For reprint orders, please contact reprints@future-drugs.com

Craniomandibular muscles, intraoral orthoses and migraine

Elliot Shevel

Intraoral splints are effective in migraine prevention. In this review, changes in the quality of life of migraineurs treated with a palatal nonoccluding splint were measured. Using the Migraine Specific Quality of Life Instrument (Version 2.1), it was found that the palatal nonoccluding splint significantly improved the quality of life of migraineurs. The role of the craniomandibular muscles in the pathophysiology of migraine is also discussed.

Expert Rev. Neurotherapeutics 5(3), 371–377 (2005)

CONTENTS

The Headache Clinic,
Suite 256, P Bag X2600,
Houghton, 2014, South Africa
Tel.: +27 114 840 933
Fax: +27 114 840 982
drshevel@headclin.com

KEYWORDS:
craniomandibular, migraine, muscle dysfunction, orthosis

Migraine is a common disorder with a lifetime prevalence of 16% worldwide, and a last-year prevalence of 10% [1,2]. It may significantly diminish quality of life, even between attacks, and impairs quality of life more than diabetes, hypertension and osteoarthritis [3–5]. Although the pathogenesis of migraine headache remains poorly understood, current theories suggest a primary, possibly genetically determined, CNS dysfunction to be involved. There is activation of the trigeminovascular system [6,7], which is comprised of the meningeal vessels, trigeminal nerve and trigeminal nucleus, in particular the trigeminal subnucleus caudalis [8].

Tenderness and dysfunction of the craniomandibular muscles is a common finding in migraine [9–15]. Intraoral interocclusal orthoses, used in the treatment of craniomandibular muscle dysfunction [16–21], are also effective in preventing migraine [22–24]. Their therapeutic muscle-relaxing effect is attributed to the fact that they encourage the mandible to assume the physiologic rest position, thereby altering habitual neuromuscular patterns within the masticatory muscles [21]. When a nonoccluding palatal orthosis is worn, there is increased resting length and relaxation of the craniomandibular muscles [25,26]. This study determined the effect of wearing a nonoccluding palatal orthosis on the quality of life of migraineurs.

Materials & method

Patient selection

In total, 152 patients, 117 female and 35 male, were admitted to the study. The inclusion criteria were:

- Age of onset of migraine before 50 years
- Subjects with all or most of their own teeth, and who did not wear a removable dental prosthesis
- History of migraine of 1 year or more, with at least one attack per week in the previous 3 months
- Headache free between attacks
- A diagnosis of migraine without aura (i.e., group 1.1 in the guidelines laid down by the Headache Classification Committee of the International Headache Society)

To make the diagnosis of migraine without aura, the following criteria must be met [27]:

A. At least five attacks fulfilling criteria B, C and D

B. Headache attacks lasting 4–72 h (untreated or unsuccessfully treated)

C. Headache has at least two of the following characteristics: unilateral location, pulsating quality, moderate or severe intensity (inhibits or prohibits daily activities), aggravated by walking up stairs or similar routine physical activity

D. During the headache at least one of the following: nausea and/or vomiting, photophobia and phonophobia

 10.1586/14737175.5.3.371 ISSN 1473-7175

Factors that could influence the frequency or intensity of migraine, such as pregnancy, the use of prophylactic migraine medication or ergot derivatives, a history of drug or alcohol abuse, or serious illness were exclusion criteria. All participants were fully informed of the nature of the project and their prior consent was obtained.

Palatal speech-adjusted appliance

The posture-modifying appliance (PMA) was fabricated using the maxillary cast of the subject. It consisted of a 3 mm thick acrylic resin reinforced with a chrome cobalt strip (FIGURE 1). The appliance covered the hard palate, with the exception of the anterior part where the tip of the tongue normally touches during speech.

The PMA was adjusted for fit and overall comfort. Patients were told that the PMA should not interfere with the free movement of the tongue during speech. They were asked to speak with the PMA *in situ* using the words listed in BOX 1, which are phonetically balanced and designed to test the whole range of English sounds in various combinations [28]. The PMA was then removed and the part that the tongue had touched during speech indicated by the patient. The offending acrylic was ground away and the process repeated, until the patient was no longer aware of any interference with tongue movement. The final shape and thickness of the PMA was, in most patients, very different to the original (FIGURE 2). Subjects were instructed to wear the PMA day and night, but to remove it during tooth brushing, eating and drinking, and when playing contact sports. Subjects were requested to return for adjustment of the PMA if they experienced discomfort or speech difficulty.

