Facial Pain

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Abstract: Facial pain is a debilitating disorder if left untreated. Too often, patients are labeled as having psychopathology when face pain etiology is unclear. These patients are categorized as "atypical," "idiopathic," or "psychogenic." Cases of facial pain involving neuropathic, neurovascular, musculoskeletal, as well as intracranial and extracranial systems will be reviewed. Peripheral and central mechanisms associated with these disorders are used to provide an update of these frequently seen clinical issues.

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Idiopathic, when referring to a medical problem suggests there is something unknown, and does not define the problem. The same applies to terms incorporating the word "atypical." It has been reported that patients described as "atypical" or "idiopathic" all have an ascribable diagnosis if evaluated by someone with more experience.[1] The International Association for the Study of Pain[2] and the International Headache Society (I-H-S)[3] take the position that there are better terms for facial pain diagnoses other than "atypical" or "idiopathic." These terms have long been used as a wastebasket for situations where the clinician can not make a diagnosis.

The term idiopathic facial pain has included diagnoses such as atypical facial pain, atypical odontalgia, masticatory muscle disorders, and traumatic neuralgia. These categories serve to perpetuate our limited knowledge of pain presenting in the face when the etiology is unclear.

The most comprehensive facial pain classification to date incorporates the I-H-S criteria and expands it to include disorders that were not clearly defined.[4] Tooth pain is one of the most common causes of orofacial pain. Most frequently, dental pain is related to dental caries, presenting as a reversible pulpitis. The reversible pulpitis is characterized by poorly localized pain, often sensitive to hot or cold stimuli. The reaction to the noxious stimulus (hot or cold) disappears soon after its removal. Eventually, when the carious lesion invades the pulp, an irreversible pulpitis begins. This is characterized by a lingering reaction to noxious stimuli such as hot or cold. If the microorganisms and inflammatory products invade the area around the root apex (periapical), this is called a periodontitis and may present with toothache associated with chewing, touch, and percussion sensitivity. Periapical pathology may be observed as an area of increased radiolucency on radiograms. In clinical practice, differentiating reversible and irreversible pulpitis is difficult. In situations where the diagnosis is not obvious, careful observation over days or weeks is recommended. An intermittent pain that is triggered by biting on an offending tooth characterizes a cracked tooth syndrome. Unfortunately, the cracks are often difficult to find and do not show on all x-ray images. This pain is often confused with pulpitis or trigeminal neuralgia (TIC), resulting in frustration and unnecessary treatment. Using a tongue blade, placed carefully so that bite pressure is transmitted to an individual cusp, and then asking the patient to bite and release, may evoke sharp, electrical pain on release. This is indicative of a cracked tooth. A "tooth slooth" is a specific instrument designed for this function.

Understanding and Diagnosing Neuropathic Pain

Neuropathic pain suggests that there has been some tissue or nerve injury.[6] With injury there is a permanent peripheral nerve and or central nervous system (CNS) change. It is surprising that with all that the human endures; falls, scrapes, fractures, surgery, etc—that so few patients develop chronic pain. This is likely due to the brain's ability to inhibit or control the permanent changes seen following tissue injury.[5]

We should start out by differentiating "transient pain" from "chronic pain." Short-lived pain following a stimulus that is potentially tissue damaging, also referred to as acute pain, is a protective mechanism. Acute pain resolves in an appropriate time period and then normal function is restored. What happens when the stimulus results in a chronic pain? Once the injury seems to have healed there is pain that is nonprotective. It is postulated that this may be due to central and peripheral nervous system changes.[6] These changes may include the presence of ongoing peripheral nociception, CNS sensitization, or down-regulation of CNS inhibition.[7]

Clinically neuropathic pain can be divided into continuous and intermittent temporal patterns. Table 1 is a clinical classification for neuropathic facial pain.

