Suprahyoid Spaces of the Head and Neck
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When one refers to the deep spaces of the neck, one includes all of the tissue outside the aerodigestive system mucosal and submucosal structures. The major lesions of the aerodigestive mucosal space (AMS) are surface epithelium neoplasms. Squamous cell carcinomas (SQCCAs) account for most AMS neoplasms, and in concert with minor salivary gland neoplasms and lymphoma of Waldeyer’s ring, they comprise over 95% of the neoplasms of the AMS. Thus, there is a reasonably limited differential diagnosis for masses of the AMS. In contrast, the variety of lesions found in the deep spaces of the head and neck is astounding, and one is required to include spread of AMS lesions to the deep spaces with one’s differential diagnoses.

The classification of regions of the deep spaces is not wholly arbitrary, yet engenders some controversy amongst head and neck radiologists. For the radiologist who is not reading head and neck cases on a daily basis, the model derived from the works of Harnsberger, Osborne, and Smoker serves as the most practical approach. This model divides the nonmucosal spaces of the neck into regions defined by layers of the deep cervical fascia. The deep cervical fascia has superficial, middle, and deep layers, and these layers, in effect, form the boundaries for different spaces of the head and neck.

One separates the neck into the suprahyoid and infrahyoid compartments because of the termination of some of the spaces at the hyoid level. In the suprahyoid region, the spaces of the neck include the masticator space, the prestyloid parapharyngeal space (PPS), the post-styloid parapharyngeal space or carotid space, the parotid space, the retropharyngeal space, and the perivertebral space. In the infrahyoid region, the visceral space (encompassing the trachea, esophagus, thyroid and parathyroid glands) comes into play, whereas the masticator space, the PPS, and the parotid space are no longer present. The carotid space, retropharyngeal space, and the perivertebral space span the suprahyoid and infrahyoid compartments.

Before analyzing these spaces, it is helpful to understand how to localize a lesion to a particular space. Central to the suprahyoid spaces is the prestyloid parapharyngeal space, which is relatively unique among the spaces because its major component is fat. It is also the most mobile of the spaces of the head and neck because it is incompletely ensheathed by the deep cervical fascia. Most head and neck radiologists use the displacement of the PPS to help decide in which space a lesion resides. Thus if the PPS fat is pushed posteromedially by a mass, the assumption is that the lesion arose in the masticator space. If the fat is pushed posterolaterally, an AMS mass is suspected. If the mass is pushed anteriorly or anteromedially with predominantly an anterior direction, a carotid space lesion is the likely culprit. (Distinguishing a pre-styloid process mass from a post-styloid carotid space mass requires visualization of the styloid process by computed tomography (CT) and the styloid musculature by magnetic resonance imaging [MRI]). If there is predominantly a medial displacement with some anterior component, a deep lobe parotid mass is the likely source. A retropharyngeal lesion will usually push the PPS fat anterolaterally. A perivertebral lesion may not affect the fat at all, but if it does, there will usually be an anterior component of displacement.

The second important structure in the neck for localizing lesions is the longus colli musculature complex. When these muscles are displaced posteriorly, the lesion is usually arising from the AMS or the retropharyngeal space. If displaced anteriorly, a perivertebral source is indicated. The muscles themselves are part of the perivertebral space so an intrinsic longus colli mass is within the perivertebral space.

At this point, an analysis of each space may help to gain confidence in diagnosing deep space lesions.

MASTICATOR SPACE

The masticator space encloses the muscles of mastication (medial and lateral pterygoids, masseter, and temporalis), the neck and condyle of the mandible, and for simplicity’s sake, the buccal...
region. Many of these structures are innervated by branches of the trigeminal nerve and are supplied by external carotid artery branches. The proximity of the pterygoid muscles to the AMS and the mandible to the retromolar trigone and floor of mouth predisposes this space to infiltration by AMS SQCCA.

A lesion of the masticator space will displace PPS fat posteromedially and the longus musculature will be unaffected or displaced posteriorly. If the lesion is primarily located in the masseter or temporalis muscle, the PPS fat may be unaffected.

