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INVITED REVIEW

# Sleep disturbances in temporomandibular disorders: a narrative review

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## Abstract

Sleep complaints are frequently reported by patients with temporomandibular disorders (TMD). This review aims to offer dental practitioners and allied specialties a basic understanding of sleep quality, measure it subjectively and objectively and common sleep disturbances that are present in TMD patients. Guidance in identifying and managing patients with TMD and comorbid sleep-related complaints will be provided as well. Dentists should be able to screen sleep disorders such as insomnia, sleep disordered breathing (apnoea, snoring) or sleep bruxism, and to refer patients to the appropriate specialist when necessary. Individualised management and a multidisciplinary approach must be pursued when managing patients with TMD and comorbid sleep disturbances and/or sleep disorders, such as insomnia or obstructive sleep apnoea.

## Introduction

Patients with painful temporomandibular disorders (TMD) also present several sleep-related complains[1]. They may feel that their sleep is not restorative/recuperative as expressed by wake time tiredness, fatigue and lack of energy with an important impact on mood. Poor sleep in vulnerable subjects can contribute to the presence and maintenance of pain, while pain also may interfere with sleep onset or maintenance. Although the directionality of the sleep-pain relationship is still debated, both sleep and pain (including TMD) have important consequences in the individual's well-being and in the socioeconomic system[2].

The objective of this narrative review was to guide dentists in identifying and managing TMD patients with sleep-related complaints. Dentists need to screen sleep disorders such as insomnia, sleep disordered breathing (apnoea, snoring) and sleep bruxism. In some cases, dentists may also check for the impact of other conditions such as periodic limb movement syndrome. Possible management options in situations where sleep disturbances and TMD coexist will be described as well. Sleep bruxism will not be covered in this review, as it will be addressed in another article from this journal issue.

For more information about the impact of sleep on other orofacial pain disorders, a series of recent comprehensive reviews may also be consulted[3–5].

## What is pain

Pain has been defined by the International Association for the Study of Pain as 'an unpleasant sensory and emotional experience associated with actual or

potential tissue damage, or described in terms of such damage'[6]. When pain lasts beyond what is considered a normal expected healing period, it is categorised as chronic (often delimited as more than 3 or 6 months). It is to be noted that a consensus is not yet reached on the very accurate definition of pain, and other definitions such as the following: 'Pain is a distressing experience associated with actual or potential tissue damage with sensory, emotional, cognitive, and social components' have also been proposed[7].

Orofacial pain is a type of pain present in the face or in the oral cavity thus comprehending different painful disorders affecting the teeth, nerves and musculoskeletal structures. TMD is an umbrella term referring to different disorders that affect the temporomandibular joint (TMJ) and/or muscles of mastication[8]. Signs or symptoms of TMD include pain and tenderness in or around the ear, the jaw joint or the muscles of the jaw, face or temples. Other symptoms include mechanical problems when opening or closing the mouth, such as difficulty to open the mouth and clicking, popping, crunching or grinding noise when chewing, yawning or moving the jaw. Painful TMD can be broadly divided in articular, when the pain is originated in the TMJ, and muscular, when the origin of pain is in the muscles of mastication. However, a combination of both is quite frequent. After pain of dental origin, TMD are the most frequent orofacial pain disorders, affecting up to 12% of the general population[9].

The exact pathophysiological mechanisms of painful TMD are currently unclear, although it is thought to be a combination of peripheral and central mechanisms[10–13]. TMD frequently coexist with other painful trigeminal conditions such as migraine and tension type headache and with other extra-trigeminal ones such as fibromyalgia, being often categorised as one of the 'chronic overlapping pain conditions'[14–17]. Patients with chronic TMD usually present psychosocial issues, which include mood disorders or different sleep disturbances (Fig. 1)[18,19].


