. The superficial

layer of the deep cervical fascia extends from the ligamentum nuchae and spines of the cervical vertebrae, moving anteriorly to completely enclose the neck. Its bony attachments are, superiorly, the external occipital protuberance, the mastoid process, and the zygoma (after surrounding the parotid gland); anteriorly, they are the mandible and the hyoid bone; inferiorly, they are the scapula, the clavicle, and the manubrium of the sternum.

Traced forward from its origin at the posterior midline, the superficial layer of the deep cervical fascia starts as a single layer, and upon reaching the trapezius it splits to encircle this muscle; it then fuses at the trapezius' anterior border and, once again, becomes a single layer. It then crosses the posterior triangle as a single layer and again splits to encircle the sternocleidomastoid muscle, fusing at the anterior border and continuing anteriorly to join the superficial layer of the deep cervical fascia of the opposite side. The two sides of the superficial layer of the deep cervical fascia fail to join in the midline for a distance of approximately 1 to 3 cm above the sternum, and this makes possible a space that has been termed the suprasternal space of Burns.

Anteroinferiorly, the superficial layer of the deep cervical fascia attaches to the clavicle and then continues superiorly, splitting to encircle the sternocleidomastoid muscle and attaching to the hyoid bone. From the hyoid bone, the investing fascia runs superiorly to the inferior border of the mandibular symphysis and the anterior portion of the mandibular ramus. Additionally, there are attachments between the superficial layer of the deep cervical fascia and sheaths of the anterior bellies of the digastric muscle. Because of this midline attachment of the superficial layer of the deep cervical fascia from the hyoid bone to the mandible and the anterior bellies of the digastric muscle, the investing fascia forms a floor for the submental region, creating the potential for a midline submental space. More laterally, the superficial layer of the deep cervical fascia extends from the greater cornu of the hyoid bone, splits to encircle the submandibular gland, and attaches to the lateral two thirds of the mandibular ramus. It should be noted that the posterolateral border of the submandibular gland curves medially and anteriorly around the posterior border of the mylohyoid muscle. Thus, the submandibular gland is in contact with the floor of the mouth and provides a potential route between the mouth and neck. After encircling the submandibular gland, the deep portion of the submandibular gland's capsule joins together with the superficial layer of the deep cervical fascia sheaths of the stylohyoid and posterior belly of the digastric muscle to develop a ligament that attaches from the posterior angle of the mandible to the styloid process, which is known as the stylomandibular ligament. After splitting to encircle the submandibular gland, the two layers of the investing fascia join again superiorly with attachments to the mandible. Additionally, a sheet continues superiorly to the edge of the parotid gland at which point it splits into superficial and deep layers investing the parotid gland. The superficial layer of this splitting becomes the capsule of the parotid gland and is commonly referred to as the parotidomasseteric fascia. This is a tough, thick layer that is firmly adherent to the parotid gland and sheath of the masseter muscle. The parotidomasseteric fascia terminates superiorly by attaching to the inferior border of the zygoma. At this point a continuation of the fascia extends superiorly as the outer layer of the temporalis muscle fascia. This layer then terminates at the lateral orbital margin and temporal line of the skull. The deep or posterior layer of the parotidomasseteric fascia forms the deep capsule of the parotid gland. This deeper layer continues anteriorly to cover the pterygoid muscles and then passes anterior to the mandible to fuse with the superficial layer. This fascial encircling of the mandible, with the parotid gland and masseter muscle laterally, and

the deep parotid gland and pterygoid muscles medially, creates two potential spaces known as the parotid and masticator spaces.

The Middle or Visceral Fascia of the Deep Cervical Fascia. This develops from extensions of the superficial layer of the deep cervical fascia and encircles the viscera of the neck - namely, the pharynx, esophagus, larynx, trachea, and thyroid gland. In addition, the visceral fascia encloses the strap muscles that attach to the hyoid bone and thyroid cartilage and the neurovascular structures associated with the carotid artery. The visceral fascia arises from the investing fascia at the deep surface of the sternocleidomastoid muscle. Initially, the numerous septae are thin and unsubstantial, but they thicken as they approach the structures they will encircle. This large space enclosed by the visceral fascia is important because of the potential communication of infections from the mouth, pharynx, esophagus, larynx, or trachea with the mediastinum.

The visceral layer of the deep cervical fascia has, in its turn, been divided into three layers: the middle cervical fascia, which encircles the strap muscles; the cervical visceral fascia, and the carotid sheath.

The Cervical Visceral Fascia of the Deep Cervical Fascia. This forms the capsule of the thyroid gland and encircles the pharynx, esophagus, larynx, and trachea. As such, it is continuous with its counterparts covering the trachea and esophagus in the mediastinum. Superiorly, it attaches to the base of the skull, the superior constrictor muscle of the pharynx, the pharyngeal aponeurosis, the mandible, and the pterygomandibular raphae. Also enclosed within this fascial segment is the thyroid gland.

The buccopharyngeal fascia is that portion of the cervical visceral fascia that covers the constrictor muscles of the pharynx and buccinator muscle, extending from the base of the skull to approximately the level of the cricoid cartilage.

The Carotid Sheath

The carotid sheath is a circular condensation of connective tissue running vertically in the neck extending from the skull base to the level of the clavicle. It is made up of contributions from the fascial coverings of the sternocleidomastoid muscle, strap muscles, and prevertebral fascia. Within the sheath are contained the carotid artery, vagus nerve, internal jugular vein, and cervical sympathetic chain posterior to the carotid artery. These individual structures are enclosed by their own separate sheaths within the main carotid sheath. However, only the carotid sheath has a significant separate covering of connective tissue. At the level of the clavicle, the carotid sheath fuses with the covering of the great vessels at the root of the neck and the pericardium. Thus, the carotid sheath is one of the main avenues for spread of infection from the neck into the chest or mediastinum.

The Deep Cervical Fascia or Prevertebral Fascia

The prevertebral fascia lies anterior to the cervical vertebral bodies medially and covers the paraspinous muscles (musculus longus colli, musculus longus capitis, scalene muscles) laterally. It extends laterally to the tips of the transverse processes of the cervical vertebrae. The following structures lie anterior to the prevertebral fascia: the carotid sheath

and its contents; the pharynx, hypopharynx, and esophagus; the laryngotracheal complex and strap muscles; the trapezius, sternocleidomastoid, and stylohyoid muscles; and the phrenic nerve.

