Comorbidities of chronic facial pain and obstructive sleep apnea

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Purpose of review
This article explains the high comorbidity of craniofacial pain (chronic face pain, temporomandibular disorders, and primary headaches) with obstructive sleep breathing disorders and obstructive sleep apnea (OSA). It is recommended that physicians treating OSA should be aware of the concurrent chronic pain that affects the quality of sleep, and also dentists treating chronic pain be aware of a sleep breathing origin so that proper reciprocal referrals be made for optimal patient treatment outcome.

Recent findings
These comorbid relationships are not limited to adults. The most recent literature demonstrates that children diagnosed with primary headaches are highly comorbid with OSA and frequently have chronic facial pain complaints.

Summary
It is recommended that patients who seek care for the symptoms of sleep-related breathing disorders (OSA), or patients seeking care for chronic head and face pain be screened with intake forms that include questions of both to insure optimal treatment outcomes for either chief complaint.

Keywords
chronic pain, craniofacial pain, obstructive sleep apnea, primary headaches, temporomandibular disorder

INTRODUCTION

Reviewing the current literature on chronic facial pain and obstructive sleep apnea (OSA) should begin with a broader definition. Chronic facial pain is inclusive of musculoskeletal disorders, orthopedic inflammatory disorders of the jaw such as temporomandibular disorders (TMDs), tooth and oral structures, as well as neuropathic disorders (neuralgia and neuritis) and autonomic disorders (referred pain). Musculoskeletal disorders can produce and/or aggravate primary headaches such as tension type, chronic daily headache, and migraine through the trigeminal spinal tract nucleus (centrally) and innervation (peripherally) by a sterile inflammatory orthodromic process. The term craniofacial pain is inclusive of these disorders and will be used as reference for this article. OSA and its associated central disturbance as respiratory effort-related arousals will be included to explain the mechanism of action. These relationships are not limited to adults.

One in six adults who visited a general dentist during the last year experienced chronic facial pain. Pain in the muscles and temporomandibular joints was reported as frequently as that in the teeth and surrounding tissues in patients visiting general dentists [1]. A meta-analysis of world literature has found that one in six children and adolescents has clinical signs of temporomandibular joint (TMJ) disorders [2▪]. Over 23% of preschool age children have pain when chewing and jaw joint noises [3]. All jaw joint noises are pathologic.

In the United States and throughout the world, the prevalence of OSA is increasing [4]. A total of 26% of the American population is at high risk of OSA [5]. In the same report, 57% of obese individuals were at high risk for OSA.

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KEY POINTS

- Patients with primary headaches and facial pain are at high risk for sleep breathing disorders and vice-versa.
- The high comorbidity of sleep breathing disorders and chronic face pain and primary headaches is not limited to adults. Children have the same or greater risk of these relationships.
- Screening for chronic face and primary headaches and sleep breathing disorders should be performed for all patients seeking care for either set of disorders.

CRANIOFACIAL DISORDERS (TEMPOROMANDIBULAR DISORDER) AND OBSTRUCTIVE SLEEP APNEA

An established relationship exists between OSA and TMD that is evident in the prevalence rates that are bidirectional. There is an increased prevalence of TMD in patients diagnosed with OSA [6]. There is an increased prevalence of OSA in patients diagnosed with TMD [7]. Two studies [8] tested the hypothesis that OSA signs and symptoms were associated with TMD: the Orofacial Pain: Prospective Evaluation and Risk Assessment (OPPERA) prospective cohort study of adults aged 18–44 years at enrollment ($n = 2604$) and the OPPERA case–control study of chronic TMD ($n = 1716$). Both studies supported a significant association between OSA symptoms and TMD, with prospective cohort evidence finding that OSA symptoms preceded first-onset of TMD: patients with two or more signs and/or symptoms of OSA had a 73% greater incidence of first-onset TMD. In a study designed to measure objective sleep parameters, sleep disorders, and TMD, young women with low BMI were found to have high rates of multiple sleep disorders and TMD, (bruxism 67%, insomnia 37%, and OSA 23.3%) [9].

