Anterior Throat Pain Syndromes: Causes for Undiagnosed Craniofacial Pain

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Anterior Throat Pain Syndromes: Causes for Undiagnosed Craniofacial Pain

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ABSTRACT: It is not uncommon for practitioners who treat craniofacial pain to see patients with undiagnosed throat and submandibular pain. Usually, these patients will already have been seen by their primary care physician and frequently, several others doctors including otolaryngologists, oral and maxillofacial surgeons, and even neurologists. Far too often these patients have three common features: 1. they have endured multiple expensive diagnostic tests; 2. they have received treatment of multiple courses of antibiotics; and 3. no specific diagnosis for their pain complaints has been determined and their pain persists. In this article, five disorders, Ernest syndrome, Eagle's syndrome, carotid artery syndrome, hyoid bone syndrome and superior pharyngeal constrictor syndrome are briefly described. All five produce common symptoms, making diagnosis difficult, which is often followed by ineffective or no treatment being provided to the patient. Diagnostic criteria and suggested treatment modalities are also presented.

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Diagnosing the cause of pain arising from the anterior throat is a common problem shared by otolaryngologists, primary care physicians, and dentists who treat craniofacial pain. Usually, all occult possibilities have been ruled out, and the patient has been reassured that the persistent pain is not life-threatening, and yet such chronic and unrelenting pain forces these unfortunate patients to continually seek help. Much has been written about temporomandibular disorders (TMD), both in the dental and medical literature, but sadly, little has been written about the causes of throat or anterior cervical pain.

Patients afflicted with various craniofacial pain disorders reportedly suffer over four years and see, on an average, nearly four practitioners in an attempt to get help for their pain complaints.[1] In this article, five disorders, Ernest syndrome, Eagle's syndrome, carotid artery syndrome, hyoid bone syndrome and superior pharyngeal constrictor syndrome are briefly described. All five produce common symptoms, making diagnosis difficult, which is often followed by ineffective or no treatment being provided to the patient.

Ernest Syndrome

Throat pain associated with the styloid process has been discussed in the literature since at least the 19th century.[2,3] The best known pain disorder involving this structure is known as Eagle's syndrome.[4-8] It is felt that the pain from this syndrome is due to mineralization of the stylohyoid ligament, elongation of the styloid process, or continued growth and subsequent ossification of the second branchial arch cartilage.[9-11] Clinically, this condition is known to cause a multitude of symptoms, which is described later in this article.

A similar, yet quite different and more common disorder that also involves the styloid process, has recently been reported.[12-18] In contrast to Eagle's syndrome in which the primary structure affected is the stylohyoid ligament, Ernest syndrome (insertion tendinosis of the stylomandibular ligament) involves the stylomandibular ligament, especially its insertion into the mandible (**Figure 1**).

The styloid process is a tapered, cylindrical bony process approximately 2.5 to 3.0 cm in length. Originating from the petrous portion of the temporal bone on the base of the skull, this structure extends anteriorly and inferiorly towards the medial wall of the mandibular ramus and connects to the hyoid bone (via the stylohyoid ligament), the tongue (via the styloglossus muscle), the pharynx (via the stylopharyngeus muscle) and through the stylomandibular ligament to insert on the medial side of the mandible[19] approximately 10 to 15 mm superior to the angle (**Figure 2**).[15]

The functions of this ligament are to limit the maximum opening and protrusion of the mandible.[20-22] Innervation of the stylomandibular ligament has not been specifically established to date in the anatomical literature. However, based upon Ernest's studies of blocking the third division of the trigeminal nerve at foramen ovale and thus providing total anesthesia to the ligament,[14] and when considering the embryological development of this region (from the first branchial arch), it is reasonable to assume that this structure is served by a branch of the third division the trigeminal nerve.

The pathogenesis of all these syndromes is similar. Stucke[23] demonstrated that degenerative changes in a muscle or ligament occur at the zone of insertion into the bone (Sharpey's fibers). This condition, whether involving muscle or ligament insertions, has been termed insertion tendinosis.[24] Pederson and Key[25] confirmed these findings histologically. It appears that as degenerative changes occur after strain or trauma, normal mechanical stress through the Sharpey's fibers produces pain, limitation of motion, and referred pain.[14]

Clinically, symptoms of Ernest syndrome are similar to those of internal derangement of the temporomandibular joint (TMJ).[26] These symptoms are: 1. pain in and around the TMJ; 2. ear pain and fullness; 3. anterior temporal pain and headache; 4. pain in the body of the mandible; 5. eye pain; 6. odontalgia; and 7. throat pain (**Table 1**).

