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CLINICAL REVIEW

Sleep disorders and chronic craniofacial pain: Characteristics and management possibilities

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SUMMARY

Chronic craniofacial pain involves the head, face and oral cavity and is associated with significant morbidity and high levels of health care utilization. A bidirectional relationship is suggested in the literature for poor sleep and pain, and craniofacial pain and sleep are reciprocally related. We review this relationship and discuss management options.

Part I reviews the relationship between pain and sleep disorders in the context of four diagnostic categories of chronic craniofacial pain: 1) primary headaches: migraines, tension-type headache (TTH), trigeminal autonomic cephalalgias (TACs) and hypnic headache, 2) secondary headaches: sleep apnea headache, 3) temporomandibular joint disorders (TMD) and 4) painful cranial neuropathies: trigeminal neuralgia, post-herpetic trigeminal neuropathy, painful post-traumatic trigeminal neuropathy (PTTN) and burning mouth syndrome (BMS). Part II discusses the management of patients with chronic craniofacial pain and sleep disorders addressing the factors that modulate the pain experience as well as sleep disorders and including both non-pharmacological and pharmacological modalities.

Introduction

Pain is a complex experience that encompasses cognitive, emotional, and discriminating sensorial dimensions, probably the most frequent and disabling symptom in medicine [1]. Chronic pain patients exhibit poorer sleep than controls in terms of sleep latency, sleep efficiency and awakenings after sleep onset [2,3]. Fifty to seventy percent of patients with chronic pain conditions report interference with sleep [4], with pain rated as the sole reason for sleep disturbances in most of these patients. A bidirectional relationship is suggested in the literature for poor sleep and pain, and a reciprocal relationship between chronic craniofacial pain and sleep has been described [1,5].

When insomnia and chronic pain occur together their consequences are even more devastating [2], and treatment of one can be beneficial for the other, [6,7]. Sleep impairments reliably predict new incidents and exacerbations of chronic pain, more than pain predicts sleep impairments [5].

Pain-related awakening occurs in about one third of patients with persistent craniofacial pain conditions, and correlates with pain intensity [8].

The aim of the present review is to provide an update on the relationship between chronic craniofacial pains and sleep disturbances and their management options. It is a narrative, clinically orientated review with references to relevant literature. The relevant literature published in English language journals in the past 20 years was reviewed. Extracted data included self reported and polysomnographic available research. Adult-based outcomes, which occupy the major bulk of the literature, were primarily included. When available, relevant adolescent or child-based studies were included. We focus only on chronic craniofacial pain conditions and their association with sleep, and not on acute craniofacial pain or causality/mechanisms, which are discussed elsewhere [5,9,10].

Part I will discuss chronic craniofacial pain as related to sleep disorders and Part II will discuss their management.

Part I. Chronic craniofacial pain and sleep disorders

Classification of craniofacial pain. Approximately 39 million American adults suffer from chronic craniofacial pain [11]. Chronic

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Abbreviations

BMS	burning mouth syndrome
CBT	cognitive behavioral therapy
CH	cluster headache
CPAP	continuous positive airway pressure
DC/TMD	diagnostic criteria for temporomandibular disorders
FDA	food and drug administration
HH	hypnic headache
ICHD-3 beta	International classification of headache disorders, 3rd edition, beta version
ICSD-3	International classification of sleep disorders
MMD	masticatory muscle disorders
MRI	magnetic resonance imaging
NSAIDs	non steroidal anti-inflammatory drugs
OSA	obstructive sleep apnea
PH	paroxysmal hemicranias
PHN	post herpetic neuralgia
PSG	polysomnography
PSQI	Pittsburgh sleep quality index
PHTN	post-herpetic trigeminal neuropathy
PTTN	painful post-traumatic trigeminal neuropathy
REM	rapid-eye-movement
RLS	restless legs syndrome
SDB	sleep-disordered breathing
SRB	sleep related bruxism
SUNCT	short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing
TAC	trigeminal autonomic cephalalgias
TMD	temporomandibular disorders
TMJ	temporomandibular joint
TN	trigeminal neuralgia
TTH	tension-type headache
VAS	visual analog scale

craniofacial pain will be discussed according to four major diagnostic categories classified according to the: International classification of headache disorders, 3rd edition, beta version (ICHD-3 beta) [12], diagnostic criteria for temporomandibular disorders (DC/TMD) [13], and a recent comprehensive review of craniofacial pain by Sharav and Benoliel [10]. Additionally, the International Classification of Sleep Disorders (ICSD-3) is used [14].

Chronic craniofacial pains discussed in this review are detailed in Table 1 and include: 1) primary headaches: migraines, tension-type headache (TTH), trigeminal autonomic cephalalgias (TACs) and hypnic headache, 2) secondary headaches: sleep apnea headache, 3) temporomandibular joint disorders (TMD) and 4) painful cranial neuropathies: trigeminal neuralgia, post-herpetic trigeminal neuropathy, painful post-traumatic trigeminal neuropathy (PTTN) and burning mouth syndrome (BMS).

Headaches and sleep

Headaches and sleep are closely related [15–19] and most epidemiological studies characterize headaches according to frequency or proximity to sleep (i.e., chronic daily, awakening, or morning headache) rather than the formal diagnoses of the ICHD [12,15].