PRIMARY HEADACHES AND OBSTRUCTIVE SLEEP APNEA

Headaches are the most prevalent neurological disorders and one of the most frequent symptoms reported in general practice [10,11]. Headache rates of up to 51% have been reported in children/adolescents [10]. Migraine is a highly prevalent disorder, currently estimated to occur in 10–18% of the population worldwide [12]. Prevalence of migraine is 7.7% in children and adolescents. Tension-type headache prevalence is 52%. The female preponderance of headaches emerges at puberty, with female children having a 1.5-fold greater risk of headaches and 1.7-fold greater risk of migraine than male children and adolescents [13–15]. Female sex, depression, coronary heart disease, chronic obstructive pulmonary disease, ischemic stroke, and hypertension are positively associated with migraine [16]. Migraine is the result of intracranial vascular swelling that results in compression of the A-delta and C fibers of the pia layer that surrounds the blood vessels in the meninges along with peripheral inflammation of any of the branches of the trigeminal nerve that results in central sensitization at the nucleus caudalis of the trigeminal spinal tract nucleus. There is a genetic predisposition for migraine [17].

Sleep disorders occur disproportionately among idiopathic primary headaches (migraine, tension-type, and cluster) and other headache patterns (chronic daily headache, 'awakening' or morning headache) irrespective of diagnosis [18]. It has been suggested that all headache patients, particularly those with episodic migraine and tension-type headaches, would benefit from evaluation of sleep disorders [19]. Children diagnosed with migraine are 8.25 times more likely to have a sleep breathing disorder, whereas children diagnosed with chronic tension-type headache are 15.23 times more likely to have a sleep breathing disorder [20].

PRIMARY HEADACHES AND TEMPOROMANDIBULAR DISORDER

TMD and primary headaches are comorbid. TMD symptoms are more common in migraine, tension-type headache, and chronic daily headache [21]. Women with migraine are more likely to have muscular and articular TMD [22]. Migraine is the most prevalent primary headache in patients with TMD [23]. Headache is one of the most commonly associated conditions observed in children and adolescents diagnosed with TMD [24–26]. Signs and symptoms of TMD occur more often in adolescents with headache in comparison with those who are headache-free [27,28].

CHRONIC WIDESPREAD PAIN/ PERIPHERAL NEUROPATHY AND SLEEP DISTURBANCES

Chronic pain, including musculoskeletal and joint pain, neck and back pain, afflicts about 20% of the adult population worldwide. It is the most common chronic pain syndrome encountered in general medicine and rheumatology [29]. Fibromyalgia is one of the main causes of chronic widespread pain. Pain has been found to be the most important determinant of subjective sleep quality [30]. The mechanism is the plastic change of the nervous

system to produce central nervous system sensitization. It is defined as 3 months of tenderness in 11 of 18 axial skeletal sites above and below the waist. Associated symptoms include fatigue, sleep disturbances, difficulties with memory and concentration, irritable bowel syndrome, headache, and depression [31]. Women are predominately affected by fibromyalgia between the ages of 35 and 55. Sex differences with patients with OSA demonstrate that women with OSA are more likely to have obesity, fibromyalgia, migraine, depression, and irritable bowel syndrome [32].

The U.S. Centers for Disease Control and Prevention states that approximately 60–70% of people who have diabetes also have comorbid neuropathy [33]. Effective reduction in pain improves sleep quality [34]. Longitudinal studies have shown that OSA is significantly linked to incident diabetes [35–40]. The mechanisms are chronic intermittent hypoxemia, recurrent arousals, and neurohumoral changes, resulting in metabolic disturbances including insulin resistance independently of other known risk factors.

INSOMNIA AND CHRONIC PAIN

The high prevalence of chronic insomnia among patients with chronic pain has been well established, with reported rates of insomnia as high as 88% [41,42].

The relationship between musculoskeletal pain and insomnia has been documented in the literature [43–46]. Musculoskeletal pain stimulates the sympathetic nervous system and when profound it produces plastic changes (central sensitization). Central sensitization or sympathetic dystrophy results in a sustained stimulation of the sympathetic state. The resultant stimulated adrenals produce an increase in cortisol that accelerates the metabolic rate, heart rate, and blood volume. This condition prevents the restful transition to sleep and contributes to insomnia. In this frightened state, the patient exhibits dilation of the bronchioles for increased oxygen intake and dilation of the pupils for optimum visual acuity. Blood is diverted to the vital organs of the heart and brain for optimal function, resulting in peripheral vasoconstriction with a symptom of cold hands and feet. Gastric peristalsis is reduced in the sympathetic state and results in abdominal discomfort and pain. Von Korff *et al.* published in *Pain*, 1993, that abdominal pain had an odds ratio of 6.3 versus headache at 4.3, TMD at 3.7 and back pain at 2.1 of predicting a chronic pain condition over a 3-year follow-up.

