



COMMON CAUSES OF NON-DENTAL OROFACIAL PAIN

Introduction

Each day, all of us engaged in treating patients who suffer from orofacial pain are required to make important decisions concerning the cause, diagnosis and treatment of these various types of facial pain. Head and orofacial pain provide a tremendous challenge to the clinician and are taxing and frustrating to the one afflicted, not to mention family members. At least two major reasons account for this observation. Anatomically, the orofacial region is one of the most highly innervated areas of the body, especially the oral cavity. Psychologically, we human beings are known by our faces; the most complex and expressive area of our bodies. Through our faces we share our emotions; all we feel can be expressed through this very vulnerable and prominent portion of our body. The first and last signs of life are conveyed through our faces. Obedience to pain is most fully conveyed by this mask we wear, attempting in most situations, to hide within us this most private event.

In addition, the head and face are subjected to chronic or recurring pain more than any other portion of the body (Merritt, 1968). Further, the most frequent source of facial pain is dental disease (Mumford, 1982) and therefore, dentists are usually the common type of doctor consulted for facial pain. It is fortunate for all when a patient presents with a toothache and it's discovered that there's obvious disease such as a grossly carious tooth. However, every practitioner has examined the patient who complains of facial pain but yet, no evident etiological factor can be located. Thus may begin a long and involved diagnostic process which may or may

not lead to a definitive answer concerning the cause of the pain. Unfortunately, the dentist may be just one in a long line of doctors consulted for the relief of pain by patients. For example, our office was the sixth, on average, that was consulted concerning undiagnosed facial pain (Shankland, 1995).

What's the answer for the busy practitioner concerning effective diagnosis and treatment of facial pain? In this writer's opinion, *the only answer is obtaining an accurate diagnosis*. Then, decisions can be made pertaining to treatment. In this article, two common causes of frequent facial pain, other than those of odontogenic (dental) origin, will be presented. The diagnosis and treatment of these pain disorders will also be discussed.

Syndromes

Before the topic of pain disorders can be addressed, a brief discussion concerning syndromes (disorders) must be conducted. The term *syndrome* is defined as the aggregate of signs and symptoms associated with any morbid process, and constitute together the picture of the disease (Stedman, 1982). Signs, or what the physician objectively discovers, and symptoms, what the patient subjectively reports, are observed and gathered during the initial interview and physical examination. A syndrome comprises several signs and symptoms, not all of which need be present to confirm an accurate diagnosis.

By contrast, a *disease* is a more specific clinical entity; generally, an identifiable group of signs and symptoms with a recognized etiological factor and consistent anatomical

*Illness is the doctor
to whom we pay most heed;
to kindness, to knowledge
we make promises only;
pain we obey.*

Marcel Proust

alterations. All of these must be present to constitute a disease. A disease usually has a known etiological factor; a syndrome may have many different factors. A disease is depicted by a fairly specific set of signs and symptoms. However, a syndrome usually exhibits a broader group of signs and symptoms which may be common to other syndromes. Finally, a disease usually manifests itself by affecting consistent anatomical structures; a syndrome, while affecting fairly specific anatomical regions, also may produce referred pain and dysfunction which can confuse both the patient and doctor.

Therefore, a disease is usually consistent in its historical pattern and clinical presentation; a syndrome, sometimes consistent, may vary greatly concerning history and clinical manifestation. An example of a disease is chronic periapical periodontitis, or an abscessed tooth. By contrast, an example of a syndrome is temporal tendinitis. The former is recognizable by even the third-year dental student and presents with consistent symptoms; the latter often presents various symptoms and may confuse even the most experienced practitioner.

Temporal Tendinitis

Temporal tendinitis is simply a tendinitis of the insertion of the temporalis muscle at the coronoid process of the mandible. Just like tendinitis of any other tendon in the body, temporal tendinitis involves inflammation of the tendon with subsequent symptoms and referred pain.

The temporalis muscle is a broad, thick muscle which originates from the temporal fossa of the skull and the deep surface of the temporal fascia. The fibers, divided into anterior, middle, and posterior divisions, join together as they descend, passing deep to the zygomatic arch, and insert as a tendon into the coronoid process of the mandible (Fig. 1). Two separate heads form this tendon: the short or lateral head, which inserts into the coronoid process tip and the long or medial head, which inserts into the anteromedial border of the ascending ramus of the mandible. As with all muscles of mastication, the temporalis muscle is innervated by the third division of the trigeminal nerve (specifically, the anterior and posterior deep temporal nerves).