The establishment of the diagnosis of Ernest syndrome is based upon three primary criteria: 1. historical report-

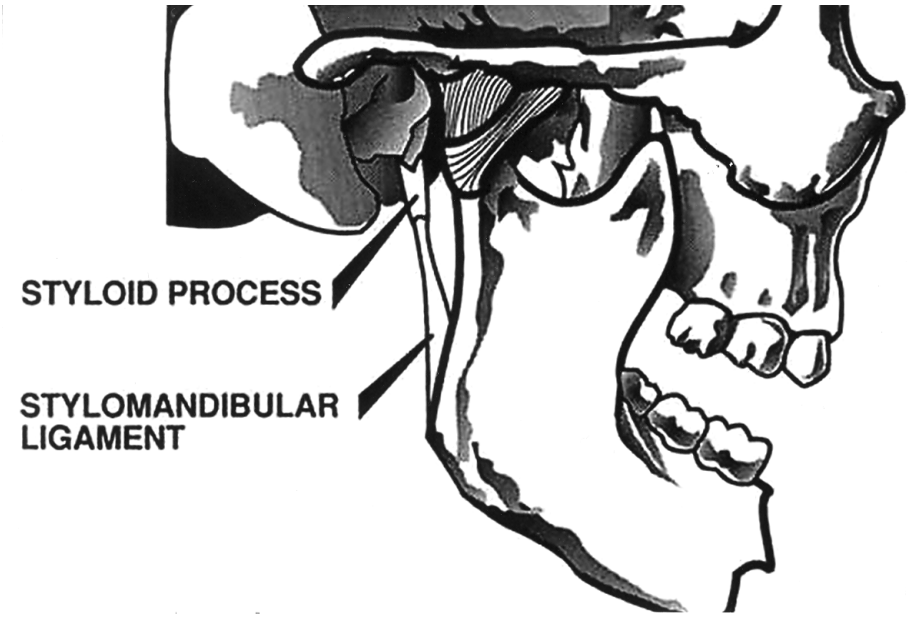

Figure 1
Stylomandibular ligament.

Table 1
Comparison of Symptoms of Disorders that Produce Anterior Throat Pain

Symptoms	Ernest syndrome	Eagle's syndrome	Carotid artery syndrome	Hyoid bone syndrome	Superior pharyngeal constrictor syndrome
TMJ pain	X			X	X
Otalgia	X	X		X	X
Temporal pain	X	X	X	X	X
Eye pain	X				
Pharyngeal pain	X	X	X	X	X
Odontalgia	X			X	
Foreign body feeling		X		X	
Mandibular pain	X				
Vertigo			X		
Dysphasis		X	X	X	X

ing of the patient with associated pain complaints; 2. pain upon palpation of the insertion of the stylomandibular insertion; and, 3. relief of pain after the administration of a diagnostic anesthetic injection into the ligamentous insertion. Historically, the most common etiological factor for the development of Ernest syndrome is trauma to the mandible.[15]

Palpation associated with the elicitation of pain from the insertion of the ligament is the second criterion for establishing a diagnosis of Ernest syndrome. The sensitive insertion is located with a blunt probe and pressure is applied in an anteromedial direction. If the ligament is the source of the patient's pain complaints, then provocation of the ligamentous insertion produces intense pain.

The third criterion, the successful elimination of pain following an anesthetic injection into the insertion of the stylomandibular ligament, is certainly the most important to satisfy. Using a blunt probe, the skin over the angle of the mandible is depressed medially (**Figure 3**) and approximately one cc of local anesthetic is slowly injected