Potential Spaces of the Neck

There are two types of fascial planes in the neck, those associated with muscles and those surrounding viscera and vessels. The muscular fascial planes always ultimately insert into bone and are therefore limited as to the extent an infection may progress. Conversely, the fascial planes associated with viscera and vessels allow much greater accessibility for infections to pass from one region to another and are associated with the most serious infections. Numerous potential spaces within the head and neck have been described and designated with a variety of confusing terms. Since the most important spaces are those associated with viscera, these will be the ones primarily considered in this discussion.

The Lateral Pharyngeal or Parapharyngeal Space

The lateral pharyngeal space has also been referred to as the pharyngomaxillary space. It consists of loose connective tissue lateral to the fascia covering the constrictor muscles of the pharynx (buccopharyngeal fascia), but medial to the pterygoid muscles, the mandible, and the carotid sheath. The lateral pharyngeal space extends superiorly to the base of the skull, and inferiorly it ends at the level of the hyoid bone because of the submandibular gland sheath's attachment to the sheaths of the stylohyoid muscle and posterior belly of the digastric muscle. The posterolateral wall of the lateral pharyngeal space contains the medial portion of the carotid sheath. Posteromedially, there is a potential communication with the retropharyngeal space. Anteriorly and inferiorly, the lateral pharyngeal space communicates with the spaces associated with the floor of the mouth. The medial wall of the lateral pharyngeal space if formed by the buccopharyngeal fascia lying on the constrictor muscles surrounding the tonsillar fossa. Because of its central location and multiple connections, the lateral pharyngeal space not only is the space most frequently involved with serious head and neck infections but also it allows the easiest access for infections to progress from one region to another, most significantly to the carotid sheath or retropharyngeal space. The most common routes by which the lateral pharyngeal space may become involved by infection are lingual connections and lymphatics, submandibular gland infections, infections of the masticator or parotid space, and spread from peritonsillar abscesses.

Submandibular Space

The submandibular space is actually composed of two spaces partially separated by the mylohyoid muscle. The space below the mylohyoid muscle is termed the *submaxillary space* and is often considered to have an anterior subunit referred to as the *submental space*, which is bounded by the anterior bellies of the digastric muscle laterally, the mandible superiorly, and the hyoid bone inferiorly. Thus, infection may readily spread from the main portion of the submaxillary space to the submental space and from there to the opposite side. The space located above the mylohyoid muscle is referred to as the *sublingual space* and consists of loose connective tissue surrounding the tongue and sublingual gland and, again, this space readily communicates with its counterpart on the opposite side.

The submandibular space as a whole is bounded by the oral mucosa and tongue superiorly and medially, the mandible superiorly, the superficial layer of the deep cervical fascia with its tight attachment to the mandible and hyoid bon laterally, and the hyoid bone inferiorly. The submandibular space, or one of its components, is perhaps the space most commonly involved by significant primary infections of the head and neck. Infections may arise from injuries to the floor of the mouth, sublingual or submandibular gland sialadenitis, or infections from the roots of the mandibular teeth.

Once an infection has entered a section of the submandibular space, there is ready communication between the submaxillary and sublingual subunits via the space between the mylohyoid and geniohyoid muscles referred to as the mylohyoid cleft. Also passing through the mylohyoid cleft are Wharton's duct, the lingual nerve, the hypoglossal nerve, a branch of the facial artery, and multiple lymphatics. Additionally, there is free communication across the midline from either the sublingual or submaxillary space. Ludwig's angina is a rapidly spreading cellulitis that usually begins in the submaxillary space, resulting from an infected molar, and then rapidly spreads to involve the sublingual space, usually on a bilateral basis. With the mandible and superficial layer of the deep cervical fascia presenting relatively unyielding barriers superiorly and laterally, the tongue is forced upward and posteriorly, giving rise to the severe airway obstruction associated with this condition. Submandibular space infections may also spread posteriorly to the carotid sheath or retropharyngeal space, or both, by crossing the lateral pharyngeal space.

The Masticator Space

The masticator space is formed by the splitting of the superficial layer of the deep cervical fascia as it encloses the mandible and primary muscles of mastication. The two portions of the fascia enclose a space containing the masseter muscle, the medial and lateral pterygoid muscles, the ramus and posterior portion of the body of the mandible, and the tendinous insertion of the temporalis muscle. The masticator space is enclosed at its borders by attachments of the cervical fascia to the zygoma, mandible, pterygoid muscles, and base of the skull, the exception being superomedially at which point it communicates with the temporal space medial to the zygomatic arch. Thus, infections may communicate freely between the masticator and temporal spaces. The most common cause of infection within the masticator space is from abscessed third molars.

Retropharyngeal Space

The retropharyngeal space lies between the deep layer of the deep cervical fascia (prevertebral fascia) and the buccopharyngeal fascia covering the constrictor muscles of the pharynx superiorly and the fascia covering the esophagus inferiorly. Some confusion has arisen regarding this space because of the presence of an ancillary portion of the deep cervical fascia referred to as the alar layer. This alar layer of the deep cervical fascia extends from the base of the skull to approximately the second thoracic vertebra at which point it fuses with the sheath of the esophagus. The more significant portion of the deep cervical fascia is the prevertebral fascia, which lies over the vertebra and paraspinous muscles and runs from the base of the skull to the diaphragm. Thus, there are two potential spaces in the retropharyngeal space, and there is considerable variation in the terminology describing them. The first space lies between the fascia covering the pharynx and esophagus and the alar layer of deep cervical

fascia. This is commonly referred to as the retropharyngeal or retrovisceral space. As mentioned, this space ends at approximately the second thoracic vertebra because of the fusing of the alar fascia and esophageal fascia. This is the space most commonly involved by retropharyngeal abscesses. Lying posterior to the alar fascia, but anterior to the prevertebral fascia, is the danger space (so named by Grodinsky) or prevertebral space. Although less commonly involved than the retropharyngeal space, it offers the opportunity for much wider spread of infection as space extends to the level of the diaphragm, and thus it has a much greater capacity to allow extension of infections to the mediastinum. The danger space is most commonly involved by rupture of retropharyngeal space abscesses posteriorly, which then spread to the mediastinum.

The retropharyngeal space is readily involved by progression of infections from lateral pharyngeal space infections, masticator space infections, and parotid space infections (via the lateral pharyngeal space), as well as primary infections from abscessed lymph nodes or perforations secondary to foreign bodies or endoscopy. It has been estimated that 71 per cent of head and neck infections involving the mediastinum spread via the retropharyngeal space of the danger space, or both. Thus, the retropharyngeal space is one of the major routes of spread for infections from the head and neck into the mediastinum.