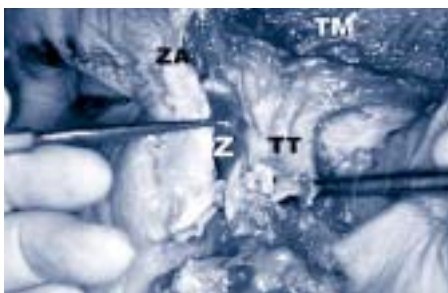


Figure 1: The temporalis muscle and accessory belly (newly described muscle). Legend: TM: temporalis muscle; TT: temporalis tendon; Z: zygomandibular muscle; ZA: zygomatic arch (cut).

Recently, a newly described temporal muscle has been reported (Shankland, 1995; Shankland, 1996). This muscle, now known as the zygomandibularis, originates from the posterior region of the orbital part of the zygomatic (malar) bone, sphenozygomatic suture, and outer lateral region of the greater wing

of the sphenoid bone (Fig.1). Although this muscular structure might be confused with the anterior belly of the temporalis muscle, its innervation and blood supply are separate from those of the temporalis.

The actual process of insertion tendinosis can be described as one of degeneration and inflammation which develop in the narrow bony projections (Sharpey's fibers) (VanderWiel, 1983) inserting the muscle via its tendon into bone. However, the detailed knowledge of the disorder of tendinitis is still lacking (Heuleu, 1991). In addition to the coronoid process, insertion tendinosis has been reported in other bones such as the atlas (Fahlgren, Jansa & Lofstedt, 1966), the transverse processes of cervical and lumbar vertebrae (Belart, 1957), the humerus (Erickson, et al., 1992), and the trapezoid (Schmidt, 1987).

The symptoms of temporal tendinitis, which are similar to internal derangement of the temporomandibular joint (Ernest, et al., 1987), (a displacement of the articular disc) are as follows: (1) temporomandibular joint (TMJ) pain; (2) ear pain and pressure; (3) maxillary posterior odontalgia, especially the maxillary teeth; (4) pain radiation from the zygoma to the eye; (5) temporal headache with pain radiation to and behind the ear, to the occiput and the neck; and, (6) aching pain at the attachment of the tendon to the coronoid process (Table 1).

Table 1: Areas of Reported Pain With Temporal Tendinitis

<i>Symptoms of Temporal Tendinitis</i>
Temporomandibular Joint Pain
Ear pain and pressure
Maxillary Posterior Odontalgia
Pain in zygoma and eye
Temporal headache
Pain at the coronoid process

The pain in the TMJ is often accompanied by restriction of mandibular maximum incisive opening due to muscle splinting and pain from the stretching of the tendon during wide opening movements. The TMJ pain does not originate from the joint itself, demonstrable by the lack of pain with palpation of the lateral pole of this joint. However, the joint may be tender in the case of capsulitis (Shankland, 1993), discal dislocation or active osteoarthritis of the joint, all of which may occur together with temporal tendinitis. Temporal tendinitis is the disorder plaguing a patient that initially improves (for about 5 to 10 days) with an acrylic splint or orthotic, but then has the symptoms return, often with more intensity than before the placement of the splint. This may occur because the use of the appliance increases the vertical distance between the surfaces of the maxillary and mandibular teeth, thus causing a prolonged and increased stretching of the degenerating tendon, accelerating the degeneration to a point that the patient may not be a candidate for conservative therapy but destined for surgery. This dramatic, re-precipitation of symptoms following the placement of a splint should immediately warn the clinician to one or two probabilities: (1) the initial diagnosis for which a splint was recommended might have been correct, but also existing concurrently with temporal tendinitis; or, (2) the type or even the use of a splint needs to be reevaluated and perhaps, temporal tendinitis is not present. However, from this writer's clinical experience of over 22 years, the most common disorder producing the above symptoms is temporal tendinitis and the initial improvement with subsequent reoccurrence of symptoms after placement of a splint is practically pathognomonic for this most interesting syndrome.

Two other symptoms, ear pain and especially pressure in the ear, are also quite common. Often, the patient will

have been previously examined by an otolaryngologist with the chief complaint of “water in the ear” or with the feeling of riding in an airplane, being unable to clear the pressure feeling in the ear. Otoscope examination of the ear generally is normal. The patient may even complain of a reduction of hearing in the ear or ears affected.