People with headache have fragmented sleep, frequent awakenings, and changes in sleeping position [15]. Children with headaches complain about sleep quality and experience excessive daytime sleepiness [20]. Insomnia is the most common sleep complaint in headache clinics, observed in half to two-thirds of patients [21].

Sleep quantity is reduced in primary headaches, with symptoms of central sensitization, such as allodynia and pericranial tenderness [2]. Average sleep duration (7–8 h) was associated with reduced headache, whereas the extreme ends of sleep durations (i.e., short [<6 h] long [>8.5 h]) are associated with increased headache intensity [15].

Sleep can both trigger and cure headaches, for example, sleep can be a trigger factor in cluster headache (CH) [22], whereas in migraines and TTH it both triggers and alleviates the problem [15]. Headache may be the first symptom of a sleep disorder [16]. The International classification of sleep disorders (ICSD) classifies migraine, hypnic headache, CH and chronic paroxysmal hemicrania as “sleep-related headaches” [15,16].

Primary headaches and sleep

Migraine and sleep

Migraine is characterized by unilateral moderate to severe pain of a paroxysmal nature, with a pulsating quality, aggravated by routine physical activity and associated with nausea and/or photophobia and phonophobia [12]. There are two main subtypes: migraine without aura and migraine with aura, primarily characterized by the transient focal neurological symptoms that usually precede or sometimes accompany the headache [12]. Migraine attacks occurring more than 15 days a month are termed chronic migraine [12].

Disturbances in sleep are highly prevalent among migraine sufferers, including: restless legs syndrome (RLS), parasomnias, daytime sleepiness, poor sleep quality as well as insomnia [3,15,23,24]. Insomnia-like symptoms, shortened sleep periods and poor sleep quality are associated with a worse symptom profile and increased disability among patients with migraine [23]. Non-apnea sleep disorders are considered an independent, predisposing factor for developing subsequent migraine in adulthood [19].

Migraine and insomnia. Insomnia is the most common sleep disorder associated with migraine [15], occurring in 1/2 to 2/3 of individuals in headache clinics (vs 10.8% of the general population) [21,23]. Migraine is significantly related to insomnia symptoms with odds ratio (OR) estimates ranging from 1.4 to 1.7 and from 2.0 to 2.6, for frequent, comorbid or severe headache [25].

Migraine and obstructive sleep apnea (OSA). Although 30%–70% of OSA patients suffer from headaches [6], no relationship has been found between OSA and migraine in the general population [26]. However, migraine in patients with OSA improves when OSA is treated [27].

Migraine and parasomnias. A higher incidence of parasomnias, including somnambulism, sleep talking and nightmares, has been documented in children with migraine [24].

Migraine and restless legs syndrome (RLS). Higher lifetime prevalence and worse symptoms of RLS were found in migraine patients, associated with impaired sleep quality, sleep latency and sleep disturbances, more sleep medication and daytime dysfunction [24].

Migraine and sleep related bruxism (SRB). SRB is repetitive jaw muscle activity characterized by clenching or grinding of the teeth and/or bracing or thrusting of the mandible [28].

The prevalence of orofacial pain in subjects with SRB ranges from 66% to 84% in different studies [6]. Frequent headaches have been associated with SRB, in both adults and children [6,29].

Sleep as trigger of migraine. Sleep disturbances are among the common “triggers” of migraine, reported in nearly 50% of sufferers [23]. Lack of sleep is frequently reported as precipitating migraine [26] and so is excess sleep; also reported in pediatric migraine [30]. In a study involving polysomnography (PSG) and pressure, heat, and cold pain thresholds relative sleep deprivation, increased strain

Table 1
Characteristics of chronic craniofacial disorders.

Craniofacial pain disorder	Clinical presentation
Primary headaches	
Migraine	Unilateral pain
	Moderate or severe pain
	Paroxysmal nature
	Pulsating quality
	Aggravation by routine physical activity
	Associated with nausea and/or photophobia and phonophobia
Tension-type headache	Bilateral pain in the temple and occipital areas
	Pressing or tightening quality
	Mild to moderate intensity
	Does not worsen with physical activity
	Not associated with nausea
	Photophobia or phonophobia may be present
Cluster headache	Severe short throbbing pain
	Unilateral, periorbital pain
	Major autonomic activation
	Distinct circadian/circannual pattern
	Attacks occur in series for weeks or months
	Remissions of several months to years
	Divided into episodic type (80–85%) and chronic type (15%)
Hypnic headache	Frequently recurring headache attacks developing only during sleep, causing wakening
	Lasting from 15 min to 4 h, occurring at least 10 days per month. for more than three months
	No cranial autonomic symptoms or restlessness
	Not attributed to other pathology
Secondary headaches	
Sleep apnea headache	A morning headache caused by OSA
	Usually bilateral
	Recur >15 days/month
	Duration less than 4 h
Temporomandibular disorders	Pain in the pre-auricular region and/or masticatory muscles
	TMJ sounds
	Limitation of mandibular movement
	Can be cyclical and self-limiting
Painful cranial neuropathies	
Trigeminal neuralgia	Recurrent unilateral brief electric like pains
	Abrupt in onset and termination
	Limited to the distribution of the trigeminal nerve
	Triggered by innocuous stimuli
	Refractory period after triggering
Post-herpetic trigeminal neuropathy	Caused by herpes zoster
	Unilateral head and/or facial pain
	Pain in the distribution of the trigeminal nerve
	Persisting or recurring for at least three months
	Variable sensory changes
Painful post-traumatic trigeminal neuropathy	Pain following trauma to the trigeminal nerve
	Unilateral facial or oral pain
	Other symptoms and/or clinical signs of trigeminal nerve dysfunction may be present
Burning mouth syndrome	Intraoral burning or dysaesthetic sensation recurring daily for more than 2 h.
	Persisting for more than three months
	Without clinically evident causative lesions