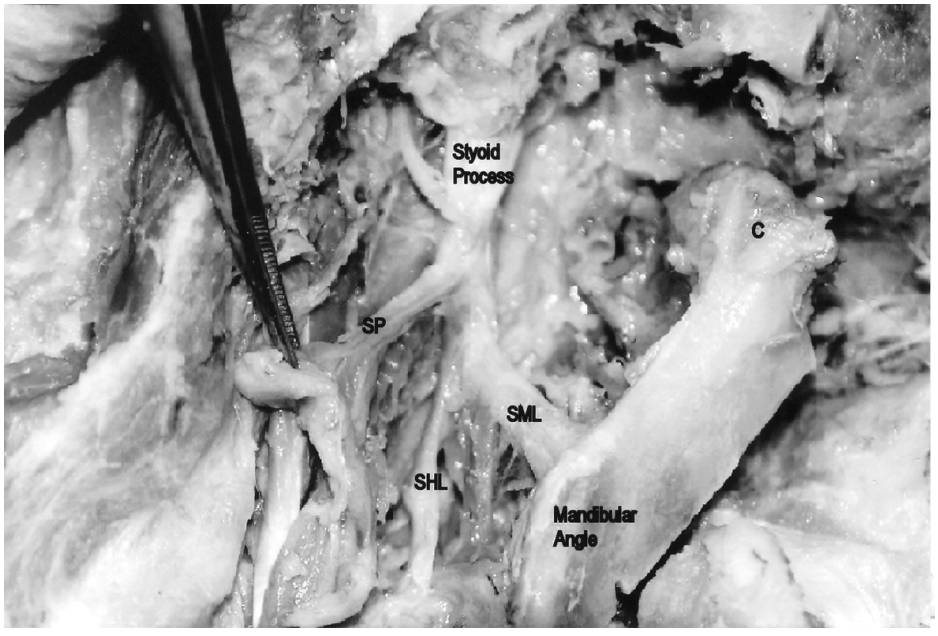

Figure 2
Dissection of lateral neck to demonstrate the stylomandibular ligament.
SML: stylomandibular ligament;
SP: stylopharyngeus muscle;
SHL: stylohyoid ligament;
C: mandibular condyle.

on the medial side of the mandible. If all or a significant amount of the complaints are eliminated, suspect the diagnosis of Ernest syndrome. At times, a second injection will be necessary as some stylomandibular ligaments are quite broad and not easily anesthetized. Due to the close proximity of the facial nerve to the insertion of the stylomandibular ligament, frequently seventh nerve anesthesia is produced after administering the diagnostic anesthetic injection. The patient should be cautioned about this prior to giving the injection.

Treatment for Ernest syndrome may be divided into conservative (or, nonsurgical) and surgical modes of therapy. Shankland[15] reported that successful non-surgical treatment, defined as elimination of pain and restoration of normal ranges of motion of the mandible, was accomplished in 77.94% (53 of 68) of patients treated with Ernest syndrome. Nonsurgical treatment consists of local anesthetic blocking of the affected stylomandibular ligament insertion, injecting one cc of synthetic cortisone or Sarapin (High Chemical Co., Levittown, PA) (a nonsteroidal, anti-inflammatory medication) (**Figure 3**), prescribing a systemic synthetic cortisone dose pack (e.g, methyprednisolone dose pack four mg), and placing the patient on a soft diet. At times, an exaggerated pain response (post-injection flair) might occur after the injections of anesthetic and cortisone. Conservative therapy, if necessary, is repeated at two-week intervals two to three times. Also, physical therapy in the form of iontophoresis, high voltage stimulation, stretch and spray, and the use of ice and moist heat are beneficial. Lastly, if the patient exhibits symptoms of parafunctional activities (clenching, bruxing, etc.), prescribe a flat, superior repositioning intraoral splint to be worn at least at night time or when under stress and preferably, 24 hours a day until their symptoms subside. Then the splint may be worn at nighttime only, if necessary.

Figure 3
Local anesthetic injection to block the insertion of the stylomandibular ligament. A blunt probe is placed posterior to the ascending ramus of the mandible, approximately one cm above the mandibular angle, and pressure is applied in an anteromedial direction on the medial side of the mandible and approximately one cc of local anesthetic is slowly injected.

Surgical management, if necessary, is described as either an open retromandibular approach to resect the ligamentous insertion[13] or radiofrequency thermoneurolysis of the insertion of the stylomandibular ligament.[12,14-17] Approximately 22% of Ernest syndrome cases require surgical intervention.[15]

Eagle's Syndrome

In contrast to Ernest syndrome, the malady most often associated with pharyngeal pain is Eagle's syndrome. First fully described by Dr. Watt Eagle in the 1930s, references to pain in and about the styloid process, producing pain, appear much earlier in the literature. Sterling[2] and Thigpen[3] both described throat pain associated with the styloid complex before Eagle's publications,[4-8] and Dwight[11] described several cases in which ossification of the entire stylohyoid complex occurred.

The stylohyoid ligament originates from the tip of the styloid process and inserts into the lesser cornu of the hyoid bone. The upper fibers of the middle pharyngeal constrictor muscle insert into this ligament, and the stylohyoid ligament serves to stabilize the hyoid bone.

Of clinical anatomical importance, the styloid process lies between the internal and external carotid arteries and just posterior to the pharyngeal wall in the region of the palatine fossa (**Figure 4**). Three muscles originate from the styloid process, each innervated by a different nerve: 1. the styloglossus, innervated by the hypoglossal nerve; 2. the stylopharyngeal, which is innervated by the glossopharyngeal nerve; and 3. the stylohyoid, innervated by the facial nerve. In addition to the carotid arteries, the styloid process lies closely to five cranial nerves (the facial, glossopharyngeal, vagus, spinal accessory, and the hypoglossal) and the internal jugular vein.

Eagle[4-8] and others[27-29] reported that two separate syndromes could be collectively called *Eagle's syndrome*. The first, the classical stylohyoid, styloid-stylohyoid[30] or Eagle's syndrome, was initially described as developing immediately following tonsillectomy, which originally defined the classic Eagle's syndrome. Karlan, et al.[31] insist that without a history of recent tonsillectomy, this pain syndrome's accurate name is *stylalgia*. Today, however, most authorities do not require tonsillectomy in order to qualify the following group of symptoms as classic Eagle's syndrome.