The most common non-neoplastic lesions of the masticator space are odontogenic in origin.\textsuperscript{7-9} Infiltration of the muscles by infectious spread from dental caries or as a complication of tooth extractions is not infrequently seen. An abscess, a mass defined by peripheral contrast enhancement, may be present on either side of the mandible or maxilla or may straddle the bone (Fig 1). The presence of edematous linear stranding into the subcutaneous tissue or the PPS (if not the clinical symptoms) will suggest the inflammatory nature of the lesion. Of the noninflammatory non-neoplastic odontogenic lesions, dentigerous cysts, aneurysmal bone cysts, and simple unicameral cysts may lead to evaluation of masticator space lesions.\textsuperscript{7} The muscles are also not immune from pathology. One may see atrophy of the muscle after denervation injuries, or bilateral or unilateral enlargement of the muscles in cases of bruxism or storage disease infiltrations.

Of benign neoplasms, hemangiomas (eg, venous vascular malformations) in the pediatric age group and schwannomas and mandibular bony neoplasms in the older population will predominate. The hemangiomas may spread across spaces of the neck (transpatial) disrespecting the deep cervical fascia. The schwannomas are usually of mandibular nerve origin and may be located anywhere from the inferior alveolar canal, the soft tissue of the masticator space, or the foramen ovale. Denervation of the muscle or abnormal T2-weighted intensity and gadolinium enhancement of the masticator muscles may be seen in conjunction with the neurogenic tumor. Of benign mandibular tumors, ameloblastomas are most common.

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The most common malignancy to affect the masticator space is an AMS SQCCA. This is most commonly manifest as infiltration of the pterygoid muscles from tonsillar or retromolar trigone cancers leading to clinical complaints of trismus.\textsuperscript{10} Alternatively, mandibular invasion may occur from cancers of these sites. Metastases to the mandible and direct secondary invasion by AMS malignancies are more common than primary malignancies of the masticator space. The most common primary malignant masticator space histologies are rhabdomyosarcomas (Fig 2), lymphomas, and bony sarcomas. Tumors may spread to or from the masticator space via perineural spread along the mandibular nerve.\textsuperscript{11}

**PRESTYLOID PARAPHARYNGEAL SPACE**

The PPS contains fat, vessels, nerves, and on occasion, ectopic minor salivary gland tissue. The “styloid” of “prestyloid” refers to the styloid process and is a useful demarcation of the PPS from the carotid space. Although the styloid process is easily identified on CT, the signal void associated with it on MRI may simulate a vessel. Therefore one must identify the “styloid musculature,” which includes the styloglossus, stylopharyngeus, and stylohyoid muscles on MRI to determine if a lesion is pre- or post-styloid. If the styloid musculature is displaced anteriorly, one should consider a carotid space lesion.

It is rare to see a primary PPS lesion. The inflammatory lesions of this space are usually arising from the AMS, the tonsillar crypts, the deep

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Fig 1. Masticator infection. This contrast-enhanced CT scan shows low density within the right masseter muscle (arrowhead) as well as enlargement of the right pterygoid muscle. The parenchymal fat on the right side is displaced posteriorly and medially (arrow). The CT scan also shows infiltration of the subcutaneous tissue superficial to the right masseter muscle, suggestive of an inflammatory process.
portion of the parotid gland, or the retropharyngeal space with secondary edema of the PPS. Branchial cleft cysts may occur in this space from either cleft I or II origin.

The most common benign neoplasm of the PPS is the pleomorphic adenoma, which takes origin from the ectopic minor salivary gland tissue that is present in the PPS.\textsuperscript{5,12-15} This lesion is characterized by high signal intensity on T2-weighted scans and homogeneous enhancement (this distinguishes it from the branchial cleft cysts that will not enhance or will only show rim enhancement). Rarely one may find hemangiomas, schwannomas, and paragangliomas in the PPS,\textsuperscript{12} though the latter two are much more frequent in the carotid space. Benign lymphadenopathy secondary to tonsillitis and pharyngitis may also be seen in the PPS.