OSA: obstructive sleep apnea, TMJ: temporomandibular joint.

and varying robustness of the neurobiological arousal system may be among the causal factors of migraine [17]. OSA, insomnia, circadian rhythm disorders, parasomnias, and daytime sleepiness were shown to increase the risk of chronic headache [15].

Sleep as relieving factor for migraine. Sleep may relieve pain and terminate migraine attacks in both adults and children [6,31], and may be used as an abortive modality [31]. However, rest without sleep is less effective in pain relief.

Tension type headache (TTH) and sleep

Pain is typically bilateral, pressing or tightening in quality and of mild to moderate intensity, lasting minutes to days, does not worsen with routine physical activity, usually not associated with nausea, but photophobia or phonophobia may be present [12]. TTH has episodic and chronic (>15 headache days per month) subtypes [12].

Insomnia is the most common sleep disorder associated with TTH [15]. However, sleep diaries revealed normal sleep times and polysomnography had signs of increased sleep quality [18]. TTH patients may therefore have a greater need for sleep than healthy controls and are thus relatively sleep deprived [18]. In a manner similar to migraine, sleep dysregulation triggers episodic TTH, and sleep disorders may complicate and exacerbate headache [32].

TTH and OSA. OSA does not influence the presence and frequency of TTH, and there is no pathophysiological link between TTH and OSA [33,34].

TTH and SRB. In a polysomnographic (PSG) study, 50% of children with TTH had SRB versus 2.4% of children with non-tension type headaches [29].

Trigeminal autonomic cephalalgias (TACs) and sleep

TACs are primary headaches, characterized by strictly unilateral pain with accompanying autonomic symptoms [12]. TACs include cluster headache (CH), paroxysmal hemicranias (PH), short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT), short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA) and

hemicrania continua [12]. TACs are usually separated by the duration and frequency of the pain attacks and diurnal rhythmicity [12,16].

Sleep and CH are believed to be interconnected, but the relationship with the other TACs is uncertain and complex, with only a few case reports on the subject [16].

Cluster headache (CH) and sleep

CH is the archetypical TAC, characterized by distinct circadian and circannual patterns of severe short, unilateral periorbital pain and autonomic activation [12]. Attacks occur in series of weeks or months, separated by remissions of several months or years [35]; divided into episodic type (80–85%) and chronic type (15%), in the latter pain is present between clusters.

The International classification of sleep disorders classifies CH as a sleep related headache [14]. More than 70% of CH patients report nocturnal attacks, often waking them from sleep [16,36].

Association between CH and REM sleep. PSG studies demonstrated that sleep precipitates CH attacks in some patients especially during the REM stages [16,37]. Thus, cluster attacks began 90 min after sleep onset, coinciding with the first REM phase, with a reduced percentage of REM sleep, longer REM latency and fewer arousals [37]. However, other PSG studies did not confirm the association between CH and REM sleep [16,38].

OSA and CH. OSA prevalence in CH may be as high as six in 10 [39], and the OR for CH is increased by at least 8-fold by OSA [15,16]. Although most headache sufferers will not be diagnosed with OSA, patients with CH are at significant risk for OSA [15]. Balrose et al., questioned whether these results considered lifestyle factors relevant for sleep-disordered breathing (SDB) such as obesity and/or smoking [16]. Other PSG studies only found the association during active cluster periods [40]. Indeed, in PSG no difference was found in the prevalence of sleep apnea between CH and matched controls [37]. Nevertheless, isolated cases of improvement of CH upon treatment of OSA have been reported, with questionable causal relationship [16].

Hypnic headache (HH)

Hypnic headache is a rare disorder characterized by frequently recurring headache attacks starting during sleep, causing wakening and lasting from 15 min to 4 h, occurring at least 10 days per month for more than three months, without cranial autonomic symptoms and not attributed to other pathology [12,41–43]. HH is more common in women (male/female 1:1.5) and usually begins after the age of 50 y [41,43].

Pain is usually bilateral, mild to moderate [12] and most attacks occur between 02:00 and 04:00 h [41]. The features of HH resemble TTH, but recent studies found that migraine-like features, such as nausea may be present during attacks [12,41]. Cranial magnetic resonance imaging (MRI) is essential to rule out secondary causes of nocturnal headache such as pituitary, posterior fossa, or brainstem lesions [43]. The presence of OSA does not necessarily exclude the diagnosis of HH [12].