Intermittent neuropathic pain presents clinically as an intermittent startling, stimulating, electric sharp, or burning pain. This is typically seen in trigeminal neuralgia (TIC), glossopharyngeal neuralgia, and nervous intermedius neuralgia. These intermittent neuralgias are triggerable, usually by non-noxious stimuli. Vascular nerve compression is the proposed etiology.[8] Compression may also be secondary to other structures, including tumors and bony growths (eg, Eagles syndrome).[9,10]

The continuous neuralgias are more common yet harder to explain. There are a significant number that follow dental or surgical therapies, where no obvious error or negligent procedure was performed. It is postulated that the pain mechanism is akin to that occurring in phantom pain, after limb removal. Most cases occur in females, usually in their fourth decade.[11,12] There must be a lesion in the trigeminal nervous system, peripherally or centrally, to cause the continuous traumatic neuralgia—dysesthesia.[13,14] Sex-based differences have been seen in many pain disorders. It is not unusual to see a high frequency of temporomandibular disorders (TMDs), migraine, and fibromyalgia among females. The relationship and role of sex hormones in the generation and perpetuation of central sensitization is not fully understood but is obviously important.[15,16] In a neuropathic pain model using partial sciatic nerve ligation, female rats were more likely to develop allodynia.[15] In studies comparing female rats that have been ovariectomized, there is a greater chance that those with estrogen were more likely to develop allodynia after injury than those without estrogen.[16]

Overview of Trigeminal Neuralgia

According to the Headache Classification Committee of the I-H-S,[3] TIC is described as "a painful unilateral affliction of the face, characterized by brief electric shock-like (lancinating) pain

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TABLE 1. Neuropathic Orofacial Pain

Intermittent
Trigeminal neuralgia
Glossopharyngeal neuralgia
Nervous intermedius neuralgia
Continuous
Traumatic trigeminal neuralgia—trigeminal dysesthesia
Trigeminal dysesthesia—sympathetically maintained
Post herpetic neuralgia
Diabetic neuropathy

limited to the distribution of 1 or more divisions of the trigeminal nerve. Pain is commonly evoked by trivial stimuli including washing, shaving, smoking, talking, and brushing the teeth, but may also occur spontaneously. The pain is abrupt in onset and termination may remit for varying periods." Symptomatic TIC is described as "pain indistinguishable from TIC, caused by a demonstrable structural lesion." This lesion is usually a tumor, such as an acoustic neuroma, or may be due to demyelination, as seen in multiple sclerosis. Demyelination is also reported in compression of the nerve root as it exits the pons, secondary to vascular compression. If there is tissue or nerve injury, there may be an ensuing continuous TIC, which is usually referred to as traumatic trigeminal neuralgia—dysesthesia (TD). TIC is usually unilateral and only occurs bilaterally in 4% of subjects. There is no genetic link to the disorder. The average age at onset is between the sixth and seventh decades, with women slightly more affected than men in a ratio of 3:2. The bright, stimulating, electrical shock-like pain perceived is short-lived, lasting seconds to minutes. If not questioned carefully, the patient may report that the pain is continuous, as intermittent attacks may last all day. Additionally, there is often a dull pain associated with TIC, or the sharp volleys come and go continuously. The author believes the persistent aching pain may be secondary to a reflex muscle splinting of the muscles of mastication, and can be controlled with stretching exercise and a vapocoolant spray (myofascial pain). Mechanical maneuvering within the trigeminal sensory system usually triggers TIC pain. The area from which the pain is activated has been described as a trigger zone. Characteristically, trigger zones occur around the supraorbital, infraorbital foramina, the inner canthus of the eye, lateral to the ala, and over the mental foramen. Trigger zones are also common intraorally. Pain is not elicited from the trigger zone if deep pressure is used, or during a latency period between paroxysms. Anesthetizing the trigger zone with topical or injected local anesthetic agents may terminate the pain for the duration of anesthesia. The second and third trigeminal nerve divisions are most commonly affected. The first-division cases occur less frequently than 5%. Often, there is ipsilateral reflex facial spasm, hence the term "tic douloureux," which has been used synonymously with TIC. When present, compression of the trigeminal and facial nerves may be implicated.