SQCCA of AMS origin is the most common source of malignancy in the PPS (Fig 3). In infiltrating the PPS, SQCCAs from the AMS will usually push the PPS fat posterolaterally. Other mucosal-based malignancies may also spread to the PPS. Malignant lymphadenopathy may reside in the PPS. As for primary malignancies, these are derived from the ectopic minor salivary glands (adenoid cystic carcinoma being most common followed by adenocarcinoma) or from soft-tissue sarcomas (synovial sarcoma, hemangiopericytoma, neurofibrosarcoma). Lymphomatous nodes may be present in the PPS.

To unequivocally identify a lesion of the PPS as arising in that space, one would like to see the mass entirely surrounded by PPS fat. This is rarely demonstrated and often the PPS fat is either completely obliterated or displaced anteriorly, simulating a parotid space or carotid space lesion. The latter can be excluded if one can determine that the lesion is found anterior to the styloid musculature and/or styloid process.

CAROTID SPACE (POST-STYLOID PARAPHARYNGEAL SPACE)

The carotid space is a colloquialism used for the carotid sheath and adjacent structures. Some head and neck radiologists disdain the term "carotid space" because the fascia around the sheath appears to be incomplete above the angle of the mandible and the vessels lie within the greater parapharyngeal space. Nevertheless, because the pathology of the post-styloid parapharyngeal space is different than that of the pre-styloid compartment it is useful to separate these regions. The carotid artery, jugular vein, sympathetic nervous system plexus, jugular chain lymph nodes, and cranial nerves IX, X, XI, and XII are all included in the contents of the suprahyoid carotid space. Lesions of the carotid sheath push the PPS fat in an anterior direction, with or without a medial component. The styloid musculature or process will also be displaced anteriorly.

Second branchial cleft cysts are often associated with the carotid bifurcation and are the most

![Fig 2. Rhabdomyosarcoma of the masticator space. Unenhanced CT shows an infiltrative mass (asterisks) involving the pterygoid musculature, which demonstrates the characteristic displacement of the parapharyngeal fat in a posterior and medial direction (arrow). Compare right PPS fat with that on the left (arrow/head).](image1)

![Fig 3. Tonsil carcinoma. Note the displacement of the parapharyngeal fat (arrow) laterally by this tonsil carcinoma (T).](image2)
common congenital lesion of the carotid space. Inflammatory masses of the carotid space are often associated with pharyngitis and tonsillitis. Usually these affect the venous side, manifesting as jugular thrombosis or thrombophlebitis. Rarely one may see carotid arterial narrowing, spasm, pseudoaneurysm formation, or thrombosis secondary to an adenitis, pharyngitis, or retropharyngeal infection. Carotid artery dissection may be seen as a direct result of penetrating or blunt trauma or in association with predisposing systemic disorders (eg, fibromuscular dysplasia, Marfan’s syndrome, cystic medial necrosis, Ehler’s Danlos syndrome).

The two most common benign neoplasms to affect the carotid sheath structures are schwannomas and paragangliomas. The former may be derived from cranial nerves IX to XII in the suprahoid compartment (just X in the infrahyoid compartment) or from the sympathetic nervous system plexus (SNSP). Clinical symptoms may include difficulty swallowing, a sensory deficit in the gag reflex, palatal paresis, vocal cord paralysis, atrophy of the sternocleidomastoid and trapezius musculature, or lingual deviation. A lesion of the SNSP (and a carotid dissection) may present as a Horner’s syndrome. In general, vagus nerve lesions displace the carotid artery and jugular vein apart and anteriorly.

Paragangliomas that affect the carotid space may be classified as glomus jugulare, glomus vagale, and carotid body tumors. Glomus jugulare tumors may grow into the jugular veins from the jugular foramen, whereas the glomus vagale tumors often will displace carotid sheath structures anteriorly. The carotid body tumors, arising in the crotch of the carotid bifurcation, tend to splay the internal and external carotid arteries apart, rather than anteriorly as the vagus schwannoma will (Fig 4).

Paragangliomas and schwannomas may appear similarly on CT and MRI scans and can best be distinguished based on dynamic imaging characteristics. Paragangliomas have a characteristic rapid uptake of contrast and an early dip (simulating jugular venous dynamic characteristics), followed by persistent enhancement, whereas the dynamic curve of a schwannoma shows a slower sustained uptake curve, no early dip, and persistent enhancement. On angiography, paragangliomas are markedly hypervascular and can receive feeding vessels from numerous external carotid artery branches, most notably the ascending pharyngeal branches.