Migraine specific quality of life measurement

The Migraine Specific Quality of Life Questionnaire (MSQ) Version 2.1 was used to assess the efficacy of the PMA. The MSQ is a 14-item, self-administered questionnaire, which measures three dimensions of headache-related quality of life that are affected by migraine [29]:

Figure 1. The posture-modifying appliance before adjusting for speech.

- Role function restriction, which measures the percentage of time that the patient can perform normal daily activities
- Role function prevention, which measures the percentage productivity while working
- Emotional function, which measures the percentage of emotional and relationship disability

Patients completed the MSQ before the start of treatment and again 12 months later. Participants were instructed to continue using palliative medication whenever necessary.

Results

As there was no significant statistical difference between the results for males and females, they were combined, and the average pretreatment and post-treatment scores for each parameter were calculated. Analysis of the data using the Student's t-test showed statistically significant improvement in all three parameters. Role function restriction improved from 54.6 to 91% ($p < 0.0001$), role function prevention improved from 45.4 to 84.8% ($p < 0.0001$) and emotional function improved from 45.4 to 91.2% ($p < 0.0001$).

Discussion

Migraine is considered to be a neurovascular syndrome, with abnormal neuronal excitability in the cerebral cortex, peripheral sensitization of the trigeminovascular system and pain due to dilation of intracranial blood vessels [30–32]. The triptans were developed as cranial vasoconstrictors to mimic the desirable effects of serotonin [33,34], while avoiding its side effects [35]. An important hindrance to the more widespread use of the triptans is the unsubstantiated perception that they have harmful vasoconstrictor effects [32].

Nociceptive input to the CNS is increased due to sensitization of peripheral sensory afferents, and the resultant barrage of nociceptive impulses results in sensitization of second- and third-order neurons in the CNS. In this way, sensitization may play a role in the initiation and maintenance of migraine [36]. Consequently, current research has focussed upon prejunctional and presynaptic targets on nociceptive trigeminovascular neurons in an attempt to develop drugs that inhibit trigeminal nociceptive traffic and central sensitization without vasoconstrictor effects [32,37].

Central sensitization is induced by nociceptive afferent input from the intracranial dura mater travelling along the trigeminovascular pain pathway [38]. It results in [39–41]:

- A reduction of the threshold to cell depolarization
- Cellular activity that continues after cessation of the peripheral nociceptive input
- A spread of cellular activity to neighbouring cells

Noxious stimulation of muscle afferents also increases the excitability of spinal cord neurons [42]. Persistent stimulation leads to cellular and molecular changes, which result in neuronal hyperexcitability, to the extent that pain is elicited by low-threshold, normally non-noxious, stimuli [43–49]. After an increase in central excitability produced by the activation of peripheral chemoreceptors, cells in the trigeminal nucleus caudalis that are normally nociceptive-specific begin to respond to low-threshold, primary afferent non-nociceptive mechanoreceptors [50]. Repeated stimulation of a dorsal root produces, in some neurons, a prolonged heterosynaptic facilitation with an augmentation of the response to the conditioning root (homosynaptic potentiation) as well as to adjacent test roots (heterosynaptic potentiation) [51].

Restoring a patient's ability to function normally is now recognized as the primary treatment goal, rather than merely relieving pain [52]. The results of this study show that relaxation of the craniomandibular muscles by means of a PMA improves the quality of life of migraineurs. By reducing sensory input from the craniomandibular muscles, central sensitization is reduced. The probable mechanism is that intraoral splints may have therapeutic effects apart from those commonly attributed to the occlusal component [53]. This may be attributed to the fact that an intraoral appliance may encourage the mandible to assume the physiologic rest position, thereby altering habitual neuromuscular patterns within the masticatory muscles [54]. Further research has shown that when a nonoccluding palatal appliance is worn there is an increase in the interocclusal distance and, consequently, in the resting length of the masticatory muscles [55,56].