Secondary headaches

Sleep apnea headache

Sleep apnea headache is a morning headache, usually bilateral, occurring more than 15 days/month, lasting less than 4 h, caused by OSA and resolving with successful OSA treatment [12]. A growing body of evidence supports a relationship between SDB and other idiopathic pain conditions, including headache [15].

It should be noted that although morning headache is significantly more common in patients with OSA than in the general population, morning headache is a non-specific symptom of several other sleep and headache disorders [12].

Temporomandibular disorders (TMDs) and sleep

TMD is a common musculoskeletal condition, affecting 5–12% of the population, and is often associated with pain in the pre-auricular region and/or masticatory muscles, temporomandibular joint (TMJ) sounds and mandibular movement dysfunction [10,13]. Up to 90% of TMD patients have comorbid sleep disorders [44–46], with up to 70% meeting the criteria for at least one sleep disorder, and 43% meeting the criteria for two or more [47]. Sleep disturbances have been implicated as perpetuating factors in non-responding TMD patients and with poor treatment outcomes [44,45].

TMDs usually do not wake from sleep, but up to 24% of patients with a high muscle tenderness score reported pain-related awakening [8]. The Pittsburgh sleep quality index (PSQI) components most affected by TMDs were subjective sleep quality, sleep latency, habitual sleep efficiency and sleep disturbance [44]. Within TMDs, patients with myofascial pain reported significantly poorer sleep than those with temporomandibular joint pain or controls [44,48].

Insomnia is the most common sleep disorder in patients with TMD, and changes in insomnia symptom severity predict increases in TMD pain [49].

OSA and TMDs. TMDs are associated with OSA (28%) [50]. Other polysomnographic results did not show differences in sleep or respiratory measures between TMD patients and controls [51]. Recently, TMD patients demonstrated a higher percentage of stage N1 sleep and a higher respiratory effort related arousal index, but overall sleep and respiratory disturbances were mild [52]. However, sleep apnea symptoms can prospectively predict first-onset TMD and chronic TMD [53].

SRB and TMDs. There are numerous reports of SRB and the presence of painful symptoms of TMD [54]. Yet no correlation between TMD and the frequency of sleep bruxism activity has been found [55] and no causal relationship has been established [54].

Neuropathic orofacial pain and sleep

The studies presented should be interpreted with caution as sleep assessment based on self report and the one based on PSG recordings may not be coherent and concordant. Moreover, PSG studies are lacking in specific craniofacial pains such as BMS, PTTN and TN.

Trigeminal neuralgia (TN) and sleep

TN is characterized by recurrent unilateral brief electric shock-like pains, abrupt in onset and termination, in the distribution of the trigeminal nerve and triggered by innocuous stimuli [12]. Classical TN usually affects more the second or third divisions of the trigeminal nerve [10].

Although some clinicians report that TN does not usually awaken patients from sleep, 60% of TN patients report at least occasional awakening typically induced by innocuous stimuli at TN trigger points [56], and 22.6% of patients reported pain-related awakenings with background pain as most significant predicting factor [8].

Post-herpetic trigeminal neuropathy (PHTN) and sleep

PHTN is a unilateral head and/or facial pain persisting for at least three months along the distribution of the trigeminal nerve, with variable sensory changes, caused by herpes zoster [12]. The pain is usually burning and patients often have allodynia, anesthesia and dysesthesia within the affected dermatomes [10,12].

Pain can be severe, and is associated with impaired sleep and quality of life [57]. Objective analyses, such as polysomnography, show that patients with PHN have reduced sleep efficiency, more

fragmented sleep, reductions in stages 3, 4, and REM sleep, and an increase in stage 1 sleep when compared with controls [58].

Painful post-traumatic trigeminal neuropathy (PTTN)

PTTN is a unilateral facial or oral pain following trauma to the trigeminal nerve, accompanied by other symptoms and/or signs of trigeminal nerve dysfunction [12].

Sleep was significantly more impaired in patients with inferior alveolar nerve injuries compared to lingual nerve injuries [59]. PTTN patients were about four times more likely to be woken up from sleep than controls, with 41% pain-related awakenings [8].

Burning mouth syndrome (BMS) and sleep

BMS is an intraoral burning/dysesthetic sensation, recurring daily for more than 2 h a day for a period of more than three months, without clinically evident causative lesions [12]. Prevalence ranges from 0.7 to 4.6% [60], usually occurring in the fifth to seventh decade of life with a high postmenopausal female prevalence [12,60]. The pathogenesis is poorly understood, involving physiological and psychological factors [60].

BMS patients exhibit significant decreases in sleep quality compared with control groups and the prevalence of poor sleep in patients with BMS is between 67 and 80% [60,61]. BMS patients report a greater degree of sleep disorders, with higher scores in all items of the PSQI questionnaire compared to controls [60]. A depressed mood and anxiety correlated positively with sleep disturbances among BMS patients [60,61]. Conversely, patients with sleep disorders were found to have a higher risk of developing BMS [62].