Primary malignant lesions are exceedingly uncommon in the carotid sheath. More often, the carotid sheath may be encased by aggressive hypopharyngeal or nasopharyngeal AMS carcinomas or by extracapsular extension of malignant lymphadenopathy. If tumor involves less than 270° of the carotid artery’s circumference, there is a very good chance that the tumor can be peeled from the vessel, sparing the need for carotid sacrifice. With over 270° circumferential involvement, the carotid will have to be resected and temporary balloon occlusion studies will be required to assess the need for revascularization.

PAROTID SPACE

Besides the parotid gland, branches of cranial nerves VII and V, lymph nodes, and blood vessels are enclosed in the parotid space. Nonetheless, with
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the exception of the occasional schwannoma, it is glandular masses that are most commonly evaluated in this space. Although lesions of the superficial portion of the parotid gland will not affect the PPS fat, those that arise in or extend to the deep portion of the gland (defined by the plane of cranial nerve VII or the stylomandibular tunnel) will displace the PPS fat in an anteromedial direction (Fig 5). These lesions are always anterior to the styloid muscles and process.

Inflammatory conditions of the parotid gland may be diffuse and bilateral (mumps parotitis, Sjogren’s syndrome, Mikulicz’s diseases, radiation-induced sialadenitis), diffuse and unilateral (peripartal parotitis, Stensen’s duct sialolithiasis with sialadenitis, viral parotitis, post-traumatic parotitis), focal and bilateral (lymphoepithelial lesions associated with HIV positivity, cat scratch fever), and focal and unilateral (abscess, sialocele, pseudocyst). With the exception of the chronic sialadenitides, most of the inflammatory conditions of the parotid gland are characterized by bright signal on a T2-weighted series. Edema in the subcutaneous fat or adjacent PPS fat may suggest an inflammatory cause.

When one finds a cyst in the parotid gland, one must consider a broad differential diagnosis that includes a first branchial cleft cyst, a pseudocyst, a sialocele, a retention cyst, a lymphoepithelial cyst (HIV), or a cystic neoplasm. The patient’s history and risk factors may help distinguish these entities. Usually the cystic neoplasms (Warthin’s tumors and pleomorphic adenomas for the most part) have a solid, enhancing nodule associated with them.

Pleomorphic adenomas, a.k.a. benign mixed tumors, are the most common parotid neoplasm, account for over 70% of all parotid neoplasms and over 85% of all benign parotid tumors. Their characteristic high intensity on T2-weighted scans suggest their diagnosis, often prompting the need for contrast-enhanced studies to confirm they are not cysts. A recent publication has found that they have delayed and sometimes heterogeneous enhancement pattern on CT, but on enhanced MRI they appear to take up the contrast agent promptly and homogeneously (unless they have a cystic or fatty component). Whether assessing a deep or superficial parotid mass, pleomorphic adenoma is the most common tumor.

Other benign parotid neoplasms include the Warthin’s tumor. This is most often seen in elderly men in the tail of the parotid and is usually of heterogeneous T2-weighted intensity. It is the tumor most likely to be bilateral and multiple. Oncocytomas, monomorphic adenomas, and myoepitheliomas are other benign parotid-based masses. Hemangiomas may infiltrate the parotid gland and lipomas may occur within or outside the gland in the parotid space. Schwannomas, as mentioned before, may arise from the facial nerve or the auriculotemporal branch of the mandibular nerve.

Malignant parotid masses are usually due to mucoepidermoid carcinoma, adenoid cystic carcinoma, adenocarcinoma, or acinic cell carcinoma, in that order. The malignancies are usually intermediate in signal intensity on T2-weighted scans, especially if hypercellular and of higher grade. Their margins may be irregular and infiltrative, though margination has not been as reliable in evaluating parotid masses as elsewhere.