A limitation of this study is the lack of a placebo control group. There is, unfortunately, no remedy for this when testing a physical intervention such as an intraoral appliance, given the sensitivity of the intraoral structures. The possible placebo effect of the PMA cannot therefore be measured, and its importance must remain the subject of speculation. According to Occam's Razor, in science the simplest theory that fits the facts of a problem is the one that should be selected. This is interpreted to mean that the simplest of two competing theories is preferable. If Occam's Razor is applied, then the most likely conclusion is that the PMA does have a beneficial nonplacebo effect. The possibility of natural regression of the migraine in this group of patients is minimal, given that all the subjects had been suffering for a long time frame without improvement until the PMA was fitted.

Further corroborating evidence that the craniomandibular muscles play a role in the cascade of events in migraine pathogenesis is described below.

Anatomy

- The middle meningeal artery, dura of the middle and anterior cranial fossae, and craniomandibular muscles, all receive sensory afferents from the mandibular division of the trigeminal nerve. They all send sensory afferent input to the subnucleus caudalis, possibly enhancing central sensitization. The middle meningeal artery and dura of the middle and anterior cranial fossae via its recurrent meningeal branch, and the muscles via their individual branches [57,58].
- Volumetric analysis of the masseter and medial pterygoid muscles showed that the volume of masticatory muscles in migraineurs is nearly 70% greater than in nonmigraineurs ($p < 0.0001$) [59].

Neural pathways

- Sensory afferents from the craniomandibular muscles project to the trigeminal sensory nuclei, and in particular to the subnucleus caudalis. Subnucleus caudalis neurons, including low-threshold mechanoreceptive, wide-dynamic range and nociceptive-specific neurons, are excited by the stimulation of craniomandibular muscle sensory afferents [42,60–66].
- The subnucleus caudalis also acts as a critical interneuronal relay site in craniofacial nociceptive reflex activity involving the craniofacial muscles [67–70].

Clinical findings

The following clinical findings have been determined:

- Pericranial muscle pain and tenderness are prominent features in migraine [71–73]
- There is increased pericranial muscle electromyographic activity in migraine [74,75]
- Physical therapy can precipitate migraine attacks [76]

Treatment modalities

Treatment modalities that reduce craniomandibular muscle tension are effective in the treatment of migraine and include:

- Intraoral splints which reduce migraine intensity and frequency [77–82].
- Biofeedback to induce muscle relaxation is widely used in migraine prophylaxis. The positive treatment response to biofeedback/relaxation in migraine headache is not related to presence of changes in blood flow velocity [83].
- Intramuscular trigger point injections are effective in the treatment of acute migraine pain [84–86].

Box 1. Phonetically balanced word list designed to test the whole range of English sounds in various combinations [28].

- Iceberg
- Armchair
- Sunset
- Mousetrap
- Playground
- Inkwell
- Whitewash
- Pancake
- Cowboy
- Woodwork

Figure 2. Example of the posture-modifying appliance after adjusting for speech.

- Resection of the corrugator supercillii muscles in patients who respond positively to botulinum toxin A injection results in prolonged and effective migraine prophylaxis [87–89].

Drug therapy

Preliminary studies indicate that drugs such as botulinum toxin A, baclofen and tizanidine, which reduce skeletal muscle spasm and tone, may be useful in migraine prophylaxis [90].

Sumatriptan was developed as a cerebral vasoconstrictor, but it has also been shown to act on skeletal muscle [91–93]. It cannot be excluded, therefore, that the triptans may be effective in migraine due to altered muscle metabolism.

Expert opinion

Current theories suggest that a primary, probably genetically determined, CNS dysfunction is involved in the initiation of the migraine headache, with activation of the trigeminovascular system and sensitization of neurons in the CNS [6]. Clinical findings suggest a relationship between migraine headaches on the one hand and dysfunction of the craniomandibular muscles on the other. In this study, the quality of life of migraineurs was significantly enhanced by the use of an intraoral palatal nonoccluding appliance. This and other evidence, including anatomical evidence, the projection of sensory afferents from the craniomandibular muscles to the trigeminal subnucleus caudalis, clinical findings, treatment modalities designed to reduce muscle tension which also successfully treat migraine, and drug trials, provide a compelling argument that central sensitization in migraineurs is enhanced by sensory input originating from the craniomandibular muscles. Therefore, the best current treatment regimen must include assessment and treatment of the pericranial muscles.

