

Chronic orofacial pain and headache and the use of NTI-tss

Barry Glassman and **Don Malizia** look at the evidence for using NTI-tss splints in reducing trigeminal signalling for pain relief

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 (1982), 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 (Laskin, 2008) because, as Nitzan et al (2004) state: '...TMD became a commitment-free term frequently used by clinicians and researchers to avoid well-defined differential diagnoses.'

The problem of classification of orofacial pain can be traced back to the 'lateral hand-off of responsibility from otolaryngologists (ENT Physicians) to dentists' (Greene, 2006), when in 1934 Costen erroneously concluded that the facial pain problems he saw were caused by dental and orthopedic misalignment of the cranium and jaw structures.

Consequently, diagnosis and treatment of facial pain became the dentist's responsibility through the transfer of management that 'essentially segregated facial pain from headache, and in effect mainstream medicine' (Sessle, 2008). This has contributed to such misconceptions as labelling migraine a symptom of TMD (Feld, 2010).

Costen's initial emphasis on morphology with these disorders meant diagnosis focused on dental occlusion and consequently, treatment focused on altering the occlusion.

In our practice, diagnosis and treatment is based on the concept that it is more important to consider the action, occluding – rather than the static arrangement, occlusion.

As Gremillion stated in 1995: 'It should be recognised 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.

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. In 2010, Benliel and Sharav described three types of chronic orofacial pain (COFP):

- Musculoskeletal
- Neurovascular
- Neuropathic.

Aims and objectives

To review the evidence for using NTI-tss splints in reducing trigeminal signalling for pain relief.

Expected outcomes

Correctly answering the questions on page 86 will demonstrate you understand the rationale for NTI-tss splints in reducing trigeminal signalling is critically important in pain relief.

Verifiable CPD hours: 1



Figure 1: Nociceptive Trigeminal Inhibition Tension Suppression System (NTI-tss)

The conceptual framework

Although TMD research diagnostic criteria (Dworkin, LeResche, 1992) are well-suited for case definition in research, they are ill-suited for patient care (Benliel, Sharav, 2010; Steenks, dr Wijer, 2009). As Scrivani et al (2008) 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 speculative (Greene, 2006). This is

evidenced by the fact that there is no discernible pathology in the case of most muscle pain (Svensson, Graven-Nielsen, 2001), nor is there consistent muscle hyperactivity as demonstrated by surface electromyography (sEMG) (Stophler, 1999). Furthermore, when there are discernible changes on imaging, the data do not correlate well with the pain complaint (Nitzan et al, 2008).

For a review of the history of etiologic theories, see Clark (1991) and Greene (1992).

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 (Benarroch, 2008; Montagna, Cortelli, 2008; Montagna, Pierangeli, Cortelli, 2010).

Sessle (2008) 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 (de Leeuw et al, 2005a; de Leeuw et al, 2005b). Because the evidence points to the trigeminovascular system as the final common pathway (Piovesan, Kowacs, Oshinsky, 2003; Bartsch, Goadsby, 2005), these disorders should be recognised as maladaptive behaviour of the trigeminovascular system.

Recognising a compromised trigeminovascular system as the key component in the disease process results in a more encompassing and more accurate model than does the opposing model of structural imbalance or physical impingement. Therefore, 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

redefining

the boundaries in Private Dentistry

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previously appeared in the literature. Epidemiological evidence links COFP to headache (Franco et al, 2010; Gonçalves et al, 2010). Thalakoti et al (2007) show a pathway for the relationship of first-division headache and third-division pain in the trigeminal system through neuron-glia signalling.

In his comment on Thalakoti et al, Cady (2007) points out how this connection between the trigeminal divisions can help explain the 'coexistence and interrelationship of various trigeminally mediated pain disorders.'

The literature shows the lack of a clear dose/response gradient of bruxism/parafunction and tooth wear to COFP and headache (Svensson et al, 2008). A simple cause-and-effect relationship cannot be established (Hill, 1965). The concept can be understood, however, as a non-linear relationship.