**Figure 1** Sleep disorders/issues that can be associated with temporomandibular disorders (TMD).

## What is sleep

Behaviourally, sleep is defined as the quiescence accompanied by closed eyes, recumbent posture, limited muscular activity and a reduced response to sensory stimuli, which can be easily reversed. Sleep can be divided into two main phases: non-rapid eye movement (NREM) and rapid eye movement (REM). Each of those is categorised by different behavioural, physiological, neurochemical and electrophysical attributes[20,21]. NREM sleep can be further subdivided into three different stages according to electroencephalogram (EEG) parameters, being stage N1 and N2 considered as 'light sleep' (lower arousal thresholds) and N3 (formerly 3 and 4, now merged in one stage) as 'deep sleep' (higher arousal thresholds and dominance of slow wave sleep). NREM sleep is associated with decreased mental and physical activity, heart rate, blood pressure and breathing frequency among others, which is stable across the stages[20,21]. Additionally, REM or 'paradoxical' sleep, which follows the NREM phase in the sleep cycle, is characterised by an increase of mental activity while muscles maintained paralysed or inactive. During REM phase, EEG patterns are more variable and physiological parameters such as heart rate, blood pressure or breathing frequency are more instable. It is in this phase where dreams more frequently occur[22].

In healthy adults, sleep onset usually occurs after 20–30 min of going to bed. A typical night of sleep encompasses 3–5 NREM to REM ultradian cycles (90 min on average), where lighter sleep turns into deep sleep, which is then followed by a REM phase, for a total of 6–9 average h of sleep. As the night advances, stage N 3 decreases, stage 2 becomes more predominant, and REM phases become longer[23]. During these cycles, there are frequent brief brain, heart and muscle reactivations or microarousals called 'windows' that last 3–15 s. Those occur approximately 5–15 times per h, allowing the individual to reposition or to recognise any harmful situation[24].

Sleep is an active process, it is not coma neither anaesthesia. During sleep, a cyclic alternating pattern (CAP) is present. CAP is a visualisation method and a physiological phenomenon that represents the

balance between sleep quietness and sleep arousal. The sleep arousal is part of a protective 'fight or flight' mechanism that remains present during NREM sleep to preserve body integrity (sleep positioning, adjustments in heart rate, etc.) and to react to threatening events. CAP is divided into two phases: active and quiet. The active (A) phase is further subdivided in A1 (high dominance of slow wave sleep, promotor of sleep restoration), in A2 (a transition phase) and A3 (an arousal dominant phase, essential for survival). The high rate of A2 and A3 phase represents sleep fragmentation. The quiet phase (B), is the one during which sleep is very stable and quiet[25]. CAP phases are in the minute's domains and their rate rise with age or in presence of different conditions. For example, more active phases are observed in fibromyalgia[26], and CAP has been associated to the onset of sleep bruxism[27].

### Sleep deprivation

Sleep deprivation, which can be briefly defined as lack of sleep or too short sleep, can occur due to bad habit, poor sleep hygiene, age or disorder/disease. It can also be induced experimentally by total prevention or partial restriction of sleep, done by retarding sleep onset or waking a subject in the night for a given period.

Sleep deprivation is present in around 20% of the general population and it may be associated to sleep disorders such as insomnia or sleep apnoea[28]. Studies have shown that sleep deprivation can affect memory, mood, physical activity, immune system, metabolism and increase the risk of motor vehicle accidents, cardiovascular diseases or dementia among others[29–31], and sleep disturbances are frequently associated with pain[32].

Importantly, the intake of alcohol and tobacco, two of the most commonly used psychoactive substances in the community, can disrupt sleep through different mechanisms. For example, it is thought that alcohol disrupts sleep architecture, triggers insomnia, contributes to abnormalities of circadian rhythms, and also increases breathing-related sleep events such as snoring and oxygen desaturation[33]. Additionally, tobacco smoking, probably due to nicotine, has been also associated with disrupted sleep architecture, and it is considered a risk factor for sleep disordered breathing[34,35]. Stimulant medications such as methylphenidate or amphetamine can led to longer sleep latency, worse sleep efficiency and shorter sleep duration[36], and opioids, which can be used to treat pain, may exacerbate sleep disturbances and increase sleep apnoea episodes[37].