It is postulated that TIC may be due to a trigeminal nerve focal demyelination at any point along its course. Exploration of the posterior cranial fossa reveals that between 60% and 88% of cases have trigeminal nerve root vascular compression. The compression is present in the posterior cranial fossa as the trigeminal nerve exits the pons. This has been postulated to set up a centrally mediated disinhibition of pain modulation and/or peripheral repetitive ectopic action potentials. Once there is sensitization, there may be increased afferent fiber activity and enhanced response to tactile stimulation, resulting in trigeminal nucleus interneuron discharge and heightened trigeminothalamic neuron discharge, producing pain. Tumor has been implicated in up to 6% of cases. These include acoustic neurinomas, cholesteatomas, meningiomas, osteomas, and angiomas. Aneurysms and adhesions have also been implicated. Although the pain may be typical of TIC, there are usually additional symptoms or cranial nerve deficits present depending on the tumor size and location. When patients are in the 20- to 40-year age range and present with TIC, multiple sclerosis should be considered. Patients in this age range are relatively young for TIC, and although there may be no other signs associated with multiple sclerosis, further evaluation should be considered. The weight loss described characteristically occurs in TIC patients as they cannot eat secondary to pain triggered by chewing. However, tumor is also associated with weight loss, and once again should trigger further investigation. It is standard of care to obtain brain MRI with and without gadolinium, with specific attention to the posterior cranial fossa.

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Roberts[17] proposed that bony cavities found in the alveolar bone may be the cause of TIC, and that repetitive curettage of these cavities is curative. The presence of the bony cavities is not disputed, but their role as a cause of TIC is highly controversial. The author does not believe they are a cause of TIC.

Pretrigeminal Neuralgia

Sir Charles Symonds first described pretrigeminal neuralgia (pre-TIC).[18] Fromm and Graff-Radford have described a further 16 cases in which patients initially present with a dull continuous aching toothache in the upper or lower jaw, and in whom the pain changed to classic TIC. Further, they describe 7 cases in which the continuous pain was successfully treated with traditional TIC therapies. The diagnosis of pre-TIC is based on the following criteria: (1) description of pain as dull toothache, (2) normal neurologic and dental examinations, and (3) normal CT or MRI scan of the head.[19]

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Overview of Traumatic Trigeminal Neuralgia—Dysesthesia

The neuropathic pain following tissue or nerve injury in the trigeminal nerve distribution may be called a traumatic trigeminal neuralgia—TD. TD is defined as a continuous pain following complete or partial damage to a peripheral nerve or CNS structure. The pain is described as a continuous, burning numbness, and often pulling pain (given in Table 2).

TABLE 2. Criteria for Traumatic Trigeminal Neuralgia—Dysesthesia

History of trauma
Continuous pain
Associated hyperalgesia and allodynia
Temperature change
Block effect (sympathetic vs. somatic)

The neuropathic pain following tissue or nerve injury in the trigeminal nerve distribution may be called a traumatic trigeminal neuralgia.

There are a number of mechanisms described as causing traumatic induced neuropathy. They can be described as (*a*) peripheral sensitization, (*b*) ectopic activity due to sodium channel expression, (*c*) central sensitization, (*d*) A beta fiber reorganization, (*e*) alteration in central inhibition systems, and (*f*) sympathetically maintained pain due to alpha receptor sprouting. More than 1 mechanism may be active to create individual clinical presentations.[20]