Because the parotid gland encapsulates later in fetal development, it has lymphoid tissue within it. Therefore one may see lymphadenopathy from systemic diseases (eg, sarcoid, lymphoma, tuberculosis, HIV), regional lymphatic spread of tumors (squamous cell carcinoma and melanomas of the skin), and primary parotid lymphomas affecting the gland.

RETROPHARYNGEAL SPACE

The retropharyngeal space contains fat and lymph nodes in its suprahyoid compartment. This space
may extend inferiorly into the upper thorax or as a potential space (the danger space) to the diaphragm. Situated as it is between the AMS and the longus colli-longus capitis muscle complex, it is predominantly affected by diseases of the AMS and will displace the longus musculature posteriorly. If the retropharyngeal pathology is large enough, it will push the PPS fat anterolaterally, while it stays posterior to the styloid musculature. Owing to its limited contents, the retropharyngeal space, like the PPS, is more commonly secondarily infiltrated than affected by intrinsic lesions.33,34

Of benign conditions that affect the retropharyngeal space, tortuous carotid arteries that course medially though incompetent layers of the deep cervical fascia may present as a retropharyngeal pseudomass. Benign lymphadenopathy as a response to pharyngitis also account for many lesions in this region. Pharyngitis may give rise to lymphadenitis, which may suppurate and lead to retropharyngeal inflammatory masses. Although most "retropharyngeal abscesses" are actually necrotic supplicative lymph nodes (and usually seen adjacent to the carotid arteries), occasionally a true abscess in the more medial midline location will occur (Fig 6). Alternatively, the retropharyngeal fat may appear "dirty" without a ring-enhancing or water density collection. This is usually due to edema in this space. No true collection is present—just inflamed edematous fat.

Lipomas may arise in the retropharyngeal space. Besides the occasional hemangioma, no other benign neoplasms populate this area.

AMS pharyngeal SQCCA is the most common malignancy to invade the retropharyngeal space. Nasopharyngeal carcinoma or its lymph nodes infiltrate the RPS in 45% of cases at presentation.35 The rate is slightly less for oropharyngeal and hypopharyngeal primary malignancies. Minor salivary gland malignancies of the AMS, rhabdomyosarcomas (in children), and lymphomas may also spread posteriorly.10 Malignant lymphadenopathy from these primary tumors may present in the RPS. Parotid and thyroid malignancies also appear to have a propensity for lymphatic spread to retropharyngeal lymph nodes.36

Fig 6. Retropharyngeal abscess extending into thorax. (A) This patient had perforation of the esophagus with extension of a retropharyngeal abscess into the upper neck. This contrast-enhanced CT scan shows a low density retropharyngeal mass (arrows) in front of the longest colli muscles (c). Note the gas in the abscess. (B) The collection (arrow) is seen posterior to the esophagus (e). (C) Note that at the level of the manubrium the abscess collection (arrow) is still quite large.
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PERIVERTEBRAL SPACE

The perivertebral space includes the vertebral columns, the paraspinal musculature, the posterior triangles of the neck, and the neurovascular bundles emanating from the spinal canal. As such it encompasses all of the structures posterior to and including the longus colli-longus capitis muscular complex. When lesions of the perivertebral space are in proximity to the longus musculature, they will displace the muscles anteriorly, thereby distinguishing these masses from retropharyngeal masses or invasive AMS masses.37

The variety of lesions that are located in this “grab-bag” space is quite wide because the lesions may be of bony, muscular, neurogenic, vascular, or lymphatic origin. Of congenital lesions, lymphangiomas and venous vascular malformations are commonly seen in this region. The classic location of a cystic hygroma is in the posterior triangle of the neck or extending to the axilla. The lesion is usually multiloculated and of various densities and intensities owing to hemorrhage, lymph, or high protein content.

Developmental lesions of the spine including various cysts, fibrous dysplasia, Paget’s disease, renal osteodystrophy, degenerative spurs (especially anterior ones that push the longus musculature anteriorly), diffuse idiopathic skeletal hyperostosis, anterior longitudinal ligament hypertrophy, and traumatic injuries or hematomas associated with the cervical spine are all considered lesions of the “perivertebral” space.