Parotid Space

The parotid space lies between the superficial and deep capsules of the parotid gland, which are formed by the splitting of the superficial layer of the deep cervical fascia. The superficial capsule is quite thick and strong and is closely adherent to the underlying parotid gland. In addition, there are multiple septae running from the lateral capsule into the gland itself, thereby establishing numerous intraglandular compartments. The medial capsule of the parotid gland is quite thin and abuts the lateral pharyngeal space. Infections within the parotid gland tend not to pierce the tough lateral capsule; instead, they present medially with easy access to the lateral pharyngeal space. From the lateral pharyngeal space, infection may progress to the retropharyngeal space or carotid sheath and thus to the mediastinum. This is one of the feared complications of suppurative parotiditis.

Etiology and Pathogenesis of Deep Neck Infections

The etiology of deep neck infections varies depending on the space involved. Historically, in the preantibiotic era, the overwhelming preponderance of deep neck infections resulted from infections in the pharynx and tonsils, with approximately 70 per cent of cases resulting from these sources. Currently, this figure is much lower as a result of early antibiotic usage. Infections of dental origin are now a major source of deep neck infections, although Wright reports that in more than 50 per cent of reported deep neck infections no specific source of infection can be found.

Possible portals of entry are multiple and will be discussed individually in order to promote an understanding of the possible pathways of extension into the neck and the pathophysiology involved (Table 1). Understanding the processes and pathways involved in the origination of deep neck infections enables one to better anticipate and recognize potential complications and will also facilitate formulation of a logical treatment plan.

As noted, infections of dental origin are a major source for deep neck infections in the modern era. Spread of dental infection may result in involvement of the sublingual and submaxillary spaces, with resultant Ludwig's angina; involvement of the masticator space, with possible subsequent extension to the lateral pharyngeal space or infratemporal fossa; or involvement of the pterygopalatine fossa. Subsequent extension to the carotid sheath or retropharyngeal space may result in mediastinal spread. These infections are usually of mixed microbiologic flora and include alpha- and beta-streptococci, *Staphylococcus, Peptostreptococcus, Fusobacterium nucleatum, Bacteroides melaninogenicus, B. oralis, Veillonella, Actinomyces, Spirochaeta* and, more recently, *Eikenella corrodens.*

Table 1. Portals of Entry in the Pathogenesis of Deep Neck Infections

Dental infection
Peritonsillar cellulitis or abscess
Upper aerodigestive tract trauma
Retropharyngeal lymphadenitis
Pott's disease
Sialadenitis
Bezold's apicitis
Congenital cysts and fistulas
Intravenous-subcutaneous drug injection.

Extension of tonsillar and peritonsillar infections, though less likely now than in the preantibiotic era, is still one of the more common causes of deep neck infection. Peritonsillar abscess or cellulitis is common, usually of streptococcal origin, and may penetrate the buccopharyngeal fascia directly or extend via retrograde thrombophlebitis to involve the lateral pharyngeal space. The carotid sheath, which pierces the cone-shaped lateral pharyngeal space at its apex, may become involved and provides a dangerous conduit for infection of the mediastinum. Mosher named this potential avenue of infection the "Lincoln Highway" of the neck. Perhaps equally important, however, in the pathogenesis of the mediastinal involvement from infection of the lateral pharyngeal space is the weak fascial attachment posteriorly between the lateral and retropharyngeal spaces. This weak attachment is readily compromised and permits relatively easy spread from the lateral pharyngeal space to the retropharyngeal space and, hence, to the mediastinum.

Trauma of the upper aerodigestive tract by blunt, penetrating, or iatrogenic means is not an uncommon source of deep neck infection. The spaces involved are directly related to the region traumatized, the lateral pharyngeal and retropharyngeal spaces being the most frequently involved.

Retropharyngeal lymph nodes are distributed in two chains on either side of the midline and drain the nose and paranasal sinuses, pharynx, middle ear, eustachian tube, adjacent muscle, and bone. These nodes are found in greater numbers in children and may become abscessed secondary to draining a primary site of infection. These abscessed nodes may then go on to establish a retropharyngeal abscess. This was once thought to be essentially a disease of infancy, but more recent evidence suggests that retropharyngeal abscess may occur more commonly in adults than previously thought, especially in patients with compromised immune systems. *Streptococcus pyogenes, Staphylococcus oralis*, and anaerobic

bacteria are the most commonly implicated organisms, but several studies emphasize the polymicrobial nature of these infections. Retropharyngeal space abscesses may break through the alar fascia and involve the prevertebral space with early egress to the mediastinum.

Primary prevertebral space involvement may result from cervical vertebral body infection or necrosis. This is most commonly the result of Pott's disease from tuberculosis and is classically described as occurring in the midline through disruption of the prevertebral fascia.

Sialadenitis, usually of staphylococcal or streptococcal origin, may serve as a source for neck infection. A suppurative process involving the submandibular gland may extend to involve the submandibular space and further extend to involve the lateral pharyngeal space. Similarly, parotitis may extend to secondarily involve the masticator space or lateral pharyngeal space, the latter being of particular importance because of the thin fascial covering of the posteroinferior portion of the deep lobe of the parotid gland.

The temporal bone is an often-forgotten potential portal of entry into the lateral pharyngeal space. In addition to the well-described Bezold's abscess, which usually stems from an infection in the cells near the mastoid tip, infection involving the petrous pyramid may secondarily involve the neck via the lateral pharyngeal space. Although unusual, this potential route of spread should be kept in mind when patients exhibit signs of petrous apicitis.

Infections of congenital cysts and fistulas may also result in deep neck infections if an inflammatory process extends to surrounding fascial compartments. Foregut anomalies, bronchogenic cysts, thyroid cysts, laryngoceles, thyroglossal duct cysts, and branchial arch anomalies must all be considered as potential sources of deep neck infection. Additionally, it should be remembered that certain of these anomalies primarily involve the mediastinum, and neck involvement may occur secondarily. Occult congenital cysts probably account for more deep neck infections then is generally recognized.

Recently, more and more reports of deep neck infections from injection of illicit drugs have been appearing. These infections usually occur in the supraclavicular region and may result in an infection of the carotid sheath with possible mediastinal extension. The most commonly reported organisms are *Staphylococcus aureus* and beta-haemolytic streptococci. Additionally, the possibility of injury to the carotid artery or innominate artery with a coincident pseudoaneurysm formation should also be kept in mind, and appropriate radiologic evaluation should be obtained if drainage is contemplated.

Finally, it should be remembered that suppuration of the deep cervical lymphatics and subsequent deep neck infection may occur without an obvious primary source being identified.

Clinical Manifestations

The clinical presentation of deep neck infections will vary depending upon the space involved, and they are discussed in depth further on in the section detailing specific neck space infections; however, several general considerations should be kept in mind. The symptoms that occur most regularly in deep neck infections are pain, fever, and limitation of

mandibular and neck motion.