### Sleep and pain interaction

In healthy individuals, sleep restriction has been shown to be associated with the development of clinical somatic pain-related complaints[38], and to produce hyperalgesic effects such as decreased pressure pain tolerance[39,40], heat pain thresholds[41] and withdrawal latency[42]. Sleep disturbances have also been associated with alterations in endogenous pain modulation, with sex specificity to be confirmed[43–45]. In fact, a recent study showed increased secondary hyperalgesia in males and significantly increased temporal summation in females, suggesting that different pain mechanism and pathways may be associated to sex[43].

The interaction between sleep and pain has been frequently reported, following a linear model in acute pain states and a bidirectional model in chronic ones. In that way, pain episodes can disrupt or alter sleep, and sleep disturbances can increase, predispose or perpetuate pain sensation. Although this bidirectional or circular interaction can be dominant in chronic pain cases[4], its directionality remains debated, as there is more evidence pointing towards poor sleep increasing pain and not the other way around[2].

Sleep complains are present in around 80% of chronic pain disorders[46], and reports of insomnia can be as high as 30–40% in patients with chronic pain[47]. Polysomnography (PSG) studies have reported the important coexistence of sleep disorders/disturbances in chronic pain populations[48], and sleep continuity parameters have also been identified as risk factors for clinical pain[49–51]. A recent systematic review and meta-analysis revealed that deterioration in sleep was associated with worse self-reported physical functioning (medium effect size) while improvement in sleep was associated with better physical functioning (small effect size)[52].

## Subjective sleep quality in TMD

Subjective sleep quality has been defined as tiredness on waking and throughout the day, feeling rested and restored on waking, and as the number of awakenings experienced in the night[53]. This component is usually measured through questionnaires, which are easily administered and cost-effective,

thus facilitating the feasibility of this measurement. In TMD and orofacial pain patients, up to eight questionnaires have been used to report subjective sleep quality. Some of the most commonly used ones include the Pittsburgh Sleep Quality Index (PSQI), the Epworth Sleepiness Scale (ESS), the Insomnia Severity Index and the Sleep Assessment Questionnaire[54].

The PSQI, a questionnaire formed by 18 items measuring general sleep quality in the last month, is the most commonly used tool to measure sleep quality[55]. The PSQI is a valid and reliable instrument that has been employed in different clinical and non-clinical populations[56]. It has seven different components, which measure different aspects of sleep such as overall sleep quality, sleep onset latency, sleep duration, sleep efficiency, sleep disturbances, use of sleep medication and physical dysfunction. A cut-off of> 5 is used to differentiate poor sleepers, with a sensitivity of 89.6% and specificity of 86.5%.

When compared to pain-free controls, TMD patients exhibited poorer sleep quality and were mainly categorised as poor sleepers, with mean scores ranging from 6.69 to 13.7 in different TMD populations[57–65]. These scores and findings are similar than the ones obtained in other painful conditions such as fibromyalgia or rheumatoid arthritis[66,67]. In addition, poorer sleep quality has been positively correlated with clinical pain intensity and psychological distress in TMD patients[57]. In the Orofacial Pain Prospective Risk Assessment (OPPERA) study, which is the largest study designed to identify risk factors for TMD in pain-free participants (n = 2453 filled PSQI), TMD incidence was twice as high in participants whose baseline subjective sleep quality was poor (demographically adjusted Hazard Ratio = 2.04; 95% CI, 1.55–2.70)[68]. In addition, tertiary analysis of OPPERA data showed that subtle sleep quality impairments were noted on PSQI beforehand in participants developing TMD (n = 220)[69,70].