The infections that affect the perivertebral space are usually those that are associated with the spine as well. Discitis, osteomyelitis, epidural abscesses, paraspinal collections, and phlegmons may occur de novo, particularly in the population that abuses intravenous drugs, is exposed to tuberculosis, or undergoes spinal surgery. Staphylococcus, streptococcus, and mycobacteria are the usual pathogens.

Tumors of neurogenic origin are some of the more frequently seen benign neoplasms of the perivertebral space. Although some may emanate from the neural canal, thereby widening the foramen, still others may derive from the branches beyond the foramen and present as true soft-tissue masses in the neck. In this way, they may present behind the carotid sheath structures, simulating vagal schwannomas.

Other benign neoplasms in this region include those of bony origin.16 The chordoma is the classic lesion of this space because it may be centered in the upper cervical spine, will displace the longus musculature anteriorly, and may appear quite aggressive. However, other benign bony lesions, such as osteoblastomas, giant cell tumors (isolated or with aneurysmal bone cysts), hemangiomas, and osteochondromas may populate this space.

Of malignancies, the bony varieties include plasmacytomas, osteogenic sarcomas, Ewing’s sarcoma, and lymphoma. Nonetheless, metastatic disease to bone will be the most common malignant lesion of the perivertebral space.37 In the soft tissues of the space, one may find lymphoma and a variety of sarcomas (neurofibrosarcoma, hemangio-pericytoma, rhabdomyosarcoma, and synovial sarcoma) (Fig 7). Malignant lymphadenopathy in the spinal accessory chain, usually from an AMS SQCCA but, in the young adult population may be from Hodgkin’s and non-Hodgkin’s lymphomas, will account for many lateral perivertebral masses. In the lower neck the adenopathy may represent spread from lung, breast, or thyroid primary tumors.

TRANSSPATIAL AND MULTISPATIAL LESIONS

Obviously, from the discussion above, one notes that certain lesions seem to ignore the fascial layers that encompass the suprahyoid spaces of the neck.38 These transspatial lesions include, first and foremost, squamous cell carcinoma, which will spread through fascial planes in a very aggressive manner. Of benign masses, hemangiomas and lymphangiomas seem to be particularly infiltrative. One infectious entity that favors crossing the fascial planes is necrotizing fasciitis, a very aggressive disease that can cause sloughing of soft-tissue layers.39

By multispatial, one refers to lesions that can be found in a variety of spaces concurrently. The classic multispatial process is lymphadenopathy, which can be found in retropharyngeal, carotid, perivertebral, PPS, and parotid spaces. Generalized lymphadenopathy like that is usually ascribed to lymphoma, HIV or AIDS infection, mononucleosis, metastatic disease, or occasionally, mycobacterium infection. Schwannomas, neurofibromas, and hemangiomas may also be located in many spaces particularly in such syndromes as neurofibromatosis, Mafucci’s syndrome, and Klippel Trenaunay
Fig 7. Hemangiopericytoma in the perivertebral space. (A) This hemangiopericytoma (h) infiltrated the perispinal musculature as well as extended into to the right neural foramina, seen to be enlarged on T1-weighted scan. (B) A T2-weighted scan shows the relatively low signal intensity of the perispinal component of the hemangiopericytoma (h).

Weber syndrome. Metastatic disease to bone may affect the mandible of the masticator space and the spine in the perivertebral space.

CONCLUSION

Although the previous discussion may seem simplistic at first glance, the principles governing the analysis of deep-space masses of the head and neck will serve to help localize the lesion and limit the usually broad differential diagnoses given to masses in this area. By observing the patterns of displacement of the PPS fat, the styloid process or musculature, and the longus musculature complex, one should be able to identify the space in which a mass resides. Although there is some overlap in the entities that populate each space, there are certain favored diagnoses for each region that should be considered first (Table 1).

Table 1. Leading Diagnoses by Space and Type

<table>
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<tr>
<th>Space</th>
<th>Congenital</th>
<th>Inflammatory</th>
<th>Benign Neoplasm</th>
<th>Malignant Neoplasm</th>
<th>Other</th>
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<td>Odontogenic abscess</td>
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<td>Branchial cleft cyst</td>
<td>Pharyngitis spread</td>
<td>Pleomorphic adenoma</td>
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