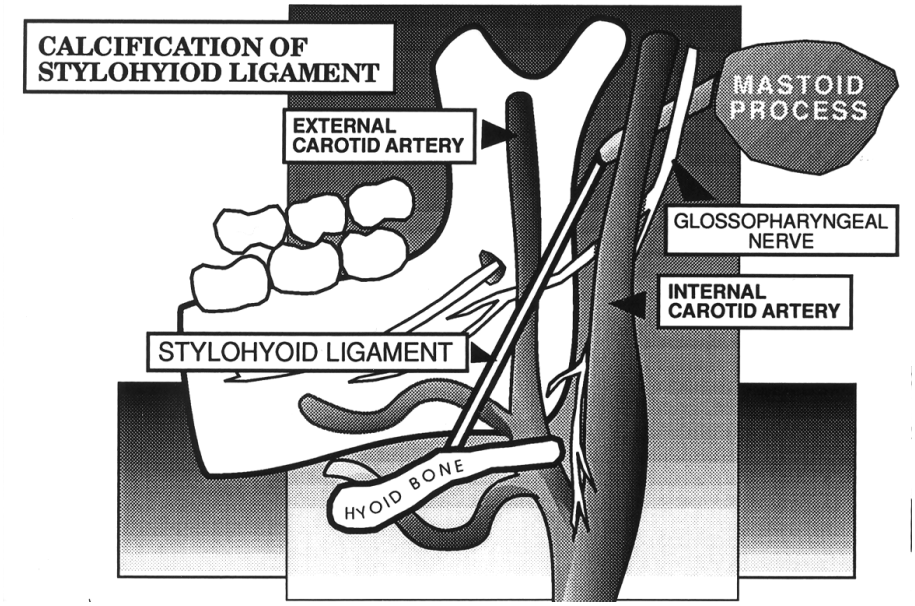

Figure 4
Drawing demonstrating the relationship of the styloid process, stylohyoid ligament and hyoid bone with the carotid arteries.

The symptoms of classic Eagle's or stylohyoid syndrome are: 1. a constant dull pharyngeal ache which may be sharp and stabbing, especially during swallowing or head rotation to the symptomatic side; 2. otalgia; 3. dysphasia; 4. temporal/parietal headache; and 5. a feeling of a foreign body in the throat (**Table 1**). Theoretically, pain may be produced by the stretching or fibrous compression of branches of the fifth, eighth, ninth, and tenth cranial nerves (or, branches of these nerves) in the tonsillar fossa region.

The symptoms of the second syndrome, termed *carotid artery syndrome* by Eagle[6] and others[32,33] or the *styloid process-carotid artery syndrome*,[34] is characterized by dull nagging to sharp pharyngeal pain, headaches, dysphasia, and, vertigo[5] (**Table 1**). Pain may be generated either by compression of the internal carotid artery by elongation of the styloid process or calcification of the stylohyoid ligament or the same type of compression upon the external carotid artery. If the internal vessel is compressed, then the headache pain involves the parietal region; if the external carotid is compressed, pain is reported in the face below the eye to the occiput. Compression of the carotid artery could result in the referral of pain to the musculature of the neck, face, throat, cranial area, the tongue, the skin of the neck, temporal region, and the arterial system itself.[35] Koebke[36] presented histological evidence of early arteriosclerosis subsequent to compression of the external carotid artery.

The diagnosis of Eagle's syndrome (Eagle's and carotid artery syndrome collectively and hereafter in this article referred to as *Eagle's syndrome*) is based upon the presentation of symptoms, intraoral palpation of the styloid process, and radiographic examination.

Reviewing the patient's symptoms allows the clinician to begin to formulate a differential diagnosis. The complaints of dysphasia, pain in the throat (reported as a *sore throat* and unfortunately, often repeatedly treated as an upper respiratory or pharyngeal infection) and the feeling of a foreign body in the throat, are generally pathognomonic for Eagle's syndrome. Frequently, the patient insists that a chicken or fish bone must be lodged in his or her throat; a complaint that usually initiates many visits to doctors of various specialties and the emergency room.

Physical examination is fairly simple. The patient is instructed to open his/her mouth as wide as possible, and then the clinician inserts a gloved finger transorally along the occlusal plane posteriorly to the region of the palatine fossa. Lateral and posterior pressure is applied, and the styloid process can be felt if it is elongated. A styloid process of normal length is difficult to palpate.[37] Elicitation of pain upon palpation is characteristic of Eagle's syndrome. This palpatory maneuver is very important to establish an accurate differential diagnosis.