Additionally, it is important to highlight fatigue, which is an important marker of poor sleep or non-recuperative process, in patients with sleep disturbances. Fatigue is a frequent complaint in chronic pain and in TMD patients that deserve attention in its diagnosis and management[71,72]. Only a few tools are currently available to assess the presence of fatigue, and it is important that clinicians differentiate fatigue as a general somatic and behavioural complaint from chronic fatigue syndrome in TMD patients[73,74].

## Objective sleep quality in TMD

Objectively, sleep quality can be defined as sufficient duration, high efficiency and low fragmentation (i.e., not too many stage shifts, body movements, breathing disturbances, drop in oxygen), as well as proper staging of sleep, which are measured through PSG[75].

PSG evaluation comprehends a series of biophysiological changes, including EEG, eye movements, muscle activity, heart rhythm and respiratory function[75]. PSG, which is the gold standard to diagnose sleep disorders such as sleep disordered breathing or periodic limb movement, involves costlier equipment and resources, including overnight stay of the person being studied when it is done in the sleep laboratory (type 1 recording), therefore limiting its research applicability. PSG is not an ideal perfect method since the sleep laboratory is not a 'natural milieu' and the electrode montage may disrupt sleep. Additionally, it should be considered that the obtained information represents a 'snapshot' or a one-night picture. An alternative method is the use of portable monitor devices or home recording, (multi channels down to one channel; type 2 to 4 respectively). The major gain of this modality is that patients sleep in their natural environment, allowing as well to record multiple nights in order to improve the precision of the test. Importantly, subjective sleep quality usually correlates poorly with objective sleep quality parameters, suggesting that each of them could measure different dimension of sleep[76].

There are different studies investigating objective parameters of sleep quality in different populations of TMD patients (Table 1). Some of them did not find any difference in sleep parameters between TMD patients and pain-free controls, although the relatively small sample and the somewhat restrictive inclusion criteria calls for caution in the interpretation of their results[77,78]. Other studies using uncontrolled samples also found sleep characteristics within normal range[79–81], yet one of them reported a positive significant correlation between sleep efficiency and endogenous pain inhibitory function measured by a conditioned pain modulation paradigm[79].

The largest study using PSG in TMD patients to date, revealed interesting differences between 124 females with myofascial TMD and 64 matched pain-free controls who underwent two nights of PSG recording[82–84]. In a series of reports, the authors pointed three main findings. In regards sleep parameters, the authors found that respiratory effort related arousals (RERA), which are arousals

**Table 1** Studies evaluating objective sleep parameters in TMD patients.

| Study | Sample | Objective | Method | Main findings |
|---|---|---|---|---|
| Camparis et al.[77] | 20 with SB and MFP20 with SB without MFP | To compare bruxism and sleep parameters | PSG, 1 night | No differences in bruxism episodes and sleep variables |
| Rossetti et al.[78] | 30 MFP30 matched controls | To evaluate association between RMMA-SB and MFP | PSG, 1 night | Significant associations were observed between RMMA-SB and MFP, as well as between daily clenching (self-report) and MFP |
| Smith et al.[80] | 53 MFP | To characterise sleep disorder rates in TMD and evaluate possible associations between sleep disorders and laboratory measures of pain sensitivity | PSG, 2 consecutive nights | 17% presented sleep bruxism, 36% insomnia disorder and 28.4% sleep apnoea |
| Edwards et al.[79] | 53 MFP (same sample as above) | To assess whether individual differences in sleep continuity and/or architecture were related to diffuse noxious inhibitory controls (DNIC) | PSG, 2 consecutive nights | Higher sleep efficiency and longer total sleep time were positively associated with higher conditioned pain modulation |
| Dubrovsky et al.[84] | 124 MFP females 46 matched controls | To evaluate measures of sleep and respiratory disturbance in a large representative sample of TMD cases in comparison with matched controls | PSG, 2 consecutive nights | TMD cases presented significant increase in stage N1 sleep, mild but significant elevations in arousals associated with all types of respiratory events and in RERAs when compared to controls. MFP predicted a lower sleep efficiency, more frequent awakenings, and higher RERA index among TMD cases |
| De Siqueira et al.[81] | 10 SB/MFP patients with widespread pain10 SB/ MFP patients without widespread pain | To investigate whether the presence of concomitant widespread pain could influence sleep characteristics of patients with SB and MFP | PSG, 1 night | Group with widespread pain presented lower sleep efficiency |