(*a*) Peripheral sensitization: Once stimulated or traumatized the peripheral nociceptors will release a variety of peptides including substance p, calcitonin gene related peptide, and neurokinins. This results in a peripheral sensitivity that is characterized by an increased response to non noxious (allodynia) and noxious stimuli (hyperalgesia). (*b*) Ectopic firing results after trauma, likely due to the sprouting of sodium channels. The nerve is easily depolarized and spontaneous shooting pains result. (*c*) Central sensitization develops once the peripheral stimuli trigger second or third order neurons. Once again there is the release of peptides, through now in the dorsal horn (trigeminal nucleus) or thalamus, resulting in pain being generated without the presence of an ongoing peripheral stimulus. The role of glial cells and release of proinflammatory cytokines also play a role in the CNS causing pain.[21] (*d*) A beta fiber reorganization is another mechanism causing centrally mediated pain. It is described as occurring when the c fibers are destroyed, which usually have their second order neurons in lamina II, allowing for spontaneous growth of A beta fibers from lamina III into lamina II, making proprioceptive and temperature stimuli activate c fiber second order neurons. Hence non nociceptive activity causes pain. (*e*) Alterations in central inhibition or disruption of normal pain inhibiting systems also may cause chronic pain. The brain has powerful opioid and non-opioid inhibitory systems. Neurochemicals such as serotonin, norepinephrine, and GABA all help the brain modulate nociception. If these inhibitory pathways are not efficient chronic pain may exist. (*f*) Sympathetically maintained pain (SMP) may be a result of alpha receptor sprouting on peripheral nociceptors resulting in norepinephrine release peripherally causing pain.[22] There is evidence that following neural injury the sympathetic innervation in the dorsal root ganglia increase with age. It is not surprising that there is a higher incidence of neuropathic pain as we age. SMP is aggravated by non-noxious stimuli and interrupted temporarily by sympathetic block or α-adrenergic block with phentolamine.[23–25] The therapy for TD is aimed at reducing peripheral nociceptive inputs and simultaneously enhancing CNS pain inhibitory systems.[25]

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Topical Applications

The use of topical therapies has not been well studied. There is some evidence that capsaicin (Zostrix) applied regularly will result in desensitization and pain relief.[26] The recommended dose is 5 times per day for 5 days then 3 times per day for 3 weeks. If the patient can not withstand the burning produced by the application, the addition of topical local anesthetic, either 4% lidocaine or EMLA is useful. Clonidine can be applied to the hyperalgesic region by placing the proprietary subcutaneous delivery patch where it is most tender. Alternatively, the use of a 4% gel can be compounded and delivered over a larger area. For local intraoral application, a neurosensory stent has been conceived. After an oral impression, an acrylic stent is manufactured to cover the painful site.[27] The topical agent is applied to the gingival surface and placed intraorally 24 hours per day.

Topical clonazepam (0.5–1.0 mg, 3 times per day) has been effective at reducing a burning oral pain.[28] Patients were instructed to suck a tablet for 3 minutes (and then spit out) 3 times per day for at least 10 days. Serum concentrations were minimal (3.3 ng/mL) 1 and 3 hours after application. Woda hypothesized that there was a peripheral, and not a central, action disrupting the neuropathologic mechanism.[28]

Procedures

Neural blockade is very effective in differentiating SMP from sympathetically independent pain. It may also be effective in controlling SMP if used repetitively. Stellate ganglion blocks, sphenopalatine ganglion blocks and phentolamine infusion have been described as useful in obtaining a chemical sympathetic block. The author has not had significant benefit using phentolamine infusion in facial pain. This is supported by Scrivani who used a 30 mg infusion without benefit.[27] Stellate ganglion blockade has been compared with sphenopalatine ganglion block in traumatic neuralgia. Approximately 70% of the patients receiving 2 stellate ganglion blockades and 80% of the patients receiving sphenopalatine ganglion blocks have a greater than 60% reduction in pain. This response is typically short-lived with the longest response being 3 months. Repeated blockade may help patients but long lasting responses are infrequent. Radiofrequency applications to the stellate ganglion and sphenopalatine ganglion also provide unpredictable responses and the usual benefit is around 3 months.[29] Current exploration of gamma knife application to the sphenopalatine ganglion in traumatic trigeminal neuralgia is underway.