Posterior odontalgia is another frequent symptom of temporal tendinitis. Although all posterior teeth may ache, throb, or be sensitive, generally the maxillary teeth appear to be affected most often and with greater severity. Patients have often consulted one or several dentists, oral surgeons, and even endodontists. Unfortunately, in an attempt to treat these patients, unnecessary and invasive procedures have often been attempted with no improvement and frequently, an exacerbation of complaints. Radiographic examination of the sensitive posterior teeth will be normal. Occlusal analysis may reveal various types of malocclusion; however, the degree of malocclusion usually does not appear to match the intensity of the patient's complaints. Cracked tooth syndrome may be suspected but rarely is this disorder accompanied with ear pain and pressure. It's also interesting to note that posterior tooth pain may occur even when no teeth are present.

A very common additional symptom of temporal tendinitis is pain radiation from the zygoma to and behind the ipsilateral eye. This pain pattern may be due to the embryological development of the associated structures but also to the development of either tendinitis or myofascial trigger points in the zygomandibularis muscle (Shankland, 1995; Shankland, 1996). The report of this pain may also simply be referred pain from the temporalis muscle as described by Travell and Simons (1983).

Prolonged opening of the mouth, bruxism and clenching, the placement of a splint or increasing the vertical dimension (especially, rapidly as with

placement of multiple dental restorations), all may be mechanical etiological factors producing or accelerating the development of tendinitis. Stress, causing an increase in parafunctional activities and a subsequent increase in muscle tension may also cause temporal tendinitis.

The diagnosis of temporal tendinitis is confirmed by three criteria: (1) the history of complaints; (2) tender palpation of the temporal tendon; and, (3) successful relief of symptoms following an anesthetic block of the deep temporal nerves (Ernest, et al., 1987; Shankland, 1995; Shankland, 1996). Historically, the patient will report the approximate time of onset of symptoms or the worsening of symptoms following prolonged opening of the mouth (e.g., a long dental appointment or intubation during a general anesthetic), trauma to the head or mandible, or after emotional distress. There may be a separate temporomandibular disorder; however, usually the patient will be able to determine when the temporal tendon pain originated. Unfortunately, the doctor is presented with the dilemma of having to make an accurate diagnosis for proper treatment.

To further aid in diagnosis, positive palpation of the temporal tendon will help confirm the diagnosis of temporal tendinitis. Often, students are taught and clinicians perceive that they are palpating the lateral pterygoid muscle during a routine examination. However, it is not anatomically possible to palpate this muscle (Johnstone & Templeton, 1980), which is protected by being placed deep within the infratemporal fossa and by the overlying superficial head of the medial pterygoid muscle. Palpate the temporal tendon by placing pressure on the tendon with a gloved index finger or a blunt object. A positive finding is tenderness to palpation of the tendon. The patient may react by trying to move away from your probing finger. Pain may also be produced in the temple, zygoma and ear.

The most accurate diagnostic procedure is the use of the anesthetic block of the deep temporal nerves. The intraoral injection site is lateral to the maxillary second molar along the medial aspect of the coronoid process. Deposit approximately one to one-and-a-half cubic centimeters at the tip of the coronoid process to block the deep temporal nerves to produce anesthesia to the tendon. If the diagnosis is temporal tendinitis, resolution of symptoms will occur within 5 to 10 minutes following the anesthetic injection. Perhaps the most dramatic will be the reduction or elimination of the ear pressure. This will be quite noticeable to the patient. All symptoms should be totally eliminated or nearly so to establish an accurate diagnosis.

Treatment for temporal tendinitis can be divided into two general categories: conservative and surgical. When treated appropriately and aggressively, conservative treatment has been shown to be successful 96% of the time (Shankland, 1991). However, both doctor and patient must agree, as with all pain syndromes, as to what is considered “successful.” Generally, total elimination of all pain all the time is quite unreasonable, however, resumption of fairly normal function and reduction of pain to a point of tolerance with little or no medication use may be considered a successful outcome of treatment.