Sympathetic stimulation produces microarousals secondary to pain or breathing disturbance and results in excessive daytime sleepiness. Excessive daytime sleepiness is highly comorbid with primary headache [47–50]. In a study of 200 consecutive migraine patients, excessive daytime sleepiness (defined as an ESS score $\geq$ 10) was present in 37% of patients overall, and in 32.4 and 39.8% of patients with episodic and chronic migraine, respectively. In another study [49], chronic headache patients showed a higher prevalence of daytime sleepiness than control patients. In children, a headache disorder is a cumulative risk factor for disorders of excessive somnolence (odds ratio: 15.061) [51].

Children with juvenile idiopathic arthritis (JIA) demonstrate problems of initiating sleep and difficulty in maintaining sleep, which interferes with their ability to heal or to have favorable outcomes for their disease [52▪]. These conditions often manifest in the jaw joints and result in face pain (craniofacial pain).

SLEEP-RELATED BRUXISM

The face is the mirror of the body. Facial pain is the result of muscle contraction via central stimulation from pain anywhere in the body and/or an alteration/interruption of proper nasal breathing. Increased contracture of the elevator muscles of the jaw (temporalis and masseter) results in headache and face pain and holding the mouth open to breathe results in fatigue of the depressor muscles (mylohyoid, stylohyoid, and geniohyoid), and results in headache and facial pain.

Sleep-related bruxism, grinding and clenching of teeth, is classified as a sleep-related movement disorder by the International Classification of Sleep Disorders [diagnosis and coding manual (ICSD-3)] [53]. Sleep-related bruxism is reported in approximately 15% of the pediatric population and 8–31% of the general population, without a difference in prevalence between the sexes [54,55]. The characteristic electromyography (EMG) pattern of sleep-related bruxism is found in repetitive and recurrent episodes of rhythmic masticatory muscle activity (RMMA) of the masseter and temporalis muscles, which are usually associated with sleep arousals [56].

SB is divided into primary, or idiopathic sleep bruxism and secondary, or associated with a medical condition. Often the secondary form is iatrogenic through the prescription of drugs such as methylphemidate (Ritalin) for attention-deficit/hyperactivity disorder, antipsychotics: haloperidol (Haldol), lithium (Lithane), chlorpromazine (Thorazine), and selective serotonin reuptake inhibitors: fluoxetine (Prozac), sertraline (Zoloft), citalopram (Celexa), and calcium channel blocker: flunarizine

(Sibelium and cinnarizine) and antiarrhythmic: flecainide (Tambocor) [57]. Patients often use chemical substances that increase teeth grinding: alcohol, nicotine, caffeine, cocaine, and 3,4-methylenedioxymethamphetamine (MDMA).

Stress and anxiety have been given the bulk of emphasis for origin of facial pain through grinding of teeth. This has not held up to scrutiny. A study of patients with self-reported stress using EMG for 15 consecutive nights found no correlation between stress and bruxism. 'No overall relationship was established between electromyographic measures and the personality variables nor between electromyographic measures and self-reported stress. . .Subjects who believed in a stress-bruxism relationship reported greater stress' [58]. This thought process that jaw and face pain are the result of anxiety has also been challenged. A study designed to examine the extent of depression, anxiety, and somatization comorbidity with TMD found no statistically significant associations between anxiety and TMD in a population of 207 patients with TMD [59▪].

Microarousals, the result of central stimulation via the sympathetic system, produce oromotor nocturnal bruxism that results in facial pain [60–63]. Mandibular jaw movements are normal during sleep and are termed RMMA. RMMA movements are amplified when stimulated by the central nervous system via chronic pain and or obstructions of the airway. Obstructions of the airway include all four points: nasal valve, nasal-nasopharyngeal, velopharynx, and oropharynx (see Fig. 1).

In the apneic patient, the superficial masseter muscles are specifically stimulated by ventilator stimuli and increasing hypercapnia [64]. It is believed that the actions of jaw opening and muscle clenching, as seen with sleep-related bruxism, help to prevent pharyngeal collapse in patients with OSA [65–67]. Upper airway-resistance (UARS) or nasal obstruction, due to greater soft tissue inflammatory swelling, may be a mechanism as women with TMD have higher respiratory effort-related arousals (RERAs) in relation to TMD pain [68].