Pain is a fairly constant finding in deep neck infections and usually gives a good idea of the primary source of infection and, additionally, it may be used as an indication of extension or resolution of the infectious process. The major exception to the prognostic value of pain in neck infections is in the case of retropharyngeal abscess in children. In this circumstance, the onset of an abscess may be quite insidious, with irritability, decreased appetite and low-grade fever being the only early signs of this potentially serious infection.

Fever is also a constant finding in deep neck infections, with the typical case being that of an initial temperature spike, after which the temperature usually remains elevated. Spiking temperatures often indicate episodes of septicemia and should raise concerns about the possibility of a septic thrombophlebitis of the internal jugular vein or mediastinal extension.

Trismus and limitation of neck motion are usually present in deep neck infections and are accompanied by variable degrees of swelling. In some cases, this swelling may actually progress to a pitting edema. Because of the fact that these infections involve deep neck spaces, it is rare to find discrete areas of fluctuance, the possible exception being the submandibular space. The amount of trismus and neck swelling will, of course, vary depending upon the site involved. For instance, with masticator space involvement, external neck swelling may be minimal, but there will be significant trismus. Conversely, a retropharyngeal abscess may result in minimal, if any, trismus and no external swelling, but it is usually associated with marked limitation of neck motion.

Other symptoms that the clinician should be aware of include those of progressive dysphagia and odynophagia, change in voice, and dyspnea. Change in voice may be particularly evident in lateral pharyngeal and retropharyngeal involvement with the production of the so-called hot potato voice. Dyspnea may occur from airway encroachment caused by tongue swelling in Ludwig's angina or from pharyngeal swelling in lateral pharyngeal and retropharyngeal space abscesses. Dyspnea is a late finding, indicating impending airway obstruction, and steps should be taken to secure the airway immediately. Finally, mediastinal extension is usually heralded by chest pain, severe dyspnea, and radiographic evidence of a widened mediastinum (Table 2).

Table 2. Possible Complications of Deep Neck Infections

Hemorrhage from necrosis of carotid wall - oral or aural
Internal jugular vein thrombosis with septic thrombophlebitis
Empyema
Mediastinitis
Asphyxia - secondary to floor of the mouth and tongue swelling or airway encroachment from lateral pharyngeal or retropharyngeal swelling
Defects of cranial nerves IX, X, XI, XII, Horner's syndrome.

General Workup

A careful history and complete physical examination are important in the workup of patients with deep neck infections. The history may prove invaluable in eliciting information that may lead to discovery of a previously unsuspected portal of entry. Likewise, a careful physical examination is essential for determining the portal of entry and the involved neck space or spaces and to raise concerns about the development of complications. This is especially important with children, in whom a reliable history may be unavailable.

The initial laboratory examination should consist of a complete blood count with differential, erythrocyte sedimentation rate, serum electrolyte levels, calcium level, coagulation parameters, and blood cultures if there is evidence of sepsis. Cultures with a Gram stain evaluation should be taken from any suspicious source of infection. Radiographic examination should be tailored to the individual patient and should include examination of the affected areas. Lateral neck films may be useful for demonstrating encroachment of the airway by floor of the mouth infections (Ludwig's angina) or retropharyngeal abscess, in addition to demonstrating the presence of air. Also, they may demonstrate a widened retropharyngeal space, which is suggestive of abscess formation or cellulitis. Noninvasive techniques such as CT scan, and more recently magnetic resonance imaging (MRI), have proved to be quite helpful in delineating the site and extent of involvement of infections and in differentiating cellulitis from abscess formation. When performing a CT scan, it is suggested that contrast enhancement is employed to determine the relationship of the infectious process to the cervical vasculature, and, additionally, it may demonstrate whether or not the internal jugular vein is patent. Arteriography should be considered in patients with deep neck infections caused by injecting drugs into the root of the neck to rule out the presence of a pseudoaneurysm secondary to injury to the innominate artery or the carotid artery.

Specific Deep Neck Infections

Ludwig's Angina

The disease process referred to as Ludwig's angina was formally described in 1836 by Wilhelm Friedrich von Ludwig when he reported on five patients in whom a rapidly progressive soft tissue infection developed, involving the submandibular space and floor of the mouth. Several of these patients died of acute airway obstruction and asphyxiation. Although his original description accurately described the disease process and relevant findings, he erroneously felt that he was dealing with a new form of neck inflammation that was infectious and occurring in an epidemic fashion. Ludwig's angina, with its potential for airway obstruction, has probably been known since antiquity with references by Hippocrates, Galen, Caelsius, and others under a variety of terms such as *cyanche, carbunculus gangraenosus, morbus strangulatorius,* and *angina maligna.*

Anatomy

Ludwig's angina is an infection of the submandibular space. As previously described, the submandibular space is composed of two spaces: the sublingual space and the submaxillary space. The sublingual and submaxillary spaces are separated by the mylohyoid muscle and are connected via the mylohyoid cleft (containing the tail of the submandibular

gland, Wharton's duct, lingual nerve, hypoglossal nerve, lymphatics, and several arteries and veins). The floor of the submandibular space is formed by the superficial layer of the deep cervical fascia attaching from the hyoid bone to the mandible. There is ready communication across the midline with the opposite sublingual or submaxillary spaces.

As will be seen, the majority of cases of Ludwig's angina are of dental origin, with the development of a submaxillary infection that then progresses to involve the sublingual space. Crucial to this process is the relationship of the mandibular dentition to the attachment of the mylohyoid muscle along the mylohyoid ridge. The anterior teeth and first molars regularly attach superior to the mylohyoid insertion, and infection arising from these tooth roots commonly results in a relatively limited sublingual abscess. The second and third molar roots are routinely below the mylohyoid ridge, and infection presenting on the lingual surface will enter the submaxillary space. Additionally, it is important to recognize that the roots of the anterior teeth and first molar approximate the lateral mandibular surface, whereas the second and third molar roots approach the lingual surface of the mandible.

Definition

Since Ludwig's original description, a variety of inflammatory conditions related to the floor of the mouth have been labeled Ludwig's angina (Table 3), but in reality many would most appropriately be termed *pseudoangina ludovici.* These pseudo-Ludwig's angina are more limited infections that involve only the sublingual space, the submandibular lymph nodes, the submandibular gland, or the submental space, or they are abscesses involving one or more of these spaces. Ludwig's original description was of a disease process that was a "gangraenous 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". There is a general consensus in the literature that to qualify as a true Ludwig's angina, the following features should be present: (1) a spreading cellulitis with no specific tendency to form abscesses, (2) involvement of both submaxillary and sublingual spaces, usually bilaterally, (3) spread by direct extension along fascial planes and not lymphatics, (4) involvement of muscle and fascia but not submandibular gland or lymph nodes, and (5) origination in the region of the submaxillary space with progression to involve the sublingual space and floor of the mouth.