TMD, temporomandibular disorders; MFP, myofascial pain; SB, sleep bruxism; RMMA, rhythmic masticatory muscle activity; RERA, respiratory effort related arousal.

from sleep without concomitant oxygen desaturations that do not technically meet the definitions of apnoeas or hypopnoeas but do disrupt sleep, were significantly increased in TMD patients and also statistically independent from sleep fragmentation measures. These findings led the authors to speculate about the possible existence of an upper airway resistance in their sample, which has also been proposed in other pain conditions such as fibromyalgia[85,86]. Other findings include the presence of higher N1 sleep stage, a tendency towards a great number of awakenings, and greater number of stage N1 shifts in TMD patients when compared to controls, thus showing more sleep instability[84]. They also found that more awakenings and less sleep efficiency was associated with more pain intensity during the following day, and that poor sleep quality reports were better attributed to depressive symptoms rather than pain intensity or objective sleep quality measures obtained through PSG[87].

Additionally, the authors also reported that sleep background electromyography (EMG), defined as masseter muscle EMG activity occurring outside of sleep bruxism or other defined motor event periods, was significantly higher in TMD cases when compared to controls and also positively associated with higher levels of clinical pain intensity[83]. They hypothesise that elevated sleep background EMG

may lead to fatigue and pain, suggesting it as a possible risk factor for pain maintenance in TMD. Relatively similar findings were also observed in other study using portable EMG devices during several nights[62].

Although this study can be considered as a reference in sleep and TMD, caution in the representability of these results is warranted, as the selected sample is very specific, and the primary objective of the study was different[82–84].

In summary, PSG studies highlight the presence of sleep disorders and the importance of sleep fragmentation, disruptions, RERAs, and also background muscular activity in TMD patients. However, caution in the interpretation of these results is warranted due to the small and/or restrictive samples.

## Insomnia and TMD

Insomnia is a disorder defined as difficulty to initiate or maintain sleep, when sleep onset does not occur after 30 min for at least three times per week for three or more months, or if spontaneous awakenings occur during the night without the ability to resume sleep[88]. The presence of insomnia can cause fatigue, lack of attention, mood alterations and gastrointestinal symptoms among others, being also considered a risk factor for coronary heart disease and depression[89,90].

Insomnia occurs in approximately 10% of the general population[91] and up to 72% of people with chronic pain[48]. It has been shown that pain can increase the risk of insomnia and vice versa[92,93], and that insomnia is associated with a reduction in pain tolerance in patients with chronic pain[40]. A recent study showed that 1/3 of patients seeking care at an orofacial pain unit (mixed orofacial pain conditions, including TMD) presented sleep disturbances, where 37% of the studied patients (352 out of 952) responded positively to a screening question for insomnia and/or hypersomnia, being the majority of them categorised as moderate to severe insomnia[1]. This is in line with findings from a prior PSG study performed in TMD patients (n = 53), which revealed that 36% of them suffered from insomnia, being 26% of those cases primary insomnia (not attributable to a medical, psychiatric or environmental cause)[80]. Moreover, it was also shown that primary insomnia diagnosis was associated with reduced mechanical and thermal thresholds in the masseter muscles and in the forearm in TMD patients[80], and that increases in the severity of symptoms of insomnia are prospectively associated with next month daily increases of pain[94]. These findings suggest that insomnia, which is a common disorder in chronic pain and orofacial pain populations, is an important and prevalent comorbidity in TMD and can have an influence in pain and affect importantly patient's quality of life. Therefore, insomnia should be suspected and screened in patients suffering from TMD.