Five-year view

It is unlikely that this treatment regimen will gain much favor. The reason being that medicine is divided into different disciplines, each with its own sphere of interest. While the general public may believe that these disciplines share information at the highest level, in reality they rarely communicate with each other. The excellent results achieved with the use of intraoral splints in migraineurs have been on record for many years. In spite of this, intraoral splints are rarely mentioned in the headache literature – there is not a single article on the subject in *Headache* or *Cephalalgia* in at least the last 3 years. Unfortunately, despite the excellent clinical results, splint therapy for migraine is still regarded with scepticism. In the words of Max Planck (Nobel Prize Physicist, 1918), "A new scientific truth does not triumph by convincing its opponents and making them see the light, but rather because its opponents eventually die, and a new generation grows up that is familiar with it". It is improbable, therefore, that, despite the proven efficacy of intraoral splints, their use will be widely adopted within the next 5 years. In the next 50 years... perhaps?

Acknowledgements

The author would like to express sincere thanks to Daniel Shevel for his invaluable input in the writing of this review.

Key issues

- Migraine is a common disorder.
- It is characterized by moderate-to-severe pain, with associated symptoms such as nausea, vomiting, photophobia and phonophobia.
- Migraine is associated with changes in the trigeminovascular system.
- Tenderness and dysfunction of the craniomandibular muscles is a common finding in migraine.
- Intraoral orthoses are used to relax the craniomandibular muscles and restore them to normal function.
- This review studies the effect on migraineurs of wearing a nonoccluding palatal orthosis.
- Placebo-controlled studies are not feasible when intraoral orthoses are used.
- The effect was therefore measured by comparing pretreatment with post-treatment quality of life.
- Statistical analysis of the results showed a significant improvement in quality of life when the orthosis was worn.

References

Papers of special note have been highlighted as:
• of interest
•• of considerable interest

1 Rasmussen BK, Jensen R, Schroll M, Olesen J. Epidemiology of headache in a general population – a prevalence study. *J. Clin. Epidemiol.* 44, 1147–1157 (1991).

2 Schwartz BS, Stewart WF, Lipton RB. Lost workdays and decreased work effectiveness associated with headache in the workplace. *J. Occup. Environ. Med.* 39, 320–327 (1997).

3 Gerbaud L, Navez ML, Couratier P *et al.* Validation of the combined SF36/MSQOL test of evaluation of quality of life in migraine patients in France. *Rev. Neurol.* 158, 719–727 (2002).

4 Michel P, Dartigues JF, Lindoulsi A, Henry P. Loss of productivity and quality of life in migraine sufferers among French workers: results from the GAZEL cohort. *Headache* 37, 71–78 (1997).

5 Osterhaus JT, Townsend RJ, Gandek B, Ware JE. Measuring the functional status and well-being of patients with migraine headache. *Headache* 34, 337–343 (1994).

6 Russell MB. Genetic epidemiology of migraine and cluster headache. *Cephalalgia* 17, 683–701 (1997).

7 Edvinsson L. Aspects on the pathophysiology of migraine and cluster headache. *Pharmacol. Toxicol.* 89, 65–73 (2001).

8 Goadsby PJ, Lipton RB, Ferrari MD. Migraine – current understanding and treatment. *N. Engl. Med. J.* 346, 257–270 (2002).

9 Lipchik GL, Holroyd KA, Talbot F, Greer M. Pericranial muscle tenderness and exteroceptive suppression of temporalis muscle activity: a blind study of chronic tension-type headache. *Headache* 37, 368–376 (1997).
•• Raises the possibility that pericranial muscle tenderness is present early in migraine without aura, and thus may contribute to the etiology.

10 Steele JG, Lamey PJ, Sharkey SW, Smith GMR. Occlusal abnormalities, pericranial muscle and joint tenderness and tooth wear in a group of migraine patients. *J. Oral Rehabil.* 18, 453–458 (1991).