The studies presented should be interpreted with caution as sleep assessment based on self report and the one based on PSG recordings may not be coherent and concordant. Moreover, PSG studies are lacking in specific craniofacial pains such as BMS, PTTN and TN

Part II. Management of patients with chronic craniofacial pain and sleep disorders

The management of patients with chronic pain conditions and sleep disorders needs to be based on a comprehensive, bio-psycho-social model of disease, and should address all the factors that modulate the pain experience and sleep disorders [10,63]. The multiple interactions between craniofacial pain and sleep highlight the necessity of an interdisciplinary approach to managing craniofacial pain-related sleep disorders [6,64]. Pain management should be addressed by the craniofacial pain specialists [10] in collaboration with a clinical team that usually includes dentists, sleep medicine specialists and other specialties as needed, including family and internal medicine, pediatrics, ENT (ear nose and throat), pulmonology, neurology, anesthesiology, oral and maxillofacial surgeons and psychologists/psychiatrists [64]. The dentist has a role in screening and assessing craniofacial pain and sleep disorders, referring patients to specialists as needed, managing these patients in collaboration with a physician, fabricating oral appliances for OSA and SRB [65] as well as in the short and long term follow-up [64]. Various dental specialists may play a significant role in the management of these patients. For example, pain management may be addressed by the orofacial pain specialists [10], maxillomandibular surgery to treat OSA is usually performed by an oral and maxillofacial surgeon or ENT and orthodontic treatment may be needed to ensure stable repositioning following surgery as well as to prevent exacerbation of OSA in children through guided craniofacial grow [64].

Screening and diagnosis. Craniofacial pain patients should be screened for sleep problems during their first visit and during follow-up visits (Table 2, Fig. 1). Diagnostic work-up of craniofacial patients ought to include examination of craniofacial pain patterns (Table 1) [10] in relation to the sleep/wake cycle assessment [15]. Patients should be asked to keep sleep and craniofacial pain diaries for at least a few weeks to monitor the frequency and variations of pain and sleep [15], but diaries should be carefully evaluated [46]. Risk factors for OSA and for SRB should also be assessed [64]. Further diagnostic investigations should be employed as needed, including validated sleep interviews, sleep questionnaires, full-night polysomnographic study, and neuroimaging [66–70]. The choice of an instrument primarily depends on the interest of the clinician and whether they are collecting objective or subjective data concerning sleep [46]. The inclusion of instruments measuring different facets of sleep disturbances is recommended [46,70]. Future studies should include mediator variables such as cognitive, emotional arousal and thoughts [46].

Interventions. Management of patients with craniofacial pain and sleep disorders can be abortive or prophylactic, tailored to individualized needs (see Fig. 1 and Table 3). Additionally, patients should receive therapy for co-morbid medical conditions, psychiatric illnesses, and/or substance abuse, that may be precipitating or exacerbating their craniofacial pain and/or their sleep disorder [63] and patients should be referred to the relevant medical discipline according to the precipitating illness.

Management of craniofacial pain (Table 3)

Management of headaches

The known effects of headache treatments on sleep should be considered, to avoid worsening pre-existing sleep complaints, or to potentiate the sleep effects of the medications in a constructive way [71]. For example, gabapentin, codeine and morphine can interfere with sleep quality and selective serotonin reuptake inhibitors such as fluoxetine, sertraline, and paroxetine, can trigger or aggravate movement during sleep [72] including bruxism [73].

Management of migraine

The management of migraine involves non-pharmacological approaches, such as sleep hygiene, detection of triggers with a pain diary, as well as stress management, relaxation techniques and cognitive behavioral therapy (CBT) [10,74]. Pharmacological treatment can be abortive aiming to stop the pain attack, or prophylactic taken daily to reduce the severity, duration, and frequency of migraine attacks [10,74,75]. Patients with frequent severe headaches require both approaches [10,75]. Drugs effective for abortive treatment of migraine include: analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs), triptans and antiemetic/dopamine receptor antagonists [10,75].

Recommendations for the preventive treatment of episodic migraine headache include: level A medications, such as divalproex sodium, topiramate, metoprolol, propranolol and timolol [76]. Second line (level B), included amitriptyline and venlafaxine as well as atenolol and nadolol [76]. Additionally, onabotulinum toxin A (botox) has been approved by the U.S. food and drug administration (FDA) for preventing chronic migraine [77].

Management of tension type headache (TTH)

Non-pharmacological management of TTH is similar to migraine, and pharmacological treatment include NSAIDs as well as tricyclic antidepressants [10,78]. Based on efficacy and safety profiles, expert recommendations suggest ibuprofen or naproxen sodium as drugs of choice for abortive TTH treatment [10,79]. Prophylactic therapy is recommended for patients who suffer from chronic TTH and includes psychologic, physiotherapeutic and TMD-aimed treatments as well as pharmacological treatments such as tricyclic antidepressants [10].

Table 2
Screening for sleep disorders associated with craniofacial pain.

Examining the of craniofacial pain pattern and history in relation to the sleep/wake cycle
Assess pre-sleep activities, sleep environment, lifestyle habits, bedtime routine
Specific validated questionnaires to evaluate sleep complaints
Record reports of partners and parents for child patients
Assess secondary causes for sleep disorders such as a medical conditions, psychiatric illnesses, stress, medications (including drug abuse), alcohol
Extra-oral examination should assess risk factors for OSA and SRB
Intra-oral examination should assess risk factors for OSA and SRB
Sleep and headache diaries should be kept for a few weeks
Further diagnostic investigations as needed (e.g., polysomnography, neuroimaging)

EEG: electroencephalogram, EMG: electromyogram, OSA: obstructive sleep apnea, SRB: sleep related bruxism.