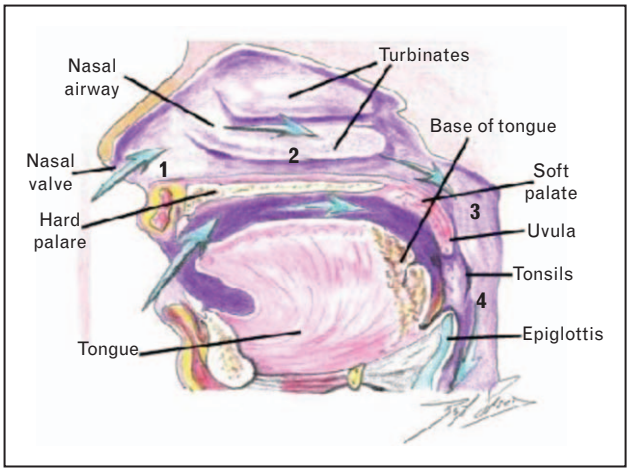

FIGURE 1. Four points of obstruction for proper nasal breathing.

Teeth grinding is an important symptom in screening for sleep disordered breathing and chronic pain. Teeth grinding causes stretching of the capsular ligaments of the TMJ. Ligament laxity allows for excessive disk movement or perforation in the disk itself resulting in jaw joint noise disease: disk displacement with reduction, then disk displacement without reduction (jaw locking) [69].

Daytime teeth clenching and grinding can often be the result of painful injuries to joint loading structures, so evaluation of the entire body is necessary in treating patients with facial pain complaints.

FORWARD HEAD POSTURE

For every inch the head is forward of the shoulders, it adds approximately 10 pounds of weight to the cervical and lumbar spine. The compressive load can result in osteoarthritis and nerve entrapment [70]. Craniofacial pain and internal derangement of the TMJs (TMD) manifest in forward head posture (FHP) [71]. The most common symptom of painful jaw joints is occipital cephalalgia at 94% [72]. The FHP is secondary to painful swallowing a postural adaption to injury. The injury described is in the absence of or in addition to a macrotrauma, and is the result of repetitive jaw compression (bruxism) originated by sympathetic stimulation during sleep. The patient wakes with temporal headaches and facial pain and jaw joint inflammation that now produces postural compensation. The cantilever strain of FHP, the result of extensor muscles of the neck (trapezius, splenius capititus, and semispinalis capititus), produces acute inflammation at their tendon insertions on the occiput. Decompressing inflamed jaw joints utilizing oral appliances, produced with a phonetic technique, has been found to upright the head 4.43 inches on average of a population of patients aged 13–74. This relates to relief of close to 45 pounds of weight from the cervical and lumbar spine [73]. Uprighting the head can eliminate the need for common therapies for migraine, which include botox injections for the tendon insertions on the occiput of the skull as well as the mouth closing muscles (temporalis and masseter), or severing the greater and lesser occipital nerves (often entrapped by the extensor muscle tendons they pass through).

FHP has also been found to be related to bruxism and nasal obstruction in children. 'Bruxism seems to

be related to altered natural head posture and more intense dental wear. A more anterior and downward head tilt was found in the bruxist group, with statistically significant differences compared to controls' [74]. Bruxism in children has been found to be related to RERA and OSA [75]. Expansion of the maxilla in mouth-breathing children restores proper nasal breathing and uprights the head [76,77]. Surgical retrusion of the mandible in prognathic conditions results in significant FHP, perhaps in defense of a compromised oropharyngeal airway [78].

CONCLUSION

The comorbid relationships of pain, obstructed sleep breathing (OSA and UARS), nasal obstruction, frequent awakenings, and daytime fatigue are well documented. It is clear that a patient intake questionnaire should be inclusive for chronic pain (specifically craniofacial pain) and disturbed sleep-related symptoms for all patients seeking care for either. This is specifically true for patients with cardiac or metabolic disorders, as there is a greater than 50% comorbidity with OSA than in the general population [79]. Those patients with primary headaches and facial pain are at high risk of sleep breathing disorders.

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There are no conflicts of interest.

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- ■ of special interest
- ■■ of outstanding interest

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