11 Lous I, Olesen J. Evaluation of pericranial tenderness and oral function in patients with common migraine, muscle contraction headaches, and combination headache. *Pain* 12, 385–393 (1982).

12 Tfelt-Hansen P, Lous I, Olesen J. Prevalence and significance of muscle tenderness during common migraine attacks. *Headache* 21, 49–54 (1981).

13 Olesen J. Some clinical features of the acute migraine attack: an analysis of 750 patients. *Headache* 18, 268–271 (1978).

14 Cohen MJ. Psychophysiological studies of headache: is there similarity between migraine and muscle contraction headache? *Headache* 18, 189–196 (1978).

15 Bakal DA, Kaganov JA. Muscle contraction and migraine headache: psychophysiologic comparison. *Headache* 17, 208–215 (1977).

16 Block SL, Apfel M, Laskin DM. The use of a resilient rubber bite appliance in the treatment of MPD syndrome. *J. Dent. Res.* 57, A71 (1978).

17 Campbell J. Extension of the temporomandibular joint space by methods derived from general orthopaedic procedures. *J. Prosthet. Dent.* 7, 386–399 (1957).

18 Greene CS, Laskin DM. Splint therapy for the myofascial pain-dysfunction syndrome: a comparative study. *J. Am. Dent. Ass.* 84, 624–628 (1972).

19 Lamey PJ, Steele JG, Aitchison T. Migraine: the effect of acrylic appliance design on clinical response. *Br. Dent. J.* 180, 137–140 (1996).

20 Matthews E. Treatment for the teeth-grinding habit. *Dent. Record* 62, 154–155 (1942).

21 Posselt U. Treatment of bruxism by bite guards and bite plates. *J. Canad. Dent. Assoc.* 29, 773–778 (1963).

22 Quayle AA, Gray RJM, Metcalfe RJ, Guthrie E, Wastell D. Soft occlusal splint therapy in the treatment of migraine and other headaches. *J. Dent.* 18, 123–129 (1990).

23 Lapeer GL. Reduction of the painful sequelae of migraine headache by use of the occlusal diagnostic appliance: an hypothesis. *J. Craniomandib. Pract.* 6, 82–86 (1988).
•• As early as 1988, Lapeer suggested that "further research into occlusal splint therapy as an adjunct to relieving or aborting the painful sequelae of migraine headaches is necessary".

24 Lamey PJ, Barclay SC. Clinical effectiveness of occlusal splint therapy in patients with classical migraine. *Scot. Dent. J.* 32, 11–12 (1987).

25 Young P. A cephalometric study of the effect of acrylic test palatal piece thickness on the physiologic rest position. *J. Philippine Dent. Ass.* 19, 5–15 (1966).

26 Minagi S, Shimamura M, Sato T, Natsuaki N, Ohta M. Effect of a thick palatal appliance on muscular symptoms in craniomandibular disorders: a preliminary study. *J. Craniomandib. Pract.* 19, 42–47 (2001).
• First suggestion in the literature that a palatal appliance could bring about improvement of myofascial pain dysfunction syndrome.

27 Olesen J. Classification and diagnostic criteria for headache disorders, cranial neuralgias, and facial pain. First edition. *Cephalalgia* 8(Suppl. 7), 19–33 (1988).

28 Berger KW. *Speech Audiometry Materials*. Herald, OH, USA, 46–50 (1977).

29 Patrick DL, Hurst BC, Hughes J. Further development and testing of the migraine-specific quality of life (MSQOL) measure. *Headache* 40(7), 550–560 (2000).
•• Shows that cumulative evidence for the migraine-specific quality of life instrument meets established criteria for validity, consistency and reproducibility. It confirmed that quality of life testing is a valid tool for measuring treatment efficacy in migraine.

30 Silberstein SD. Migraine pathophysiology and its clinical implications. *Cephalalgia* 24(Suppl. 2), 2–7 (2004).

31 Wolff HG. *Headache and Other Head Pain. First Edition.* Oxford University Press, NY, USA (1948).

32 Goadsby PJ. Prejunctional and presynaptic trigeminovascular targets: what preclinical evidence is there. *Headache Currents* 1, 1–6 (2004).