Table 3. Criteria for Diagnosis of Ludwig's Angina

Rapidly progressive cellulitis; not an abscess
Develops along fascial planes with direct extension; does not involve lymphatic spread
Does not involve submandibular gland or lymph nodes
Involves both sublingual and submaxillary spaces and is usually bilateral.

Etiology

Ludwig's angina occurs most commonly in young adults with periodontal disease. Formerly, Ludwig's angina had been ascribed to an infectious source with an epidemic occurrence. However, it now appears that the reported occurrences of clusters of Ludwig's angina are most likely the result of pseudo-Ludwig's anginas secondary to suppurative lymphadenitis in cases of scarlet fever, diphtheria, or measles. Tschiassny, in an extensive

review of the literature and detailed anatomic studies, demonstrated that the majority of Ludwig's angina cases are the result of abscesses second or third molar teeth with penetration into the submaxillary space below the mylohyoid ridge and initiation of a cellulitis. He found that a dental cause was responsible in 75 to 80 per cent of cases, followed by penetrating injury of the floor of the mouth (stab wound, gunshot wound, horse kick, and so on), with mandibular fractures accounting for the rest of the cases. It is interesting to note that extraction of a diseased molar will often initiate the infection, and Tschiassny speculated that the injection of local anaesthetic may seed the submandibular space. Patterson noted that 85 per cent of their cases were related to a dental pathological condition, with 40 per cent of the cases following extraction of a diseased molar. The remaining cases were in children younger than 3 years of age in whom no clear cause could be demonstrated.

Microbiological Features

Since most cases of Ludwig's angina are related to a dental cause, it is not surprising that the bacterial cultures mirror the oral flora. The compilation of accurate microbiologic data has been complicated by the fact that most patients have had at least several doses of antibiotics by the time surgical treatment is performed and the additional fact that a number of infections resolve with conservative treatment and no surgical drainage and, thus, no specimen is obtained. It is clear that these infections are almost always the result of mixed flora, involving both aerobes and anaerobes. The most commonly reported aerobes are alpha-hemolytic streptococci, followed by *Staphylococcus.* The data on anaerobic cultures are more difficult to interpret because of the presence of prior antibiotic therapy and the difficulty in culturing anaerobic organisms. The normal oral flora typically contains *Peptostreptococcus, Peptococcus, Fusobacterium nucleatum, Bacteroides melanogenicus, B. oralis, Veillonella,* and *Spirochaeta.* This combination of aerobic and anaerobic organisms gives rise to a synergistic effect caused by the production of endotoxins such as collagenase, hyaluronidase, and proteases, the combination of which promotes a rapidly progressive infection with the clinical features of tissue necrosis, local thrombophlebitis, putrid odor, and gas formation. As a rule, gram-negative organisms do not play a significant role in Ludwig's angina, although *Haemophilus influenza, Escherichia coli,* and *Pseudomonas* have all been reported.

Clinical Features

The clinical picture of Ludwig's angina is typically that of a young man with poor dentition that presents with a history of increasing oral or neck pain and swelling. Frequently, the patient's symptoms are unilateral, but they soon progress to involve both sides. As the infection progresses, there is increasing edema and induration of both the external perimandibular region and the floor of the mouth. The soft tissues of the floor of the mouth become tremendously swollen and, as a result of the relatively unyielding superficial layer of the deep cervical fascia and mandible, the expansion progresses superiorly and posteriorly. This expansion has the effect of thrusting the tongue posteriorly and superiorly with approximation of the palatal vault. There is increasing neck rigidity, trismus, odynophagia and, eventually, drooling. The temperature is typically elevated in the 100 to 102 °F range, and 104 °F is not uncommon. As the swelling progresses, there is increasing encroachment upon the airway, and the patient assumes an erect posture with tachypnoea. Dyspnea and stridor signal the imminent danger of airway obstruction. This is truly a desperate situation because visualization of the larynx by conventional techniques is impossible, and attempts at

intubation may actually precipitate airway obstruction. Similarly, a tracheostomy is extremely difficult because of the inability of the patient to lie supine and the presence of significant neck edema. The point to stress is the rapidity with which this process may occur; many case reports detail a progression from onset of symptoms to respiratory obstruction within the space of 12 to 24 hours.

Physical Examination

The physical examination typically shows fever, tachycardia, and variable degrees of respiratory obstruction with dysphagia and drooling. The submandibular and submental regions are tense, swollen, and tender. The floor of the mouth is tense and indurated, with massive mucosal swelling and pouting of soft tissue over the edges of the lower teeth. Fluctuance is unusual. The tongue is pushed superiorly, and there is marked trismus. Indirect examination of the larynx is, at best, difficult and usually impossible. Nasopharyngeal fiberoptic examination will reveal the presence of a greatly enlarged base of the tongue pushing the epiglottis posteriorly; the supraglottis and endolarynx are normal in appearance.

Laboratory and Radiologic Examination

The diagnosis and treatment of Ludwig's angina are based on the clinical parameters outlined; laboratory and radiologic studies offer only supporting evidence. Typically, the white blood cell count is moderately to markedly elevated in the range of 15,000 to 20,000, with a marked shift to the left. X-ray studies will show soft tissue edema, occasionally gas, and posterior displacement of the tongue with airway encroachment. Because of the fact that these infections are often odontogenic, panoramic tomography may be helpful in determining the site of origin for the infection and may aid in planning the treatment regimen.

Treatment

Treatment of Ludwig's angina depends on the stage of disease when the patient presents. Actually, Ludwig's angina is a spectrum of conditions, ranging from a periapical abscess and mild cellulitis in its early stage to massive sepsis with airway obstruction later on. Treatment may be tailored to the status of the individual patient, with the understanding that this disease may progress very rapidly, and if initial steps are not successful, more radical means must be employed. In the early stages of Ludwig's angina, with unilateral mild swelling and edema, simple intravenous antibiotics and supportive measures may be sufficient. This is often coupled with extraction of the inciting tooth, if it is identifiable. If the patient presents with more advanced disease or the disease progresses to bilateral swelling, dysphagia with drooling, or any symptoms of airway compromise, early airway intervention, in a controlled fashion, is advocated rather than waiting until emergency procedures are required. Surgical treatment is directed at securing an airway and providing surgical drainage. The choice of airway management rests on the experience and availability of the treating personnel. Although airway management by nasotracheal intubation, with or without fiberoptic assistance, is well described in the literature, it should not be borne in mind that airway manipulation may precipitate acute obstruction and, thus, a tracheostomy set should always be available. Because of the possibility of postoperative extubation, and the difficulty of reintubation, conversion to a tracheostomy is generally advocated for postoperative airway management. If intubation is not possible, a tracheostomy under local anaesthesia, often with

the patient sitting upright, is required. This may be complicated by the presence of significant edema in the neck. Those patients with a rapidly deteriorating airway may require a cricothyroidotomy.