Neural blockade in TIC produces very temporary effect. Doing multiple repetitive blocks over time does not produce an enhanced response.

Lidocaine infusion (200 mg over 1 hour) may be used therapeutically in various forms of neuropathic pain.[30,31] It is suggested that response to intravenous lidocaine may predict who responds to the lidocaine analogue mexilatine. Sinnott et al used an animal model to demonstrate that the there is a minimal lidocaine concentration (2.1 μg/mL) to abolish allodynia.[32] They also describe a ceiling effect. Many animals with experimentally induced allodynia did not obtain persistent relief. They suggest separate

TABLE 3. Common Antidepressants Used in Traumatic Trigeminal Neuralgia—Dysesthesia

Medication Trade Name	Dosage (mg/d)
Amitriptyline	10/150
Desipramine	10/150
Doxepin	10/150
Duloxatine	20/90
Imipramine	10/150
Nortriptyline	10/150
Trazedone	50/300
Venlaflaxine	37.5/225

physiological mechanisms, with differing pharmacologies, may account for variability and postulate there are different aspects of neuropathic pain.

Pharmacology

Tricyclic antidepressants: It is well documented that tricyclic antidepressants are effective in many pain problems. Solberg and Graff-Radford have studied the response of amitriptyline in traumatic neuralgia. It is noted that the effective range is 10 to 150 mg per day usually taken in a single dose at bedtime.[12] Many antidepressants may be used (as mentioned in Table 3).

Membrane stabilizers: These medications include the anticonvulsants, lidocaine derivatives and some muscle relaxants. They have been classically used in intermittent sharp electric pains. Table 4 summarizes the common medications in this group and their doses.

Behavioral Strategies

Prior to beginning therapy, it is common to perform a behavioral assessment with appropriate testing. Following the behavioral evaluation, management is directed at the factors, which may impact treatment and determining the most appropriate interventions. Consideration should be given to the following factors: (1) Behavioral or operant; (2) emotional; (3) characterological; (4) cognitive; (5) side effects; (6) medication use; and (7) compliance. Therapy such as cognitive and behavioral management techniques, relaxation, biofeedback, and psychotherapeutic and psychopharmacological interventions may be useful.

TABLE 4. Common Membrane Stabilizers Used in Traumatic Trigeminal Neuralgia—Dysesthesia and Trigeminal Neuralgia

Medication Trade Name	Dosage (mg/d)
Baclofen	10/80
Carbamazepine	100/1200
Oxcarbazepine	150/1800
Gabapentin	300/3000
Klonopin	0.5/4
Lamotragine	12.5/100
Levitricitam	250/2500
Pimozide	2/12
Phenytoin	100/400
Pregabalin	25/600
Topirimate	25/400
Valproic acid	125/2000
Zonisamide	50/200

Surgery

Although not suggested as a therapeutic modality for traumatic trigeminal neuralgia—dysesthesia, surgery is an excellent alternative for trigeminal neuralgia. The most effective surgical approach remains microvascular decompression.[33] Advances in microvascular decompression include the use of an endoscopic approach. This allows clearer observation and is less traumatic.[34] Gamma knife radiosurgery is a recent advance for trigeminal neuralgia.[35] This technique offers a relatively noninvasive means for lesioning the trigeminal nerve adjacent to the pons using a 4 mm collimator helmet. Complications are rare and to date the author has seen a number of cases of traumatic trigeminal neuralgia—dysesthesia attributed to the Gamma Knife procedure out of more than 150. The incidence of neuropathic pain is less than 1%. The use of radiosurgery may offer an effective procedure for traumatic neuralgia if aimed at the SPG. Balloon gangliolysis is a relatively non invasive and effective therapy for trigeminal neuralgia; a good outcome is in the 80th percentile.[36]

Neurovascular Pain That May Be Confused With Trigeminal Neuralgia

There are 2 disorders that may present with intermittent sharp, electrical pain in this category: chronic paroxysmal hemicrania, and short-lasting unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT).