33 Kimball RW, Friedman AP, Vallejo E. Effect of serotonin in migraine patients. *Neurology* 10, 107–111 (1960).

34 Lance JW, Anthony M, Hinterberger H. The control of cranial arteries by humoural mechanisms and its relation to the migraine syndrome. *Headache* 7, 93–102 (1967).

35 Humphrey PPA, Feniuk W, Perren MJ, Beresford IJM, Skingle M, Whalley ET. Serotonin and migraine. *Ann. NY Acad. Sci.* 600, 587–598 (1990).

36 Bendtsen L. Sensitization: its role in primary headache. *Curr. Opin. Investig. Drugs* 3, 449–453 (2002).
•• Elucidated the concept that nociceptive input to the CNS may be increased due to activation or sensitization of peripheral sensory afferents.

37 Ramadan NM, Buchanan MS, Stare H, Pearlman H. Peripheral and central targets for acute migraine therapy: early clinical trial results. *Headache Cur.* 1, 7–12 (2004).

38 Malick A, Burstein R. Peripheral and central sensitization during migraine.

Funct. Neurol. 15(Suppl. 3), 28–35 (2000).

39 Woolf CJ, Salter MW. Neuronal plasticity: increasing the gain in pain. *Science* 288, 1765–1768 (2000).

40 Coderre TJ, Katz J. Peripheral and central hyperexcitability: differential signs and symptoms in persistent pain. *Behav. Brain Sci.* 20, 404–419 (1997).

41 Dubner R. Neural basis of persistent pain: sensory specialization, sensory modulation, and neuronal plasticity. In: *Progress in Pain Research and Management.* Jensen TS, Turner JA, Weisenfeld-Hallin Z (Eds). IASP Press, WA, USA, 77–91 (1997).

42 Hu JW, Sessle BJ, Raboisson P, Dallel R, Woda A. Stimulation of craniofacial muscle afferents induces prolonged facilitatory effects in trigeminal nociceptive brain-stem neurones. *Pain* 48, 53–60 (1992).

43 Jensen TS. Mechanisms of neuropathic pain. In: *Pain 1996, an Updated Review.* Campbell JN (Ed.). IASP Press, WA, USA, 77–86 (1996).

44 Jensen TS. Recent advances in pain research: implications for chronic headache. *Cephalalgia* 21, 765–769 (2001).

45 Gracely RH, Lynch SA, Bennett GJ. Painful neuropathy: altered central processing, maintained dynamically by peripheral input. *Pain* 51, 175–194 (1992).

46 Gottrup H, Nielsen J, Arendt-Nielsen L, Jensen TS. The relationship between sensory thresholds and mechanical hyperalgesia in nerve injury. *Pain* 75, 321–329 (1998).

47 Gottrup H, Andersen J, Arendt-Nielsen L, Jensen TS. Psychophysical examination of patients following operation for cancer mammae. *Pain* 87, 275–284 (2000).

48 Koltzenburg M. Painful neuropathies. *Curr. Opin. Neurol.* 67, 307–316 (1998).

49 Yamamura H, Malick A, Chamberlin NL, Burstein R. Cardiovascular and neuronal responses to head stimulation reflect central sensitization and cutaneous allodynia in a rat model of migraine. *J. Neurophysiol.* 81, 479–493 (1999).

50 Woolf CJ, Shortland P, Sivilotti LG. Sensitization of high mechanothreshold superficial dorsal horn and flexor motor neurones following chemosensitive primary afferent activation. *Pain* 58, 141–155 (1994).

51 Woolf CJ, Thompson SW, King AE. Prolonged primary afferent induced alterations in dorsal horn neurones, an intracellular analysis *in vivo* and *in vitro. J. Physiol.* 83, 255–266 (1988–89).

52 Wenzel R, Dortch M, Cady R, Lofland JH, Diamond S. Migraine headache misconceptions: barriers to effective care. *Pharmacotherapy* 24, 638–648 (2004).

53 Greene CS, Laskin DM. Splint therapy for the myofascial pain-dysfunction syndrome: a comparative study. *J. Am. Dent. Ass.* 84, 624–628 (1972).