## Sleep breathing disorders and TMD

Sleep-related breathing disorders is another term used to describe several chronic conditions in which partial or complete cessation of breathing occurs many times throughout the night, mainly resulting in fatigue or daytime sleepiness, which interferes with a person's ability to function. Symptoms may include snoring, pauses in breathing described by bed partners and disturbed sleep[95]. The most common sleep breathing disorder is obstructive sleep apnoea (OSA), which is defined clinically by the presence of at last five respiratory events per h of sleep (including apnoeas, hypopnoeas and respiratory effort related arousals) accompanied by daytime sleepiness, loud snoring, witnessed breathing pauses, or awakenings due to gasping or by the presence of at least 15 respiratory events without the presence of signs and symptoms[88,96]. Although the respiratory disturbance index (RDI) may be more comprehensive, the main metric for the diagnosis of OSA is still the apnoea hypopnoea index (AHI). OSA prevalence ranges from 9% to 38% when an AHI of 5 is used as a threshold, being more frequent in men and in obese people, and increasing considerably with age[95,97]. The presence of untreated OSA has been related with higher risk of cardiovascular diseases, metabolic syndrome or diabetes among others[98].

Obstructive sleep apnoea is estimated to be a common comorbidity in general chronic pain populations, with a pooled prevalence of up to 37%, and also in TMD where its frequency has been recognised as 28.6%[80]. In the OPPERA study, high likelihood of OSA has been associated with the incidence of first onset TMD in the prospective cohort study (n = 2604, adjusted hazard ratio = 1.73; 95% CI, 1.14, 2.62), and also with chronic TMD in the case-control one (n = 1716, adjusted odds ratio = 3.63; 95% CI, 2.03, 6.52)[68]. In addition and as described above, TMD females present more RERAs than healthy controls[84], which may predispose them to develop OSA.

Another sleep-related breathing disorder is upper airway resistance syndrome (UARS), which is less prevalent than OSA and is characterised by episodes

of increased RERAs and sleep disruption but without meeting apnoea/hypopnoea definition[99]. As OSA, UARS can cause fatigue, daytime sleepiness and sleep fragmentation[99], and it has been associated with the presence of other conditions such as irritable bowel syndrome, insomnia or depression[100]. Despite that UARS can be considered different from OSA, its acceptance as a different syndrome remains controversial. However, it appears that UARS has a bigger impact on subjective sleep quality and fatigue compared to mild OSA[101], the reason why its presence needs to be investigated in TMD patients.

## Screening of sleep disorders in TMD

Given the prevalence of sleep disorders and their association with negative pain-related outcomes, the screening of sleep disorders and the identification of subjective sleep disturbances is pivotal to the management of patients with TMD. Recently, an algorithm based on clinical interview and a physical evaluation was proposed to identify sleep disorders in chronic orofacial pain patients[3,5].

During the clinical anamnesis, the importance of evaluating sleep complains (problems in sleep initiation and/or maintenance, sleepiness during the day, snoring, witnessed apnoeas, etc.), comorbidities and lifestyle habits (diagnosed sleep disorders, caffeine or tobacco intake, exacerbating or initiating factors, etc.) and sleep routine (sleep environment, bedtime routine, etc.) are highlighted and warranted.

During the physical exam, extraoral examination looking for signs of daytime sleepiness (droopy eyelids, repetitive yawning, irritability, etc.) or risk factors for OSA (increased neck circumference, nasal examination, retrognathia/micrognathia, etc.) should be also accompanied by an intraoral exam assessing other risk factors for OSA (soft palate, uvula, Mallampati score, tongue, nose obstruction-deviation, etc.) and by a systemic diagnostic work-up evaluating blood pressure, heart rate or body mass index.

The identification of a possible sleep disorder by the dentist should be followed by a referral to a sleep physician for evaluation and possible PSG assessment.