Once the airway is established, surgical drainage is performed. Drainage consists of a wide surgical decompression of the suprahyoid region. Generally, the infectious process is bilateral, and the approach is through a median, horizontal incision three to four fingerbreadths below the mandibular margin. The length of the incision may be variable, but generally it crosses to the submandibular region bilaterally. The mylohyoid muscle is split in the midline, and drainage is established both medially and laterally. Often, the side on which the infection started needs to be explored with decompression of the submandibular capsule and blunt dissection to the mandibular margin. The tissues have been described as having a peculiar "salt pork" appearance, with woody induration, watery edema, and little bleeding. Gross purulence is rarely encountered at the time of exploration but will often drain from the wound several days after decompression. Multiple drains are placed, and the wounds are left open.

The choice of antibiotics must be tailored to the individual patient, but high-dose penicillin (12 to 16 million units/day) is considered the drug of choice. Chloramphenicol and clindamycin are alternate possibilities, especially in penicillin-allergic patients. In immunocompromised patients, consideration should be given to providing broader coverage for the possibility of gram-negative anaerobic organisms and penicillin-resistant staphylococci.

Complications

Ludwig's original description reported a 60 per cent mortality rate, whereas Patterson's recent report had a 0 per cent mortality rate. The major cause of death in Ludwig's angina is acute airway obstruction, and the most effective means of preventing it include careful monitoring of the patient with intervention at the earliest sign of airway compromise. Tracheostomy (either initially or after intubation) is generally considered the safest means of maintaining an airway. Following acute airway obstruction, the next major potential complication of Ludwig's angina is extension of the infection to the carotid sheath or retropharyngeal space with inferior extension into the mediastinum. Although extension to the mediastinum was formerly common, it is somewhat unusual with present-day surgical drainage and antibiotics. Extension of Ludwig's angina should be suspected if there is persistent or increasing neck edema, spiking temperatures, or persistent leukocytosis. Later findings of mediastinal extension include increasing signs of septic shock with tachycardia and decreasing blood pressure, crepitation of the lower neck, or development of mediastinal crepitation or a mediastinal crunch. The patient with mediastinitis will often report increasing neck pain, chest tightness, and increasing dyspnea. The chest x-ray film may show a widened mediastinum, pericardial air, pulmonary infiltrates, and extrapleural fluid. As mentioned, in the acute phase of Ludwig's angina, there is little or no purulence because of the fact that the infection is developing so rapidly that there is no time for pus to develop; later it is common for purulence to drain from the wounds. It is also possible that isolated, undrained pockets of pus may develop, which may give rise to continuing signs of sepsis. A CT scan of the neck with contrast medium may be most helpful in detecting these residual pockets and directing surgical drainage.

Lateral Pharyngeal Space Infections

The shape created by a lateral pharyngeal space abscess approximates an inverted pyramid, its base lying superiorly on the petrous portion of the temporal bone and its apex lying inferiorly at about the level of the hyoid bone. The carotid sheath passes through the lateral pharyngeal space posteriorly and inferiorly and serves as an important potential vehicle for spread of infection inferiorly into the neck and, possibly, the mediastinum.

The lateral pharyngeal space is divided by the styloid process into anterior and posterior compartments, the posterior compartment containing the internal carotid artery, the internal jugular vain, cranial nerves IX, X, and XII, and the cervical sympathetic trunk. The parotid, masticator, submandibular, and retropharyngeal spaces are all intimately associated with the lateral pharyngeal space, and they may all serve as potential areas of primary or secondary involvement in lateral pharyngeal space infections.

According to Beck, approximately 50 per cent of deep neck infections occurred in the lateral pharyngeal space prior to the advent of chemotherapy, peritonsillar infections being the most common source. This figure, according to the same author, has been reduced to less than 30 per cent since the advent of antibiotics.

As with most soft tissue infections of the neck, lateral pharyngeal space infections are polymicrobial and usually reflect oropharyngeal flora. In the immunocompromised host, the infecting organisms may be unusual, and it is recommended that broad antibiotic coverage be instituted until culture results are available.

Clinically, the patient with a lateral pharyngeal space infection presents with fever, trismus, and perimandibular edema involving the parotid and submandibular regions. Pain is usually marked, and neck motion is limited. Trismus is usually severe; however, it may be nonexistent in cases involving only the posterior compartment of the space. Intraoral examination reveals swelling of the lateral pharyngeal wall, especially behind the posterior tonsillar pillar. Tonsillar displacement anteriorly and medially is the rule. Neurologic deficits involving cranial nerves IX, X, and XII may occur. A Horner's syndrome is also possible secondary to involvement of the cervical sympathetic chain. Hemorrhage through the external ear, though rare, has been reported with lateral pharyngeal abscess. Presumably, this occurs as the result of an expanding hematoma dissecting along the skull base and entering the external ear canal via the fissures of Santorini. This severe symptom usually mandates immediate surgical exploration with ligation of the carotid artery.

Initial treatment of lateral pharyngeal space infections should be with aggressive antibiotic therapy, fluid replacement, and close observation. Antibiotic therapy usually consists of high-dose penicillin, with appropriate modifications based on the clinical situation and culture results; often, an aminoglycoside will be added for broader coverage.

Surgical intervention will often be required to bring about resolution of these infections. In fact, Levit states that only 10 to 15 per cent of all patients with lateral pharyngeal space infections will be cured with medical management only. Clearly, there should be little doubt as to the need for immediate surgical treatment in those cases in which there is hemorrhage, subcutaneous emphysema, neurologic deficit, skin necrosis overlying an

area of cellulitis, dyspnea, severe sepsis suggestive of internal jugular vein thrombosis, or evidence of abscess formation on CT or MRI scan. In instances in which none of these clear-cut indications pertains, a decision based upon close follow-up of the patient's clinical course is required. In general, persistent or worsening symptoms after 24 to 48 hours of appropriate antibiotic therapy warrants surgical intervention.