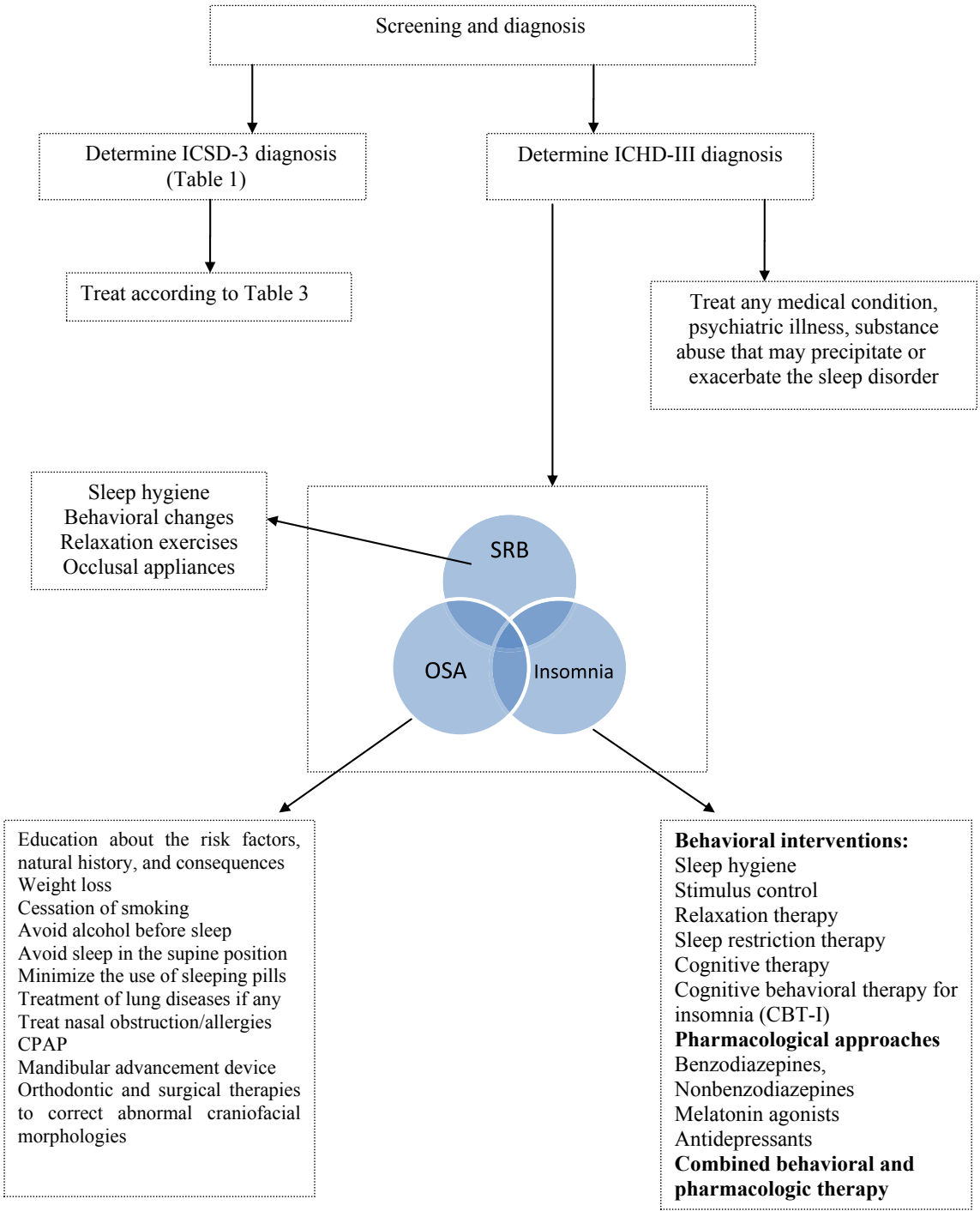

Fig. 1. Algorithm for management of patients with sleep and craniofacial pain. Overlap between conditions is common. The algorithm is empirical, based on expert experience and extrapolated from other pain or sleep disordered conditions. CPAP: continuous positive airway pressure, ICHD-3 beta: International classification of headache disorders, 3rd edition, beta version, ICSD-3: International classification of sleep disorders, OSA: obstructive sleep apnea, SRB: sleep related bruxism.

Table 3
Craniofacial pain disorders associated with sleep disorders and their management.