Caution must be exerted when observing an elongated styloid process radiographically: elongation usually has no clinical significance. Barclay and Donaldson[38] (100 edentulous patients) and Kaufman, et al.[39] (484 patients) corroborate these findings. Such findings are only significant when the patient presents with undiagnosed throat or craniofacial pain. The normal styloid process may be visible with a panoramic view; an elongated styloid process or ossified stylohyoid ligament is certainly

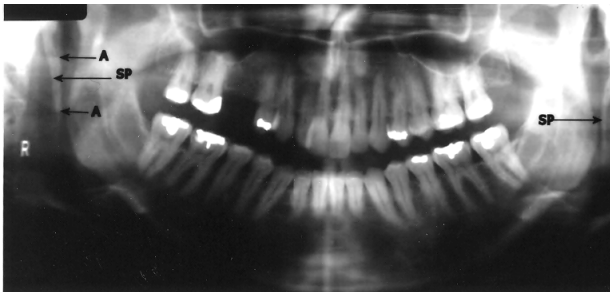

Figure 5
Panoramic radiograph demonstrating an elongated sytloid process. **SP**: styloid process; **A**: articulation of portion of calcified stylohyoid ligament.

noticeable (**Figure 5**). Multiple variations of the stylohyoid chain are often found radiographically. These variations may range from simple elongation of the styloid process, partial or complete calcification of the stylohyoid ligament, or enlargement of the greater cornu of the hyoid bone, often being fixed to the stylohyoid ligament.[40] Often areas of what appears to be articulations of portions of the stylohyoid ligament are visible. After reviewing 1771 panoramic radiographs, Correll and Wescott[41] reported approximately 18.2% of the radiographs revealed varying degrees of elongation of the styloid processes. However, there were no statistical correlations between increasing mineralization, symptomology[42] and age up to the seventh[43,44] or eight decade,[45] and then there appears to be a direct correlation to age and calcification of the stylohyoid ligament. Surprisingly, most patients with abnormal styloid anatomy exhibit no symptoms. Baddour, et al.[46] reported that at most, 4% of these patients have symptoms. Krennmair and Piehslinger[44] confirmed this incidence, but also reported that in the subgroup of patients presenting with abnormal radiographic findings and TMJ symptoms, such styloid chain abnormalities may be the cause of pain in 20% or more of these patients. Godden, et al.[47] demonstrated this point specifically in a case study in which mandibular movement produced what appeared to be clicking emanating from the temporomandibular joint but was later discovered originating from the stylohyoid chain.

To fully survey this region, both a panoramic and anteroposterior radiographs are taken to visualize these structures in two planes. Correlation of the findings of these two views will reveal deviation of the stylohyoid complex as it courses between the carotid arteries, degree of elongation of the styloid process, and ossification of the stylohyoid ligament.

The overwhelming recommended treatment for Eagle's syndrome is surgical resection of the styloid process and/or the ossified stylohyoid ligament. Surgery may simply be fracturing the process laterally or resection. Eagle[4,5,7,8] contended that fracturing only did not resolve the actual problem and in fact, this procedure is no longer considered acceptable.[48] Currently, both intraoral and extraoral surgical approaches are accepted[49] and basically left to the surgeon's discretion and training; both have advantages and disadvantages. The intraoral approach provides less visibility[50] and can be performed using local anesthesia, but leaves no cutaneous scar and avoids many vital neck structures. Amputation of the lesser cornu of the hyoid bone has also been suggested as a surgical treatment for Eagle's syndrome.[51]

Hyoid Bone Syndrome

In 1954, Brown reported a pain syndrome, which he identified as the *hyoid bone syndrome*.[52] This disorder, although not well recognized, was subsequently described by others.[53-62] This rather obscure pain disorder produces chronic or recurrent pain in the area of the carotid sinus at or near the tip of the greater cornu of the hyoid bone.

The hyoid bone is a U-shaped structure which lies in the anterior portion of the neck at the root of the tongue between the mandible and the larynx (**Figures 6**, **7**). It is the only bone of the body that does not articulate with any other osseous structure. It has a body, a pair of greater horns (greater cornua) and a pair of lesser horns (lesser cornua). This bone may be palpated approximately one finger's breadth above the laryngeal prominence. If the patient swallows while the finger is palpating the body of the hyoid bone, the bone is felt rapidly ascending.

The hyoid bone is suspended from the styloid processes of the temporal bones by the stylohyoid ligaments, which are inserted into the lesser horns. The body is irregular,

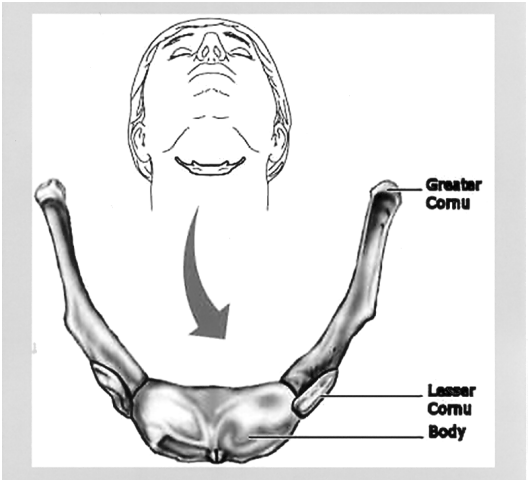

Figure 6
Hyoid bone. Note the position below the mandible.