Surgical approaches to the lateral pharyngeal space may be either intraoral or external. Intraoral approaches should be confined to treatment of peritonsillar abscesses and should not be used in true lateral pharyngeal space abscesses because of inadequate exposure if severe bleeding arises. The external approach to the lateral pharyngeal space usually consists of a transverse submandibular incision approximately two and one-half fingerbreadths inferior to the mandibular margin, which extends from the anterior limits of the submandibular gland to just past the angle of the mandible. After sharp incision of the superficial layer of the deep cervical fascia overlying the submandibular gland, the gland is freed inferiorly and posteriorly by sharp and blunt dissection. Access to the lateral pharyngeal space is achieved by dissection between the tail of the submandibular gland and the anterior border of the sternocleidomastoid muscle, passing medial to the mandible and medial to the internal pterygoid muscle. Using this approach, ascent to the base of the skull can be easily achieved, if necessary. In 1929, Mosher described a T-shaped incision for a submandibular fossa approach to the lateral pharyngeal space and carotid sheath. This incision incorporates a vertical limb at the anterior border of the sternocleidomastoid muscle in addition to the submandibular transverse incision. Easy access to the carotid sheath or visceral space can then be obtained. Use of this incision, or a variation, is highly desirable when control of the carotid artery may be necessary because of previous aural or oropharyngeal hemorrhage.

Formerly, complications of lateral pharyngeal space infection were fairly common; at present they are much rarer but are still occasionally reported. Airway compromise is possible secondary to encroachment by pharyngeal edema, and one should move to secure the airway at the first sign of respiratory distress. Internal jugular vein thrombosis occasionally occurs and may result in septic emboli with pulmonary emboli, pneumonia, emphysema, bacterial endocarditis, or uncontrolled septicemia. Under these circumstances, ligation of the internal jugular vein may be indicated. Mediastinitis may also occur as a complication of lateral pharyngeal space infection from spread along the carotid sheath, and it carries an extremely high mortality rate. The most frequent fatal complication of lateral pharyngeal space infection is carotid artery hemorrhage resulting from necrosis of the artery wall secondary to a surrounding abscess cavity. Bleeding occurs most commonly from an oropharyngeal location and usually involves the internal carotid artery. The oropharyngeal bleeding usually occurs through the site used to attempt transoral drainage of a suspected peritonsillar abscess. Rarely, bleeding may occur from the ipsilateral ear secondary to dissection of the hematoma along the skull base. At the first sign of significant hemorrhage, immediate surgical exploration of the carotid sheath and common carotid artery. If no obvious site of bleeding from the common or external carotid artery is found, the presumed site is from the internal carotid artery in the lateral pharyngeal space. Because of the difficulty in exposing and controlling the internal carotid artery in this location, ligation of the common carotid artery is generally recommended. This will usually suffice to control bleeding unless contralateral retrograde circulation through the external carotid artery is sufficient to cause persistent serious hemorrhage. In these cases, ligation of the external carotid artery at the bifurcation and packing of the lateral pharyngeal space may be necessary.

Retropharyngeal Space Abscess

Historically, retropharyngeal space abscesses have largely been considered to be a disease of infancy, usually resulting from abscessed lymph nodes draining infections of the ears, nose, or throat. More recently, however, Barratt and associates have suggested that the incidence of this entity may be higher in the adult population than previously thought. In addition to spread from acute infection of the ears, nose, and throat, retropharyngeal abscesses may result from regional trauma, such as foreign body ingestion, oral endotracheal intubation, and endoscopic procedures.

The clinical presentation of retropharyngeal abscesses differs between children and adults. Generally, children will present initially with nonspecific signs such as irritability, poor appetite, and fever. As the infection progresses, neck rigidity and tenderness become apparent. Posterior pharyngeal swelling, if left untreated, will result in a progressively muffled cry, drooling, and respiratory compromise. In adults, initial signs and symptoms are more localized and include fever, sore throat, odynophagia, and neck tenderness. Dyspnea, though less likely than in the pediatric population, may still occur. Abscesses in both groups will form lateral to the midline secondary to the midline adherence of the alar fascia to the prevertebral fascia. This may be distinguished from Pott's abscess or nontuberculous prevertebral abscesses, which tend to occur in the midline.

Radiologic evaluation of patients suspected of having retropharyngeal abscesses may be invaluable. The single most important study remains the lateral x-ray film of the neck. Wholey and colleagues have demonstrated that a retropharyngeal space measuring 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, are suggestive of a pathologic process. It should be remembered that the infant's upper aerodigestive tract may vary considerably with inspiration and expiration because of increased pliability, the tissues being thinnest during expiration. Therefore, if any doubt exists after a lateral neck x-ray film, fluoroscopy may be helpful in evaluating the retropharyngeal space during all phases of the respiratory cycle. Another useful sign in patients with retropharyngeal abscesses is loss of the normal cervical spine lordosis or curvature with straightening of the cervical vertebral column. CT scan, MRI scan, and ultrasonography may also be helpful, but usually they are not necessary. After radiographic identification of a retropharyngeal abscess, a chest x-ray study should be obtained to rule out possible mediastinal involvement.

Initial treatment of a retropharyngeal abscess should consist of antibiotics and fluid replacement. Most recent studies emphasize the polymicrobial nature of retropharyngeal infections, which, again, largely reflects the oropharyngeal flora. In light of this, high-dose penicillin is generally recommended as the initial drug of choice in an uncomplicated or nonimmunocompromised host. Once it becomes clear that a retropharyngeal abscess is present, surgical drainage is indicated.

Surgical drainage of the retropharyngeal space may be accomplished either intraorally or externally. In those cases in which retropharyngeal involvement appears relatively localized to the pharynx and in which there is no evidence of respiratory obstruction, abscess drainage can be performed by transoral incision of the posterior pharyngeal wall with the patient in the Rose position (head down) to avoid aspiration. When a retropharyngeal abscess has

progressed to the extent that it is causing airway obstruction, or if there is significant inferior extension, an anterior cervical approach as described by Dean is preferable. In this approach, an incision is made along the anterior border of the sternocleidomastoid muscle from the level of the hyoid bone to the inferior level of the cricoid cartilage. After incising the superficial layer of the deep cervical fascia, the sternocleidomastoid muscle is identified and retracted laterally. The carotid sheath is identified and retracted posteriorly, the dissection continues medial to the carotid sheath and lateral to the trachea. After complete removal of purulent material, a drain is placed. If mediastinal extension is identified, thoracic surgical consultation may be required. In most cases in which respiratory distress is noted, a tracheostomy under local anesthesia is indicated because of the difficulty with laryngeal intubation and the possibility of actually rupturing the abscess with flooding of the airway. Additionally, undrained retropharyngeal abscesses may rupture spontaneously into the pharynx and result in pneumonia or lung abscess secondary to aspiration. Retropharyngeal space infections may extend posteriorly through the alar fascia into the danger space and from there easily progress into the mediastinum.