Rompre et al (2007) have shown that while patients who have pain do tend to parafunction, many patients who are pain-free can actually parafunction at higher levels. We speculate that the trigeminal signal generated during sleep is much like the bright light, sounds and odours that can trigger a headache in a migraineur but have no effect on headache-free patients. Therefore, those who grind the most and have the most wear on their teeth are often pain-free (Janal et al, 2007).

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 (Svensson et al, 2008).

Studies assessing risk factors in COFP and headache have begun to look at genetic susceptibility as a significant co-factor that likely leads to abnormal pain processing (Stohler, 2004; Slade et al, 2008).

Sleep and headache

There is a strong association between headache and sleep (Fox, Davies, 1998). Several studies have established that cerebral blood flow (CBF) increases during masticatory muscle contraction, resulting in dental contact and subsequent trigeminal activation (Hasegawa et al, 2007; Hasegawa et al, 2011; Hasegawa et al, 2009).

Migraines are associated with similar increases in CBF: nearly 50% of migraines begin during sleep (Fox, Davis, 1998).

Several studies (Goksan et al, 2009; Goder et al, 2003) 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 (Lucchesi et al, 2010).

Parafunctional control – NTI-tss

Dental splints have long been used to prevent and treat

headache. In 1960, Berlin and Dessner published data demonstrating a significant reduction in headache using an anterior contact dental appliance. Lamey et al (1996) later showed reduction in migraine using a full-arch dental splint.

Recently the NTI-tss (Nociceptive Trigeminal Inhibition Tension Suppression System) splint received FDA approval for migraine prevention. In addition to its common use in numerous dental surgeries worldwide, this protocol is integral to treatment of migraine at the Headache Center of Southern California (2011).

Summary

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' (Cairns et al, 2010). The concept that these patients have a structural misalignment must be corrected by permanently changing their teeth remains unsupported by the evidence (Menfredini et al, 2011).

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 for NTI-tss splints in reducing trigeminal signalling is critically important in pain relief. **PD**

References

For the full list of references that accompany this article, please email the editor at siobhan.lewney@fmc.co.uk.



Questions on page 86

Comments to pd@fmc.co.uk

Barry Glassman, DMD, director of the Allentown Pain Centre, Allentown, Pennsylvania, limits his practice to the treatment of chronic pain, temporomandibular joint dysfunction, headache, and sleep-disordered breathing. Dr Glassman will be presenting a lecture in London on 24 October to mark the start of the second Bruxism Awareness Week sponsored by S4S.

Dr Don Malizia, DDS, treats patients with trigeminally mediated disorders and sleep-disordered breathing with Dr Barry Glassman at the Allentown Pain Centre in Allentown, Pennsylvania, USA.

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Q1

Although a misconception, what is often labelled as a symptom of TMD?

- a) Heart attack
- b) Migraine
- c) Weight gain
- d) Bruxism

Q2

Which of the following is described as a type of chronic orofacial pain (COFP)?

- a) Musculoskeletal
- b) Neurovascular
- c) Neuropathic
- d) All of the above

Q3

What type of evidence links chronic orofacial pain to headache?

- a) Epidemiological
- b) Pathological
- c) Observational
- d) Pathophysiological

Q4

How many migraines begin during sleep?

- a) 15%
- b) 30%
- c) 50%
- d) 85%

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Q1

Why are implants distributed evenly in the edentulous space?

- a) For predictable healing
- b) To preserve tissue support
- c) To spread the load
- d) For aesthetic reasons

Q2

What is the outcome of condensing bone at placement with hand instruments?

- a) Maxillary bone quality is improved
- b) Maxillary bone quality is reduced
- c) Maxillary bone quality is unaffected
- d) None of the above

Q3

In the mandible, dense bone is typically available in the intraforaminal area where bicortical fixation may be achieved. How many implants may be placed for an edentulous jaw?

- a) Three
- b) Four
- c) Five
- d) Six

Q4

What benefit is there to immediately loaded implants?

- a) Reduction in cost
- b) Improved patient acceptance
- c) Reduction in treatment time
- d) All of the above
- e) None of the above