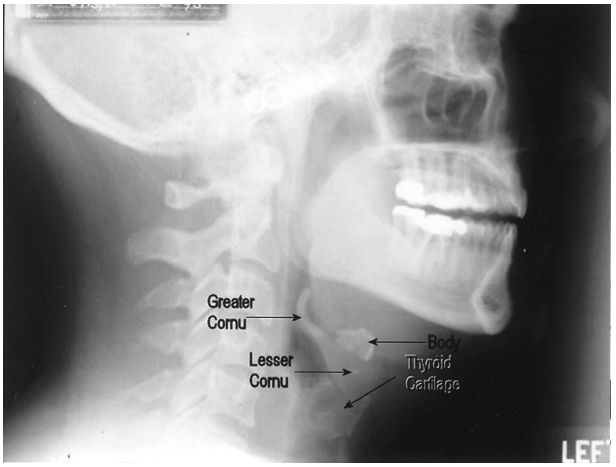

Figure 7
Lateral cervical radiograph demonstrating the hyoid bone. **Note** the close proximity of the thyroid cartilage, which can also be a source of pain if injured.

elongated and quadrilateral.[63] The greater cornua are large, somewhat flattened structures which project from the lateral borders of the body, backward and laterally. In early life the greater cornua are connected to the body by cartilage. However, after middle age, they are united by bone[63] or synovial joints.[54,55]

The primary symptom of hyoid bone syndrome is chronic or recurrent pain in the carotid area emanating from the greater cornu of the bone. The pain, which can be either dull or lancinating, radiates to the ipsilateral ear, throat,[62] temple,[60,62] zygomatic arch, temporomandibular joint,[60] sternocleidomastoid muscle and posterior pharyngeal wall.[60] In addition, some patients report pain in the mandibular molar teeth, supraclavicular area, and even to the level of the middle of the breast without extending below the nipple[57] (**Table 1**). Movement of the head towards the affected side may produce dizziness or syncope[54,55,60] or pain during swallowing.[53,59] Additionally, patients report a chronic *sore throat*, dysphasia, and tightness or pressure in the neck. Lastly, some sufferers report the sensation of a foreign body in the throat associated with dull pain on swallowing,[54,55] which, like many of these symptoms, is also common with Eagle's syndrome.

The establishment of an accurate diagnosis of hyoid bone syndrome, as with other pain syndromes, requires both a careful gathering of the history and a meticulous physical examination. Ernest has reported that the most common cause of injury to the hyoid bone is trauma,[57] which ranges from crushing the anterior neck against a steering wheel in a motor vehicle accident to fracture of portions of the hyoid bone during the act of strangulation. Other causes are bursitis,[53] mechanical restrictions and local pressures due to excess bony growth or stylohyoid calcification,[6] and insertion tendinosis of the middle pharyngeal constrictor.[57] Physically, the most common findings are an enlargement of the styloid process, which is often tender to intraoral palpation,[54] and a marked tenderness at the tip of the greater cornu of the hyoid bone.[62]

Palpation of the hyoid bone can be easily accomplished. Due to the great mobility of this structure, the hyoid bone is steadied by pressure applied on the opposite side in question. The clinician places the index finger on the greater cornu on the nonpainful side and with the thumb, stabilizes the affected greater cornu (**Figure 8**). Then the hyoid is rocked from side to side. Rocking the bone towards the affected side and maintaining pressure on the injured greater cornu will produce pain and frequently reproduce the pain radiation.[52,62]

Radiographic evaluation of the hyoid itself is of little value. However, radiographs are important when surveying the styloid processes or stylohyoid ligaments. CT scans rarely demonstrate any abnormalities in the hyoid bone.[62]

Performing a diagnostic anesthetic block completes diagnostic testing for hyoid bone syndrome. Using a 30-gauge, 1-inch needle, approximately 1/2 cc of 3% mepivacaine is injected over the greater cornu at the site of pain (**Figures 9**, **10**). If all or at least significant symptoms are relieved, then the diagnosis of hyoid bone syndrome must be suspected.