54 Posselt U. Treatment of bruxism by bite guards and bite plates. *J. Canad. Dent. Assoc.* 29, 773–778 (1963).

55 Young P. A cephalometric study of the effect of acrylic test palatal piece thickness on the physiologic rest position. *J. Philippine Dent. Ass.* 19, 5–15 (1966).

56 Minagi S, Shimamura M, Sato T, Natsuaki N, Ohta M. Effect of a thick palatal appliance on muscular symptoms in craniomandibular disorders: a preliminary study. *J. Craniomandib. Pract.* 19, 42–47 (2001).

57 Shankland WE. The trigeminal nerve. Part IV: the mandibular division. *J. Craniomandib. Pract.* 19, 153–161 (2001).

58 Asfat R. A review of the neurovascular supply of the mandible. *S. Afr. Dent. J.* 57, 414–416 (2002).

59 Lamey PJ, Burnett CA, Fartash L, Clifford TJ, McGovern JM. Migraine and masticatory muscle volume, bite force, and craniofacial morphology. *Headache* 41, 49–56 (2001).

•• The volume of masticatory muscles in migraineurs is nearly 70% greater than in nonmigraineurs ($p < 0.0001$).

60 Yamakami Y. Characteristics of responses in the trigeminal subnucleus caudalis and adjacent reticular formation evoked by the stimulation of the masseter muscle. *Showa Shigakkai Zasshi* 9, 130–135 (1989).

61 Arvidsson J, Raappana P. An HRP study of the central projections from primary sensory neurons innervating the rat masseter muscle. *Brain Res.* 20, 111–118 (1989).

62 Shigenaga Y, Sera M, Nishimori T *et al.* The central projection of masticatory afferent fibers to the trigeminal sensory nuclear complex and upper cervical spinal cord. *J. Comp. Neurol.* 22, 489–507 (1988).

63 Luo P, Wong R, Dessem D. Projection of jaw-muscle spindle afferents to the caudal brainstem in rats demonstrated using intracellular biotinamide. *J. Comp. Neurol.* 17, 63–78 (1995).

64 Luo P, Dessem D. Inputs from identified jaw-muscle spindle afferents to trigeminothalamic neurons in the rat: a double-labeling study using retrograde HRP and intracellular biotinamide. *J. Comp. Neurol.* 27, 50–66 (1995).

65 Sessle BJ, Hu JW, Amano N, Zhong G. Convergence of cutaneous, tooth pulp, visceral, neck and muscle afferents onto nociceptive and non-nociceptive neurones in trigeminal subnucleus caudalis (medullary dorsal horn) and its implications for referred pain. *Pain* 27, 219–235 (1986).

66 Amano N, Hu JW, Sessle BJ. Responses of neurons in feline trigeminal subnucleus caudalis (medullary dorsal horn) to cutaneous, intraoral, and muscle afferent stimuli. *J. Neurophysiol.* 55, 227–243 (1986).

67 Matthews B. Peripheral and central aspects of trigeminal nociceptive systems. *Philos. Trans. R. Soc. Lond. Biol. Sci.* 19, 313–324 (1985).

68 Tsai C. The caudal subnucleus caudalis (medullary dorsal horn) acts as an interneuronal relay site in craniofacial nociceptive reflex activity. *Brain Res.* 826, 293–297 (1999).

69 Tsai CM, Chiang CY, Yu XM, Sessle BJ. Involvement of trigeminal subnucleus caudalis (medullary dorsal horn) in craniofacial nociceptive reflex activity. *Pain* 81, 115–128 (1999).

70 Cairns BE, Sessle BJ, Hu JW. Temporomandibular-evoked jaw muscle reflex: role of brain stem NMDA and non-NMDA receptors. *Neuroreport* 12, 1875–1878 (2001).

71 Steele JG, Lamey PJ, Sharkey SW, Smith G. Occlusal abnormalities, pericranial muscle and joint tenderness and tooth wear in a group of migraine patients. *J. Oral Rehabil.* 18, 453–458 (1991).

72 Jensen K, Tuxen C, Olesen J. Pericranial muscle tenderness and pressure-pain threshold in the temporal region during common migraine. *Pain* 35, 65–70 (1988).