Paroxysmal Hemicrania

There are cases of episodic paroxysmal hemicrania similar to the chronic version, but with periods of remission. Diagnostic criteria according to the I-H-S are as follows:

At least 20 attacks fulfilling criteria B to E;
Attacks of severe unilateral orbital, supraorbital, or temporal pain lasting 2 to 30 minutes;
Attack frequency above 5 a day for more than half of the time, although periods with lower frequency may occur;
Pain is associated with at least one of the following signs or symptoms on the pain side:

1. Ipsilateral conjunctival injection and/or lacrimation,
2. Ipsilateral nasal congestion and/or rhinorrhea,
3. Ipsilateral eyelid edema,
4. Ipsilateral forehead and facial sweating,
5. Ipsilateral ptosis and/or meiosis,

Headache is stopped completely by indomethacin;
Not attributed to another disorder.

Indomethacin should be used at least in a daily dose of 150 mg orally, or 100 mg by injection, but smaller maintenance doses are often employed.

Short-Lasting Unilateral Neuralgiform Headache With Conjunctival Injection and Tearing

This syndrome is characterized by short-lasting attacks of unilateral pain that are much briefer than seen in any other trigeminal autonomic cephalalgia and is most often associated with prominent lacrimation and redness of the ipsilateral eye. There is no substantial evidence in the literature for episodic SUNCT. The literature suggests that the most common mimic of SUNCT would be a lesion in the posterior fossa, or TIC. I-H-S diagnostic criteria for SUNCT is as follows:

At least 20 attacks fulfilling criteria B to E;
Attacks of unilateral orbital, temporal stabbing, or throbbing pain lasting from 10 to 120 seconds;
Attack frequency from 3 to 200 per day;
Pain is associated with conjunctival injection and lacrimation;
Not attributed to another disorder.

Pharmacologic interventions that may be helpful include the following[37]:

Lidocaine 4 mg/min intravenously;
Carbamazepine 1200 mg daily;
Lamotrigine 200 mg daily;
Topiramate 200 mg daily;
Gabapentin 2400 mg daily.

Surgical interventions that may be helpful include microvascular decompression. Trigeminal neuralgia and SUNCT patients are known to have a blood vessel that may compress the trigeminal nerve root as it exits the pons. Decompressing this nerve by placing a Teflon pad between the vessel and nerve has been reported useful. Both paroxysmal hemicrania and SUNCT have been reported to coexist with TIC.

Temporomandibular Disorders

Functional disorders and pain in the anatomic region of the temporomandibular joint (TMJ) and associated musculature are referred to as TMD.

Epidemiology

TMD epidemiology has not specifically differentiated headache from facial pain. In nonpatient population studies, 75% have at least one joint dysfunction sign (clicking, limited range of motion) and about 33% have at least one symptom (pain, pain on palpation). Out of the 75% with a sign or symptom, less than 5% require treatment and even fewer have headache as the primary pain location.

Etiology

Inflammation within the joint accounts for TMD pain and the dysfunction is due to a disk, condyle incoordination. Muscle pain disorders may include spasm, myositis, muscle splinting, and myofascial pain. The most frequent muscle disorder included in TMD classification is myofascial pain. Although each may be a trigger for headache and occur together they will be discussed separately.

Inflammation. Primary inflammatory conditions of the TMJ include capsulitis, synovitis, and the polyarthritides. Polyarthritides are relatively uncommon and are associated primarily with rheumatologic disease. Inflammatory conditions such as synovitis or capsulitis frequently occur secondary to trauma, irritation, or infection and often accompany other TMD's.[38]

Several proinflammatory cytokines have been detected in painful TMD's, which suggest they may play a role in pain.[39] Capsulitis, an inflammation of the capsule related to sprain of capsular ligaments, is clinically difficult, if not impossible, to differentiate from synovitis. However, the pain related to capsulitis increases during all translatory movements and joint distraction, but not usually during clenching.[40] Both conditions may be accompanied by a fluctuating swelling (due to effusion) that decreases the ability to occlude on the ipsilateral posterior teeth. Pain associated with inflammation is localized to the TMJ capsule and the intracapsular tissues. The pain is typically dull achy, but may be throbbing. It is frequently described as sharp on movements. The pain is continuous, but worsens with jaw function.

