Chronic Orofacial Pain and Headache as Trigeminally Mediated Disorders

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Introduction
When a patient presents with chronic orofacial pain, clinicians tend to think of “TMJ,” a rather meaningless term. Temporomandibular disorders (TMD), first suggested by Bell (1), is a better, though still problematic, term for musculoskeletal disorders of the masticatory system. A developing consensus is that this term leaves much to be desired (2), because as Nitzan et al. (3) state, “…TMD became a commitment-free term frequently used by clinicians and researchers to avoid well-defined differential diagnoses.”

The nosologic problems in orofacial pain can be traced back to “the lateral hand-off of responsibility from otolaryngologists to dentists” (4), dating to Costen’s 1934 paper. Costen erroneously concluded that the facial pain problems he saw were caused by dental and orthopedic misalignment of the cranium and jaw structures (5). Consequently, diagnosis and treatment of facial pain became the dentist’s responsibility. This transfer of management “essentially segregated facial pain from headache, and in effect from mainstream medicine” (6), which has led to recent misconceptions, such as labeling migraine a symptom of TMD (7).

As a result of Costen’s initial emphasis on morphology with these disorders, the diagnosis focused on dental occlusion. Ensuing treatment consequently focused on altering the occlusion. These misconceptions based on anatomy led to the term “occlusal disease” as the main etiology. In our practice, rather than treating occlusion, the static arrangement, we diagnose and treat applying the concept that it is more important to consider the action—what one does, occluding—rather than the static arrangement, occlusion. As Gremillion (8) stated, “it should be recognized that how the teeth relate to one another in the static sense is important. However, what people do with their teeth and their individual resistance and/or adaptive potential may be much more important with regard to the development and maintenance of TMD.”

The Conceptual Framework
To reach our goal of providing diagnostically driven therapy, we have replaced the terms “TMJ” and “TMD” with specific diagnoses based on signs and symptoms. Benoliel and Sharav (9) describe 3 types of chronic orofacial pain (COFP): musculoskeletal, neurovascular, and neuropathic.

Although TMD research diagnostic criteria (10) are well suited for case definition in research, they are not suited for patient care (9,11). As Scrivani et al. (12) remind us, “the general perception that all symptoms in the head, face, and jaw region without an identifiable cause constitute a ‘TMJ’ problem is clearly unfounded.” This conclusion stems from the fact that except for trauma, the causes of orofacial musculoskeletal pain remain largely unknown and are speculative (4). This is evidenced by the fact that there is no discernible pathology in the case of most muscle pain (13), nor is there consistent muscle hyperactivity as demonstrated by surface electromyography (sEMG) (14). Furthermore, when there are discernible changes on imaging, the data do not correlate well with the pain complaint (15).

For a review of the history of etiologic theories, see Clark (16) and Greene (17).

Neurobiology
Deciphering a patient’s upper quarter pain complaint can be difficult, in part because of the complexity of the trigeminal neuroanatomy and its relationship to the central autonomic network (18-20). Sessle (6) states:

The trigeminal system provides most of the craniofacial sensory innervation and is associated with specific physiological qualities and pain conditions. For example, pain syndromes such as trigeminal neuralgia and migraine are specific to the area, and trigeminal nerve injury responses differ from those in spinal nerves. Furthermore, the trigeminal nerve innervates anatomically related but functionally
diverse organs such as the meninges, the craniofacial vasculature, the eyes, the ears, the teeth, oral soft tissues, muscles, and temporomandibular joint. In the brainstem, the trigeminal sensory nucleus overlaps with upper cervical dermatomes. Taken together, these features account for the complex and extensive pain referral pattern that often makes clinical diagnosis so difficult.

Functional brain imaging studies show changes in cortical structures that support the concept that chronic musculoskeletal orofacial pain is similar to other chronic pain disorders and may be related to abnormal pain processing in the trigeminal system (21,22). Because the evidence points to the trigeminovascular system as the final common pathway (23,24), these disorders should be recognized as maladaptive behavior of the trigeminovascular system. Recognizing a compromised trigeminovascular system as the key component in the disease process results in a more encompassing and more accurate model than the opposing model of structural imbalance or physical impingement. Consequently, limiting the term TMD to only temporomandibular disorders logically morphs into the more comprehensive and descriptive term “trigeminally mediated disorders.”

Although only now reaching a more public forum, this terminology in reference to facial pain and headache has previously appeared in the literature. Epidemiological evidence links COFP to headache (25,26). Thalakoti et al. (27) show a pathway for the relationship of first-division headache and third-division pain in the trigeminal system through neuron-glia signaling. In his commentary on Thalakoti et al., Cady points out how this connection between the trigeminal divisions can help explain “the co-existence and interrelationship of various trigeminally mediated pain disorders” (28).

The literature shows the lack of a clear dose/response gradient of bruxism/parafunction and tooth wear to COFP and headache. A simple cause-and-effect relationship cannot be established. The concept can be understood, however, as a non-linear relationship. Rompre et al. (33) have shown that while patients who have pain do tend to parafunction, many patients who are pain-free can actually parafunction at higher levels (34). We speculate that the trigeminal signal generated during sleep is much like the bright light, sounds, and odors that can trigger a headache in a migraineur but have no effect on headache-free patients. Therefore, those who brux the most and have the most wear on their teeth are often pain-free.

Those who parafunction and remain pain-free have properly functioning pain control systems. In these cases, the trigeminal signal from parafunction is interpreted as normal. Patients with trigeminally mediated disorders may have dysfunctional modulating systems that cause a normal trigeminal signal to be interpreted as pain (29). Studies assessing risk factors in COFP and headache have begun to look at genetic susceptibility as a significant cofactor that likely leads to abnormal pain processing (35,36).

Sleep and Headache

There is a strong association between headache and sleep (37). Several studies have established that cerebral blood flow (CBF) increases during masticatory muscle contraction, resulting in dental contact and subsequent trigeminal activation (38-40). Migraines are associated
with similar increases in CBF. Nearly 50% of migraines begin during sleep (37). Several studies (41,42) have demonstrated that there is no clear link between headache and obstructive sleep apnea parameters. However, an association between headache and bruxism was found during a large trial using polysomnogram (43).

**Parafuction Control**

Dental splints have long been used to prevent and treat headache. In 1960, Berlin et al. (44) published data demonstrating a significant reduction in headache using an anterior contact dental appliance. Lamey et al. (45) later showed reduction in migraine using a full-arch dental splint. Recently, an anterior midpoint stop appliance (AMPSA) design received FDA approval for migraine prevention (46). In addition to its common use in numerous dental offices, this protocol is integral to treatment of migraine at the Headache Center of Southern California (47).

Early publications (48-52) have demonstrated the efficacy of this protocol. Since AMPsAs eliminate posterior dental contact during parafunction, they are first-line therapies in the treatment of patients with trigeminally mediated disorders. It must be recognized that the forces that affect our patients’ central nervous system, as well as the dentistry we do for them, may very well be from the same source: parafunction.

In summary, many patients with trigeminally mediated disorders “will have altered central nervous system pain processing and deficits in their ability to recruit endogenous analgesic mechanisms” (53). The concept that these patients have a structural misalignment that must be corrected by permanently changing their teeth remains unsupported by evidence (54). The use of parafunctional control in patients with trigeminally mediated disorders as a means of reducing masticatory muscle contraction during parafunction, based on the concept of reducing nociceptive afferent signal can no longer be disputed. The evidence clearly supports the rationale that reduction in trigeminal signaling is critically important in pain relief.

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**REFERENCES**