73 Anttila P, Metsahonkala L, Mikkelsson M *et al.* Muscle tenderness in pericranial and neck-shoulder region in children with headache. A controlled study. *Cephalalgia* 22, 340–344 (2002).

74 Clifford T, Lauritzen M, Bakke M, Olesen J, Moller E. Electromyography of pericranial muscles during treatment of spontaneous common migraine attacks. *Pain* 14, 137–147 (1982).

75 Burnett CA, Fartash L, Murray B, Lamey PJ. Masseter and temporalis muscle EMG levels and bite force in migraineurs. *Headache* 40, 813–817 (2000).

76 Blau JN, MacGregor EA. Migraine and the neck. *Headache* 34, 88–90 (1994).

77 Lamey PJ, Barclay SC. Clinical effectiveness of occlusal splint therapy in

patients with classical migraine. *Scott. Med. J.* 32, 11–12 (1987).

78 Lamey PJ, Steele JG, Aitchison T. Migraine: the effect of acrylic appliance design on clinical response. *Br. Dent. J.* 24, 137–140 (1996).

79 Shankland WE. Nociceptive trigeminal inhibition–tension suppression system: a method of preventing migraine and tension headaches. *Compend. Contin. Educ. Dent.* 23, 105–108 (2002).

80 Shankland WE II. Migraine and tension-type headache reduction through pericranial muscular suppression: a preliminary report. *J. Craniomandib. Pract.* 19, 269–278 (2001).

81 Shankland WE. Nociceptive trigeminal inhibition–tension suppression system: a method of preventing migraine and tension headaches. *Compend. Contin. Educ. Dent.* 22, 1075–1080 (2001).

82 Quayle AA, Gray RJM, Metcalfe RJ, Guthrie E, Wastell D. Soft occlusal splint therapy in the treatment of migraine and other headaches. *J. Dent.* 18, 123–129 (1990).

83 Vasudeva S, Claggett AL, Tietjen GE, McGrady AV. Biofeedback-assisted relaxation in migraine headache: relationship to cerebral blood flow velocity in the middle cerebral artery. *Headache* 43, 245–250 (2003).

84 Tfelt-Hansen P, Lous I, Olesen J. Prevalence and significance of muscle tenderness during common migraine attacks. *Headache* 21, 49–54 (1981).

85 Hay KM. Pain thresholds in migraine. *Practitioner* 222, 827–833 (1981).

86 Hay KM. The treatment of pain trigger areas in migraine. *J. Roy. Coll. Gen. Pract.* 26, 372–376 (1981).

87 Guyuron B, Varghai A, Michelow BJ, Davis J. Corrugator supercilii muscle resection and migraine headaches. *Plast. Reconstr. Surg.* 106, 429–434 (2000).

88 Guyuron B, Tucker T, Davis J. Surgical treatment of migraine headaches. *Headache* 43, 302–303 (2003).

89 Guyuron B, Kriegler JS, Davis J, Amini SB. Comprehensive surgical treatment of migraine headaches. *Plast. Reconstr. Surg.* 115(1), 1–9 (2005).

90 Freitag FG. Preventative treatment for migraine and tension-type headaches: do drugs having effects on muscle spasm and tone have a role? *CNS Drugs* 17, 373–381 (2003).

91 Emre S, Erdem SR, Tuncer M. Does serotonin relax the rat anococcygeus muscle via 5-HT_7 receptors? *Naunyn Schmiedebergs Arch. Pharmacol.* 362, 96–100 (2000).

92 Boska MD, Welch KM, Schultz L, Nelson J. Effects of the anti-migraine drug sumatriptan on muscle energy metabolism: relationship to side-effects. *Cephalalgia* 20, 39–44 (2000).

93 Gobel H, Krapat S, Dworschak M, Heuss D, Ensink FB, Soyka D. Exteroceptive suppression of temporalis muscle activity during migraine attack and migraine interval before and after treatment with sumatriptan. *Cephalalgia* 14, 143–148 (1994).

Affiliation

- *Elliot Shevel, BDS, Dip, MFOS, MB, BCh*
  The Headache Clinic, Suite 256, P Bag X2600, Houghton, 2014, South Africa
  Tel.: +27 114 840 933
  Fax: +27 114 840 982
  drshevel@headclin.com