Conservative treatment for temporal tendinitis consists of several steps. First, after confirmation of the diagnosis with the use of a local anesthetic block, inject 1/2 to 1 cc of an anti-inflammatory medication into the temporal tendon near the coronoid process tip. Caution the patient about the possible development of a post-injection flare which may be even more painful than the original symptoms. The presence of this reaction is a good indicator that the degenerative changes in the tendinous attachment are not treatable with conservative modalities. Prescribe a

non-steroidal anti-inflammatory drug (NSAID twice a day for 5 days and then once daily for two weeks). Instruct the patient to apply ice extraorally over the anterior portion of the temporalis for 25 minutes out of 30 for approximately 4 hours after the injections are given. Recommend moist heat later that day and the next few, if necessary. Also, prescribe a muscle relaxant to be used for the next 10 days and place the patient on a soft diet and limited mandibular use. If the patient exhibits signs of parafunctional activities (e.g., bruxism, clenching or both) or if an additional temporomandibular disorder is present, fabricate an NTI clenching suppression appliance.

Re-evaluate the patient in 10 to 14 days. If improvement is noted, repeat the injection procedures, if necessary. Always give the local anesthetic prior to the anti-inflammatory injection for patient's comfort and to provide sympathetic benefits (Loftrom & Cousins, 1988), (e.g., improved edema drainage, reduction of pain, improved blood flow). Continue the NSAID, muscle relaxants, and nighttime use of the NTI (if necessary). Repeat the injection procedures every 2 to 3 weeks. Usually 2 to 4 injection sessions will be necessary if conservative treatment is to be successful.

Conservative therapy is greatly improved in severe cases by the use of specific modalities of physical therapy. Apply ultrasound to the anterior temporal region, masseter muscle, and lateral TMJ capsule. Next, administer electrogalvonic stimulation (EGS) with the negative current over the temporal tendon. Instruct the therapist to perform spray and stretch as described by Travell and Simons (1983). Finally, teach the patient passive stretching home exercises, after the application of moist heat.

The most conservative and successful surgical approach to this disorder is the use of radiofrequency thermoneurolysis (Cooperband & Goodrich, 1989; Ernest,

et al., 1987; Graff-Radford, 1992; Gregg & Small, 1986; Gregg, Banergee & Ghia, 1978; Gregg, 1971; Klemons, 1993; Shankland, 1984; Shankland, 1995; Ziccardi, Braun & Ochs, 1992).

Neural Inducing Cavitation Osteonecrosis (NICO)

As stated at the beginning of this article, chronic orofacial pain is both common and frustrating to the patient and doctor. A 23.3% risk of developing chronic facial pain exists by age 50 and a 33.8% risk by age 70 (Von korff, Dworkin, Le Resche & Kruger, 1988). Many researchers and clinicians alike contend that peripheral neural damage is a major etiological factor for the development of trigeminal-like pain. One theory (e.g., the peripheral theory) states that a low-grade, chronic, intraosseous infection near a branch of the trigeminal nerve may produce neuronal degeneration or demyelination producing inappropriate nociceptive signals to the central nervous system (Roberts & Person, 1979). This typically explains the pain of neuralgia inducing cavitation osteonecrosis (NICO) or cavitation lesions (Figure 2).



Figure 2: Mandible upon autopsy demonstrating several NICO lesions. N: NICO lesions; IA: inferior alveolar nerve; MN: mental nerve.

Simply defined, osteonecrosis is dead bone; osteomyelitis is inflammation of the bone marrow and adjacent bone. In 1915, G.V. Black first described these jawbone lesions as follows: "An osteomyelitis-like bone disease which seemed

not to be a true infection, but rather a slow, progressive, unexplained death of cancellous bone and marrow, cell by cell." These lesions were not new to dentistry, for dental surgeons in the United States accepted this theory in the 19th century (Grandin, 1841; Tomes, 1848). The theory, of osteomyelitic and osteonecrotic jawbone lesions producing trigeminal neuralgia-like pains, is supported by laboratory evidence that Gasserian (trigeminal) ganglion degeneration occurs after damage to the pulp of teeth (Black, 1974; Westrum & Canfield, 1976) and by the high rate of relief of long-term trigeminal neuralgia pain after surgical treatment of the jawbone cavitations which failed to respond to traditional medical procedures.

The symptoms of NICO are: (1) a history of undiagnosed facial and cervical pain; (2) a history of tooth extraction, which may have occurred decades earlier; (3) the presence of trigger areas; and (4) normal radiographic findings.¹

Patients suffering from cavitation lesions will generally have a history of tooth extraction perhaps many years before the onset of pain complaints. Also, a common history includes the placement of a large restoration or crown, followed by the development of sensitivity or pain, neither of which can be resolved. The patient then demands or the doctor recommends endodontic therapy, but unfortunately, the pain continues.

Etiological factors known to influence the development of NICO lesions are listed in Table 2.