Disc Derangement Disorders. Articular disc displacement is the most common TMJ arthropathy and is characterized by several stages of clinical dysfunction that involve the disc-condyle relation. The usual direction for displacement is in an anterior or anteromedial direction although other directions have been described.[41,42]

The causes of disc displacement are not agreed upon; however, it is postulated that in a majority of cases stretched or torn ligaments that bind the disc to the condyle permit the disc to become displaced.[43] An increased horizontal angle of the mandibular condyle has been associated with more advanced TMJ internal derangement.[44,45] Lubrication impairment has also been suggested as a possible etiologic factor of disc displacement.[46] Disc displacement is subdivided into disc displacement with reduction or disc displacement without reduction.

Disc displacement with reduction is described when a temporarily misaligned disc reduces or improves its structural relation with the condyle when mandibular translation occurs during opening. This produces a joint noise (sound) described as clicking or popping. Disc displacement with reduction usually is characterized by what is termed reciprocal clicking, a reciprocal noise that is heard during the opening movement and again before the teeth occlude during the closing movement. Because disc displacement with reduction is so common, it may represent a physiologic accommodation without clinical significance.[47,48] In fact, clicking in reducing disc displacement is not pathologic because over one-third of an asymptomatic sample can have moderate to severe derangement and as many as a quarter of clicking joints show normal or only slightly displaced disc positions. Disc displacement may or may not be a painful condition. If the condition is painful, inflammation of the retrodiscal tissue, the synovial tissues, the capsule or the ligaments, or pressure and traction on the disc attachments are more likely causes of the pain.[49] Disk displacement with reduction may progress to disc displacement without reduction. This condition is characterized by sudden cessation of clicking and sudden onset of restricted mouth opening and is frequently accompanied by pain. This condition will be described in detail below.

Disc displacement without reduction is described as having a permanently displaced disk which does not improve its relation with the condyle on translation. When acute, it is characterized by sudden and marked limited mouth opening because of a jamming or fixation of the disc secondary to disc adhesion, deformation, or dystrophy. Pain is often present and especially related to the patient's attempt to open the mouth.

Disk displacement is generally treated with reassurance and education, rest, instructions to avoid loading (bite pressure such as chewing or clenching), control of contributing factors and mobility exercises within the pain free range. In the presence of pain, mild analgesics or anti-inflammatory medications are the drugs of choice. Additional management may consist of splint therapy, physical therapy, arthrocentesis, or arthroscopy to restore range of motion. Surgical treatments such as arthroscopy and open joint surgeries may be considered, but only after reasonable nonsurgical efforts have failed and when the patient's quality of life is significantly affected.[50]

Understanding and Diagnosing Myofascial Pain

Myofascial pain syndromes, as classified by the International Association for the Study of Pain Subcommittee on Taxonomy,[2] may be found in any voluntary muscle, and are characterized by trigger points (TPs), which may cause referred pain and local and referred tenderness.[51–53] When "active," TPs are painful to palpation and spontaneously refer pain and autonomic symptoms to remote structures in reproducible patterns characteristic for each muscle.[52–54] It is this referred pain that is usually the presenting complaint. When "latent," TPs are still locally tender but do not produce referred phenomena.[51] The pain quality is pressing, tightening, deep, aching, and often poorly circumscribed. It may be associated with sensations of swelling, numbness, and stiffness. Pain, although usually constant, may fluctuate in intensity and shift anatomic site. Associated symptoms may include autonomic phe-

nomena, most commonly reactive hyperemia or erythema, although photophobia and phonophobia are described.