Various modalities of conservative, nonsurgical treatment have been employed in the treatment of hyoid bone syndrome. These include physical therapy, infiltration of local anesthetic with cortisone or Sarapin (often, repeated),[53,54,59-61,64] non-steroidal anti-inflammatory med-

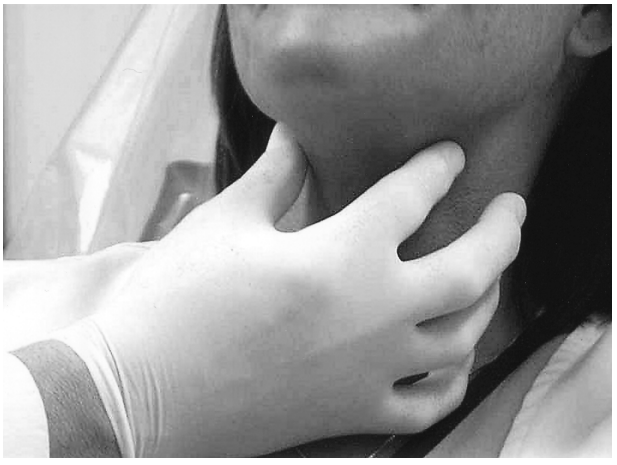

Figure 8
Palpation of the hyoid bone. **Note** that the index finger is placed on the greater cornu on the left side and the thumb is placed on the greater cornu on the right side.

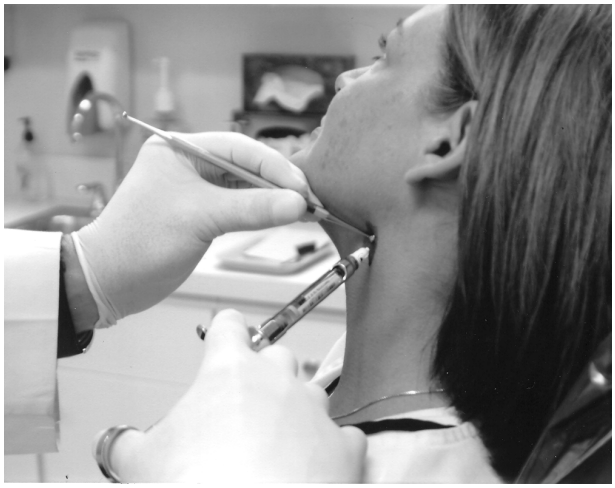

Figure 9
Local anesthetic injection of left greater cornu of the hyoid bone. The skin is stretched over the greate cornu of the hyoid bone and the bone is actually stabilized by the burnisher (or other instrument) during the injection prodcedure.

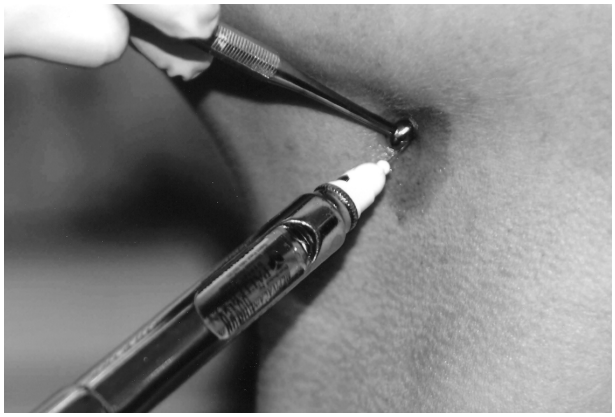

Figure 10
Close up of the injection of the greater cornu of the hyoid bone.

ications,[62] prescription of systemic cortisone and topical anti-inflammatory medications.[62] If conservative therapies are ineffective, most authorities feel that the only viable treatment for this disorder is surgical resection of the greater cornu of the hyoid bone.[52-59]

Superior Pharyngeal Constrictor Syndrome

This is a relatively newly described syndrome. Those who treat craniofacial disorders have long recognized that some patients present with undiagnosed deep pharyngeal pain with no history of trauma, temporomandibular disorders, or even parafunctional activities and yet, complain of pain in the throat and ear. Usually, like most other patients, they have seen numerous doctors of all types of specialties, have undergone numerous diagnostic-testing procedures, and still have undiagnosed pain. This condition is further confused if the patient happens to exhibit ipsilateral clicking of the temporomandibular joint, even though the joint itself may be painless.

First reported by Ernest, et al.[65] in 2006, superior pharyngeal constrictor syndrome is actually a myofascial disorder of the superior pharyngeal constrictor muscle.[66] The development of trigger points in skeletal muscles is common, and the pharyngeal muscles are no exception. However, this particular disorder apparently not only affects the superior constrictor muscle, but also the median raphe, the pterygomandibular raphe, or both. It is most likely that the involvement of such connective tissues that prolongs and intensifies the myofascial pain complaints.