Craniofacial pain disorders	Treatment
Primary headaches	
Migraine	**Nonpharmacological approaches:** Sleep hygiene Detection of triggers with a pain diary Stress management Relaxation techniques CBT **Abortive migraine treatment:** Analgesics, NSAIDs, triptans Antiemetic/dopamine receptor antagonists **Prophylactic migraine treatment** Anticonvulsants:divalproex sodium, topiramate Beta blockers:metoprolol, propranolol and timolol Antidepressants:amitriptyline and venlafaxine **Prophylactic chronic migraine treatment** Onabotulinum toxin A (Botox)
Tension-type headache	**Nonpharmacological approaches:** Sleep hygiene Detection of triggers with a pain diary Stress management Relaxation techniques CBT **Pharmacological approaches** NSAIDs Tricyclic antidepressants
Cluster headache	**Abortive CH Treatment:** Oxygen Triptans Dihydroergotamine (DHE) parenteral or intranasal **Bridge therapy** Steroids **Prophylactic CH treatment:** Verapamil Steroids Lithium carbonate Topiramate Divalproex sodium Melatonin Indomethacin
Hypnic headache	**Prophylactic treatment** Lithium Caffeine Indomethacin Verapamil Flunarizine Gabapentin Topiramate Melatonin Amitriptyline
Secondary headaches	
Sleep apnea headache	Resolves with successful treatment of the OSA
Temporomandibular disorders	**Nonpharmacological approaches:** Limiting jaw movements Soft diet Parafunctional habit modification Moist heat and/or ice therapy Occlusal appliances Sleep hygiene Detection of triggers with a pain diary Stress management Relaxation techniques CBT (Cognitive behavioral therapy) **Pharmacological approaches**: Analgesics, NSAIDs Antidepressants Muscle relaxants Local anesthetics Injectable corticosteroids, hyaluronate and botulinum toxin **Surgical interventions** Arthrocentesis, arthroscopy, arthrotomy
Painful cranial neuropathies	
Trigeminal neuralgia	**Pharmacological approaches:**

(*continued on next page*)

Table 3 (*continued*)

Craniofacial pain disorders	Treatment
	Carbamazepine
	Oxcarbazepine
	Baclofen
	Lamotrigine
	Gabapentin
	Pregabalin
	Neurosurgical options:
	Microvascular decompression
	Gamma knife radiosurgery
	Gasserian ganglion percutaneous techniques
Post-herpetic trigeminal neuropathy	Tricyclic antidepressants
	Gabapentin
	Pregabalin
	Opioids
	Topical capsaicin
	Topical lidocaine
Painful post-traumatic trigeminal neuropathy	**Nonpharmacological approaches:**
	Reassurance
	CBT
	Pharmacological approaches:
	Topical 5% lidocaine patches
	Tricyclic antidepressants
	Gabapentin
	Pregabalin
	Carbamazepine
Burning mouth syndrome	**Nonpharmacological approaches:**
	Reassurance
	CBT
	Pharmacological approaches:
	Analgesics
	Tricyclic antidepressants
	Gabapentin
	Pregabalin
	Clonazepam
	Antifungal
	Sialagogues
	Anxiolytics
	Antipsychotics
	Alpha-lipoic acid
	Topical capsaicin
	Topical lidocaine
	Vitamin, mineral, and hormonal replacements

CBT: cognitive behavioral therapy, CH: cluster headache, NSAIDs: non steroidal anti-inflammatory drugs.

Management of cluster headache (CH)

Abortive treatment of episodic CH includes oxygen, triptans or dihydroergotamine (DHE) [80]. Subcutaneous sumatriptan and parenteral DHE have a US FDA indication for CH. Verapamil, steroids, lithium carbonate, topiramate, divalproex sodium, melatonin and indomethacin, are used for preventive treatment [80,81]. Corticosteroids can serve as a short term bridge therapy for 5–7 days, to allow the patient to comfortably initiate prophylactic therapy with verapamil [80].

Hypnic headache (HH)

Treatment is prophylactic, using lithium, caffeine and indomethacin, verapamil, flunarizine, gabapentin, topiramate, melatonin and amitriptyline [12,43].

Management of temporomandibular disorders (TMD)

Many TMDs are cyclical and self-limiting, with periods of complete remission of symptoms, thus conservative reversible treatment is recommended [10,78]. Primary treatment options include home self-care programs, pharmacological care and surgical care. Home care programs involve resting the masticatory muscles by limiting jaw movements, soft diet, parafunctional habit modification (oral para-functional habits include any habitual use of the mouth unrelated to eating, drinking, or speaking. (e.g bruxism, fingernail biting, pecil or pen chewing)) and moist heat and/or ice therapy [10,78]. Occlusal appliance therapy is a useful adjunct therapy for some TMD cases [10,78]. Commonly used pharmacological agents for TMD treatment include analgesics, NSAIDs, antidepressants, muscle relaxants, local anesthetics, oral and injectable corticosteroids, sodium hyaluronate injections and botulinum toxin injections [10,78]. Surgery, such as arthrocentesis, arthroscopy and arthrotomy, is only indicated when non-surgical therapy has failed [10,78].

Management of painful cranial neuropathies

Management of trigeminal neuralgia (TN)

Carbamazepine is recommended as first line of therapy (level A evidence), and oxcarbazepine (level B evidence) is recommended for patients who do not respond to or tolerate carbamazepine. Baclofen, lamotrigine as well as gabapentin and pregabalin are second line drugs [10,82]. Neurosurgical options, such as microvascular decompression, gamma knife radiosurgery and gasserian ganglion percutaneous techniques (level C evidence), may be considered if medications are no longer effective or if unmanageable side effects develop [10,78,82].

Management of post-herpetic neuralgia (PHN)

First-line treatments for PHN include tricyclic antidepressants, gabapentin, pregabalin and topical lidocaine while second- and

third line treatments include opioids, topical capsaicin and tramadol [10,82].