¹ Normal radiographic findings as defined by traditional teaching of oral radiology.

However, as one becomes familiar with evaluating panoramic radiographs, it is soon apparent that radiographs which contain NICO lesions are anything but normal.

Table 2: Common Etiologies of Osteonecrosis of the Jaws

<i>Etiologies of Osteonecrosis</i>
Blunt trauma
Dental trauma in the form of surgical procedures
History of a large dental restoration followed by development of pulpitis and ultimately, chronic periapical periodontitis
Heavy and/or chronic cortisone use
Estrogen replacement therapy
Radiation therapy
Variable atmospheric pressures in occupation
Alcoholism
Sickle cell anemia
Heavy cigarette smoking
Pregnancy
Thrombophilia
Hypofibrinolysis

The underlying microscopic problem is vascular insufficiency, with intramedullary hypertension and multiple intraosseous infarctions occurring over time (Bouquot & LaMarch, 1999; Westrum & Canfield, 1976). In other words, an occlusion of one or several of the tiny intramedullary vessels by thrombi within the maxilla or mandible occurs, producing ischemic changes distal to the vessel blockage, ultimately resulting in the development of osteomyelitis and/or osteonecrosis. These thrombi may be caused by rigid erythrocytes blocking the vessels of the bone marrow. Glueck achieved a substantial breakthrough when he reported that a majority (65% to 87%) of osteonecrosis patients had major hereditary or acquired clotting disorders that had not previously been diagnosed or even suspected (Glueck, Freiberg, et al., 1994; Glueck, McMahon, Bouquot, et al., 1996).

The best methods to diagnose NICO lesions are the evaluation of panoramic radiographs and elimination of pain by using anesthetic blocking (Shankland,

1993) without vasoconstrictor. Treatment of NICO lesions always requires surgical removal of the affected bone, whether the actual diagnosis is osteomyelitis or osteonecrosis. Unfortunately, if an infected bony area does not have a blood supply, no remedy, be it homeopathic or allopathic, can be distributed within the region. Also, localized injections of homeopathic remedies using intrabony injections have not been successful long-term. Thus, only surgery gives the patient a chance for any degree of recovery.

Surgery consists of surgical curettage of the bony lesions, with total removal of all the contents of the cavitation lesion and a small amount of surrounding normal bone. Also, any teeth located within these pathologic regions must be removed, as they are shown to be non-vital upon post-operative examination.

Summary

Two disorders that cause facial pain and which may be confused with pain of odontogenic origin were presented. As with all types of pain, the key to appropriate treatment is first obtaining an accurate diagnosis, especially before non-reversible procedures are attempted. The general dentist is frequently consulted by patients because of the prevalence of toothache pain in our society. Pain practitioners must acquaint themselves with disorders other than the routine dental diseases that produce facial and head pain. Such improvement in diagnostic skills will only help doctors better serve their patients.

Also contained within this paper is the report of a newly described muscle termed the zygomandibularis. It too, may develop tendinitis at its insertion into the mandible which may produce similar to temporal tendinitis.

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Clinical Meeting Report

11TH ANNUAL CLINICAL MEETING



This year's Annual Clinical Meeting was in Las Vegas, Nevada, September 21-24, 2000. This state-of-the-art meeting assembled the most outstanding clinical expertise in the interdisciplinary pain management field to provide a comprehensive view of pain management trends for the new millennium. The interdisciplinary professionals in attendance at the meeting had an opportunity to interact with 107 faculty and 88 exhibitors. The feedback has been wonderful:

"Once again you have organized a wonderful meeting program. This is why I have attended your program annually for the past six years."

Karlus Artis, MD

"Once again I applaud you for the outstanding accomplishment of the Year 2000 meeting. It amazes me the energy that you have to set forth the excellent standards obvious in the convention."

A.V. Anderson, MD, DC

"Thank you very much for a wonderful meeting."

Valapet Sridaran, MD

A monitor from the California Psychological Association Accrediting Agency wrote: *"I also conducted brief informal interviews of the participants who attended those courses I was unable to directly monitor. In general, their observations were consistent with my overall*

impression of the conference: Excellent organization, presentation and content....As a group, the instructors were superb...Thank you for allowing me to audit this outstanding conference."

Richard E. Landis, PhD

The American Academy of Pain Management continues to integrate the various pain management disciplines and continues to provide quality continuing educational opportunities that are truly interdisciplinary in nature.