Masticator Space Infection

The masticator space is anterior and lateral to the lateral pharyngeal space and contains the masseter and pterygoid muscles, the inserting tendons of the temporal muscle, the ramus and a portion of the body of the mandible, and the inferior alveolar vessels and nerve. The space is continuous superiorly with the temporal space and is divided by the inferior border of the mandible into deep and superficial compartments. Virtually all masticator space infections results from a dental pathologic condition or trauma, and the infecting organisms generally reflect oral cavity flora. An infection from a lower molar will result in involvement of the superficial compartment of the masticator space if it perforates the buccal plate of the mandibular body. Involvement of the deep compartment of the masticator space results if perforation of the lingual plate of the mandibular bone occurs. Involvement of the superficial compartment produces extensive swelling and, as a result of masseter muscle involvement, severe trismus and pain. If left untreated, spread may occur to the superficial portion of the temporal space. Conversely, involvement of the deep compartment, though producing trismus and pain, results in little external facial swelling. Patients with deep masticator space infections will complain of marked dysphagia and odynophagia and will exhibit intraoral swelling in the retromolar trigone, which is frequently mistaken for a peritonsillar abscess.

If the patient is unresponsive to initial antibiotic therapy, surgical drainage may be required. Drainage may be accomplished through either an intraoral or external incision, depending on which space is involved. The external incision usually employed is horizontal and is placed 2 to 3 cm beneath the angle of the mandible in a skin crease. The inferior border of the mandible is exposed, taking care not to injure the marginal mandibular branch of the facial nerve; this usually requires ligation of the facial vein and artery. The tendon of the masseter muscle is then incised as it attaches to the mandible, and the lateral component of the space is easily entered. An intraoral incision placed along the inner margin of the mandibular ramus in the retromolar trigone region provides access to the medial component of the masticator space. If superior extension to the temporal space has occurred, additional drainage may be achieved with either a preauricular incision or through a Gillies incision, remembering that the temporalis fascia must be incised to enter the temporal space.

Visceral Space Infections

The visceral space lies adjacent to the esophagus and trachea, is bounded by the middle layer of the deep cervical fascia, and includes the carotid sheath. Superiorly it is bounded by the hyoid bone; inferiorly it extends into the mediastinum. Infection of the visceral space usually results from contamination as the result of trauma to the upper aerodigestive tract or secondary to infection resulting from a surgical procedure such as thyroidectomy. Other less common causes that should also be kept in mind include laryngopyocele, thymic cyst, bronchogenic cyst, thyroid cyst, branchial arch anomalies, and thyroiditis. Secondary involvement of the visceral space, particularly the carotid sheath, may occur through extension of infection from the lateral pharyngeal or retropharyngeal spaces or from a primary process in the lungs or mediastinum.

Branchial cleft cysts are not an uncommon source of deep neck infections. The presence of a cyst is usually unsuspected, with no history of a prior neck mass being noted. Typically, the patient presents with acute onset of a midcervical neck mass, neck pain, and fever. The history and physical examination are usually not contributory toward determining a cause. However, the CT scan with contrast presents the typical picture of a fluid-filled mass with enhancing walls, which is pushing the great vessels medially and lies along the anterior border of the sternocleidomastoid muscle. Second branchial cleft cysts are the most common cause of these types of infections; however, third branchial cleft cysts with connections to the pyriform sinus and thyroid gland have also been reported.

Trauma from food, foreign bodies, or instrumentation may result in a primary visceral space infection, usually through pharyngeal or esophageal perforation. These infections, especially following esophageal perforation, are extremely serious and may result in significant morbidity and mortality.

Pharyngoesophageal perforation may also occur after blunt trauma to the neck. Frequently after this type of injury, there may be extensive damage to the larynx, trachea, or esophagus, without signs of significant external neck trauma. In this situation, airway considerations are paramount; however, one should always consider the possibility of a pharyngeal or esophageal perforation and give serious consideration to endoscopic or radiographic evaluation.

Clinical manifestations of a visceral space infection will depend on the cause and location of the infection. Esophageal perforation, for instance, will result in marked neck pain, odynophagia, tachycardia, and fever. Crepitation may be noted if subcutaneous emphysema is present, and a pneumomediastinum will usually be noted on a chest x-ray film. In cases involving blunt neck trauma, the acute signs and findings of laryngotracheal fracture may obscure the esophageal injury unless one keeps this possibility in mind.

Treatment of these infections is also largely a function of the cause and site of primary involvement. In the case of an abscess following neck surgery, reopening the wound with a short course of antibiotics may be all that is necessary. In cases secondary to an unusual cause, such as bronchogenic cyst or branchial cleft anomaly, surgical drainage of the abscess and then a staged removal of the cyst is generally advocated.

In general, there is no consensus regarding the therapeutic approach to esophageal perforation with subsequent visceral space infection. Some authors are strong advocates of aggressive surgical management with exploration and drainage. They feel that early surgical management reduces the possibility of spread of infection into the mediastinum and tends to shorten the hospital stay. Sawyers and co-workers condemn nonoperative management because of the difficulty in determining the size and significance of a perforation and predicting its ultimate outcome. Conversely, patients with small perforations that were detected early have been successfully managed with nonoperative therapy consisting of high-dose antibiotics, nasogastric tube placement, and careful monitoring.

The major factor for determining eventual morbidity and mortality from esophageal perforations appears to be the time from injury to diagnosis and initiation of therapy. Blichert-Toft found in his review of 221 cases of esophageal perforation that 92 per cent of patients left untreated during the initial 48 hours subsequently died. Toward this end of early diagnosis, radiographic procedures may be extremely helpful; posteroanterior and lateral chest x-ray films as well as soft tissue neck films are requisite. Meglumine diatrizoate (Gastrografin) or thin barium swallow esophageal examinations may be helpful in determining the site as well as the severity of injury. Hagen has emphasized the importance of direct endoscopy in cases of blunt neck trauma with suspected esophageal perforation.

If surgical treatment is decided upon, a lateral cervical approach along the anterior border of the sternocleidomastoid muscle is usually used. Other incisions, such as a collar type or paratracheal, are also acceptable, depending on individual preference. In any event, complete drainage of the unilateral visceral space with drain placement is imperative and may require thoracic surgical consultation for chest tubes or open chest mediastinal drainage.