Management of burning mouth syndrome (BMS)

Management of BMS includes topical medications, systemic medications, behavioral therapies (e.g., reassurance, CBT) and combined psycho-pharmacotherapy [10]. Medications used to treat BMS include analgesics, tricyclic antidepressants, gabapentin or pregabalin, clonazepam, antifungal, sialagogues, anxiolytics, antipsychotics, alpha-lipoic acid, topical capsaicin, topical lidocaine and vitamin, mineral, and hormonal replacements [10,83], although supporting evidence is scant and on the whole BMS does not readily respond to therapy [10].

While evidence based treatments for migraines, TTH, CH and TN exist and are presented above, it should be noted that the treatments presented for TMD, BMS and HH are based on the current literature, and not on evidence based treatment guidelines.

Management of sleep disorders in craniofacial pain conditions (Fig. 1)

Treatment of the underlying sleep disorders (SRB, OSA and RLS) can greatly reduce craniofacial pains such as headaches [6]. Considering that there is often an overlap between sleep disorders (as depicted in Fig. 1), a combination of treatment modalities should be employed.

Management of OSA. Conservative management includes sleep hygiene, weight loss, cessation of smoking, and treatment of related medical conditions [65,84]. Continuous positive airway pressure (CPAP) [85,86] or a mandibular advancement appliance device during sleep may be utilized [65]. CPAP or BiPAP devices may improve or resolve headaches [87]. For example, CPAP has reduced pain scores, attack duration and frequency of migraines in OSA patients [27].

Management of SRB. Management consists of sleep hygiene, behavioral strategies, pharmacotherapy, and oral appliance, although these management strategies are not yet fully evidence-based [64,69]. Furthermore, the initial decrease in the rhythmic masticatory muscle activity index in the first period of treatment with occlusal splints seems to be transitory, and values return to baseline after a short time or even show increased EMG activity during sleep [69]. When SRB is related to OSA using CPAP may eliminate bruxism during sleep [88]. Mandibular advancement devices may also reduce SRB [89] and may be considered for SRB patients with concomitant OSA [69].

Treatment of insomnia -behavioral interventions. Treatment of insomnia should begin with non-pharmacological therapy, because of the potential side effects, associated with pharmacological therapy [66]. Behavioral therapies beyond sleep hygiene and stimulus control include relaxation therapy, sleep restriction therapy, cognitive therapy and cognitive behavioral therapy for insomnia (CBT-I) [10,66,90,91].

CBT-I approaches for pain management in headaches [92] and TMD [93] provide relief. Indeed, poor sleep hygiene contributes to sleep disturbances among patients with chronic migraine [94], and targeted behavioral sleep intervention to modify this issue reduced headache frequency and the headache index, and chronic migraine reverted to episodic migraine in adults [31] and children/adolescents [95].

However, a recent meta-nalysis of group CBT-I found medium to large effect sizes for sleep onset latency, sleep efficiency, and wake after sleep onset and small effect sizes for pain outcomes [96]. Hybrid behavioral therapies that combine elements for pain and sleep may be promising [10].

Pharmacological interventions. Pharmacological intervention is indicated when non-pharmacological measures do not produce the desired improvement [63,66]. None of the medications used to treat insomnia has been approved by any medical agency in the context of co-morbid neurological disease and are therefore off-label [97]. Another approach involves combination therapy, where CBT-I and a medication are initiated (usually for six to eight weeks), then the medication is tapered off or used as-needed while continuing CBT [98,99].

Discussion of individual drugs used for the management of sleep disorders associated with chronic craniofacial pain is beyond the scope of this review. A short list of drug classes is presented in Fig. 1.

Practice points

1. Craniofacial pain and sleep are reciprocally related: pain may lead to sleep difficulties that, in turn, may exacerbate pain, i.e., a vicious cycle.
2. Sleep deprivation is considered a risk factor for headache and headache can be the first symptom of a sleep disorder.
3. Sleep can be both a trigger and cure for headache.
4. Insomnia is the most common sleep disorder associated with migraine and TTH.
5. Assessing sleep history and sleep quality and disturbances should be part of the routine diagnostic work-up for craniofacial pain patients.
6. The management of patients with chronic craniofacial pain conditions and sleep disorders is based on a comprehensive, multidisciplinary team approach, addressing all the factors that modulate the pain experience and sleep disorders.
7. Treatment of the underlying sleep disorders may greatly improve craniofacial pain including headaches and therefore, re-evaluation of headache is advised following treatment of a sleep disorder. Treatment includes behavioral interventions as well as medications.

Research agenda

1. Research on the mechanisms underlying the bidirectional association between craniofacial pain and sleep disorders is essential.
2. Longitudinal human studies based on subjective self-reported measures and sleep diaries, as well as on objective measures, such as polysomnography and neuroimaging should be used.
3. There is a lack of data regarding the association between TMD and painful cranial neuropathies with sleep, which should be addressed in future studies.
4. Hybrid behavioral therapies that combine elements for pain and sleep may be a promising approach and should be further assessed among chronic craniofacial pain patients.
5. The efficacy of medications used to treat insomnia should be explored in the context of co-morbid craniofacial pain.

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Conflict of interest

The authors have no conflicts of interest to disclose.

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