The superior pharyngeal constrictor muscle is a large and complicated quadrilateral muscle that has four origins, namely: 1. the inferior surface of the medial pterygoid plate and pterygoid hamulus of the sphenoid bone; 2. the pterygomandibular raphe; 3. the medial surface of the mandible superior to the mylohyoid line; and 4. the lateral border of the body of the tongue. Curving posteriorly from the origins, the fibers of this muscle encircle the pharynx and insert into the median raphe of the posterior pharyngeal wall, which anchors and unites the both superior pharyngeal constrictor muscles. Howland and Brodie[67] have described the pterygomandibular raphe as the "sphenoid tendon between the jaws." That statement in itself may sum up the basic cause of this type of pharyngeal pain: tendonitis of the pterygomandibular raphe. Each time the patient swallows, biomechanical stresses, transferred into and through the raphe, ultimately stimulate nociceptive fibers in the muscle. Sicher[68] stated that this tendinous structure, stretching from the pterygohamulus to the retromolar pad region of the mandible is greatly stretched when the mouth is opened wide. The innervation of this muscle is via the pharyngeal plexus, which is composed of branches of both the glossopharyngeal and vagus nerves.

The symptoms of superior pharyngeal constrictor syndrome are somewhat similar to those of the previous syndromes, namely: 1. pain in the throat; 2. pain in the TMJ; 3. otalgia; 4. temple pain; 5. pain when swallowing; and 6. incoordination with the swallowing reflex at times (**Table 1**). There may be a history of trauma, such an intubation for a general anesthetic, but oftentimes, the patient has no recollection of any form of trauma. The common or Halsted technique for injecting the alveolar nerve could also produce this syndrome, as the pterygomandibular raphe is one of the structures pierced by the

injection needle. Ernest, et al.[65] reported that one patient developed this disorder after an oral impression was taken, and it was his opinion that the cause was hyperextension of the mandible while the impression was being taken.

The patient may report generalized throat pain, but if asked about the pain specifically, he or she will report pain in the superior-lateral area of the throat with some radiation to the median raphe in the posterior pharyngeal wall.[66] The temporomandibular joint pain, which could easily be misdiagnosed as a displaced articular disk, especially if the joint exhibits clicking, is deep within the joint. These patients do not respond to any type of joint treatment, whether it is medications or splint therapy.

Diagnosis of pharyngeal constrictor syndrome is accomplished with palpation and diagnostic anesthetic confirmation. A cotton-tipped applicator or ball burnisher is used to examine the posterior and lateral areas of the pharyngeal wall posterior to the pterygoid hamulus (**Figure 11**). If a sore or painful area is discovered near pterygoid hamulus, consider hamular process bursitis[69-72] as a differential diagnosis. If an exquisitely tender area in the superior constrictor muscle is located, a small needle

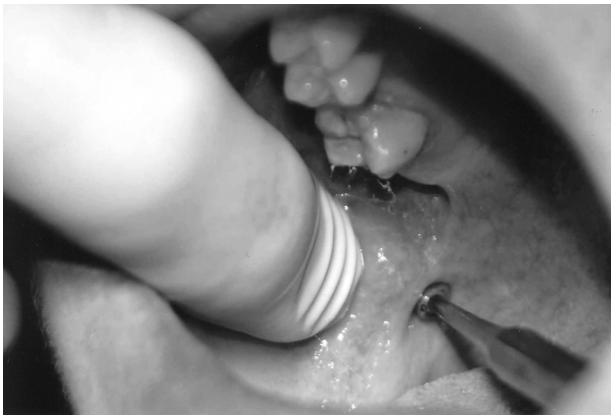

Figure 11
Using a ball burnisher to locate the painful area in the right superior pharyngeal constrictor which may be producing throat pain.

is used to inject 1/4 to 1/2 cc of local anesthetic. These painful areas are usually located either at or along the pterygomandibular raphe or along the bony surface of the medial pterygoid plate.

Treatment is straightforward and according to Ernest,[66] approximately 90% successful. If the patient's symptoms are eliminated with the anesthetic injection, a small amount of anti-inflammatory medication is deposited at the formerly painful site. The patient is placed on the appropriate anti-inflammatory medication and re-evaluated in 10-14 days. These procedures can be repeated if necessary. Cases that do not respond to conservative treatment can be treated with radiofrequency thermoneurolysis.

Pain originating from the superior constrictor muscle is often misdiagnosed as Eagle's syndrome, Ernest syndrome, a temporomandibular disorder, or glossopharyngeal neuralgia.[73]

Conclusions

The diagnosis of the vast multitude of craniofacial pain disorders is difficult. Throat pain is usually not considered or discussed in dental school, residency programs, and certainly in very few post-graduate continuing education programs. When patients have seen otolaryngologists, neurologists, and possibly even oral and maxillofacial surgeons, and all have ruled-out occult disorders and cranial nerve neuralgias, the stylohyoid complex and the pharyngeal areas must be evaluated in order to determine if one of the structures discussed in this paper might be the offending structure, producing the undiagnosed throat and craniofacial pain.

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