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The primary basis for myofascial pain is the referred pain. The referral patterns often do not make neurologic sense. As an example, pain from a trigger point in the trapezius, innervated by cranial nerve 11, may refer to the forehead, innervated by cranial nerve 5. Despite the poor mechanistic understanding, clinically myofascial pain is widely accepted. It is imperative that we understand how this referral may take place.[55,56]

Mense described a hypothesis for muscle pain referral to other deep somatic tissues remote from the site of the original muscle stimulation or lesion.[57] He criticizes the convergence–projection pain referral theory, by pointing out there is little convergence in the dorsal horns associated with deep tissues. Mense's hypothesis adds 2 new components to the convergence-projection theory. First, the convergent connections from deep tissues to dorsal horn neurons are opened only after nociceptive inputs from muscle are activated. The connections opened after muscle stimulation are called silent connections. Second, the referral to muscle outside the initially activated site is due to spread of central sensitization to adjacent spinal segment.[57] The initiating stimulus requires a peripheral inflammatory stimulus. In the animal model described by Mense, the noxious stimulus was bradykinin injected into the muscle. It is unclear what triggers the muscle referral in the clinical setting where there is usually no obvious inflammation producing incident.

This Mense theory has been used by Simons to discuss a neurophysiological basis for trigger point pain.[58] Simons hypothesizes that when the tender area in the muscle is palpated there are neurotransmitters released in the dorsal horn (trigeminal nucleus) resulting in nociceptive inputs, openings that were previously silent. This causes distant neurons to produce a retrograde referred pain.[58] This model accounts for most of the clinical presentation and therapeutic options seen in myofascial pain but does not account for what initiates the peripheral tenderness that must be present to activate the silent connections.

Fields has described a means whereby the CNS may switch on nociception.[59] He describes the presence of "on" cells which when stimulated may produce activation of trigeminal nucleus nociceptors. Olesen has used Fields' model to describe a hypothesis for tension type headache.[60] This model describes the interaction of 3 systems, the vascular, supraspinal, and myogenic. The proposed hypothesis suggests that perceived headache pain is facilitated by the CNS depending on inputs from either muscle or blood vessel. In migraine, the inputs are proposed as primarily vascular whereas in tension-type headache there are primarily muscular inputs. This model helps explain why the clinical presentation and therapeutic options in migraine and tension-type headache are often similar, as well as why there is temporary relief seen with peripheral treatments such as trigger point injections.

The resultant hyperalgesia or trigger point sensitivity may represent a peripheral sensitization related to serum levels on serotonin (5H-T). Ernberg et al showed a significant correlation with serum 5H-T and allodynia associated with muscular face pain.[61] In rheumatoid temporomandibular pain, serum 5H-T concentrations correlated with pain. There was no correlation with circulating serum levels of neuropeptide Y or interleukin-1B.[62]

It is therefore proposed that patients presenting with facial pain where the etiology is not obvious may have myofascial pain. In these patients careful physical examination will allow the clinician to reproduce the pain by digitally palpating the muscles. Confirmation with trigger point injections is also helpful. It is suggested that the trigger point be injected with 1 to 2 mL of 1% procaine for best results.

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The therapy for myofascial pain requires enhancing central inhibition through pharmacology or behavioral techniques and simultaneously reducing peripheral inputs through physical therapies including exercises and trigger point specific therapy.[63] It is essential that patients are aware that the goals in therapy are to manage the pain and not to cure. It is important to stress the role patients' play in managing the perpetuating factors.[63,64]

CONCLUSION

Idiopathic facial pain is misnomer. As we better understand pain mechanisms, it is incumbent on the astute clinician and scientist to challenge, modify, and develop a more comprehensive facial pain classification, and thereby improve therapies that ultimately reduce the resultant pain and suffering.

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