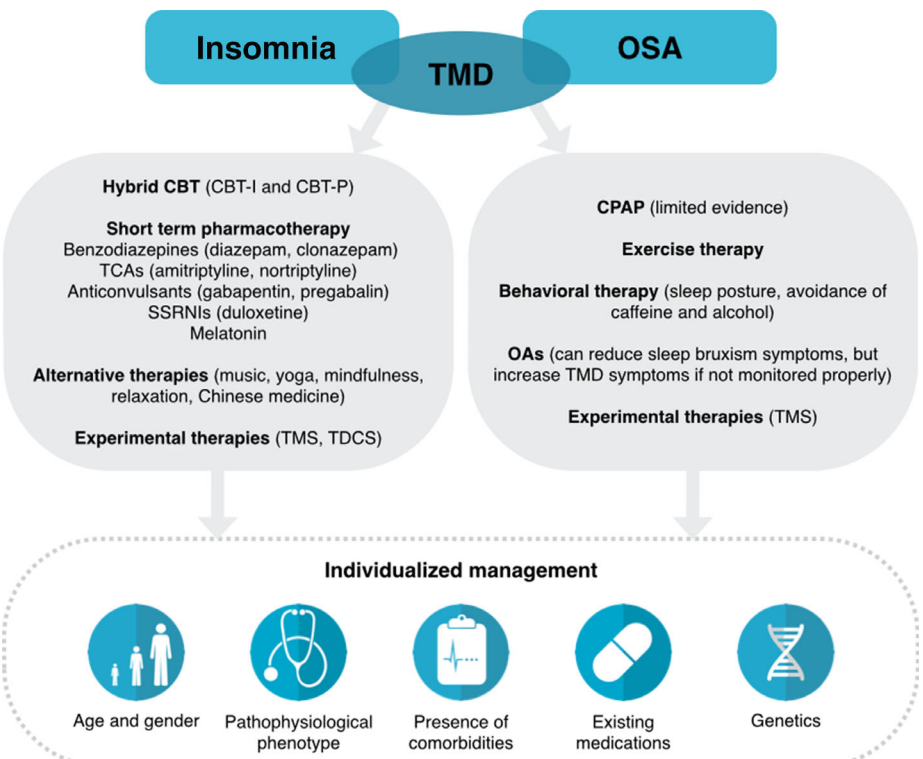


**Figure 2** Management avenues for primary insomnia and obstrutive sleep apnoea (OSA) concomitant to temporomandibular disorders (TMD). CBT: cognitive behavioural therapy; CBT-I: cognitive behavioural therapy for insomnia; CBT-P: cognitive behavioural therapy for pain; TCAs: tryciclic antidepressants; SNRIs: serotonin and norepinephrine reuptake inhibitors; TMS: transcranial magentic stimulation; tDCS: transcranial direct current stimulation; CPAP: continuous positive airway pressure; OAs: oral appliances.

## Management of sleep disorders in TMD

When treating sleep disorders in chronic pain, patients can benefit from strategies targeting the sleep disorder or also from treatments that can help to manage both sleep and pain. Ideally, an individualised management approach should be implemented considering several aspects and characteristics of the individual, such as morphology, comorbidities, existing treatments and medications or genetics (Fig. 2).

### Insomnia

Currently, the first treatment option for the management of chronic insomnia is cognitive behavioural therapy focused on insomnia (CBT-I). CBT-I is a structured program that helps to identify and replace thoughts and behaviours that cause or worsen sleep problems with habits that promote sleep, usually including sleep hygiene, sleep restriction and relaxation[102]. CBT-I has been shown to be effective in the long-term for different sleep parameters such as sleep onset or efficiency, and also in a small manner for pain outcomes[103] also in TMD[104]. CBT can also be focused on pain (CBT-P), and hybrid models of CBT for both sleep and pain have been proposed as a synergic management plan[105,106]. CBT is a safe, non-invasive modality with good long-term outcomes, but it can suppose a high initial cost requiring as well as high level of compliance, which limits its implementation.

Another approach, especially for short-term periods of time, is the use of pharmacological interventions. Their effects are usually immediate and can have lower cost than CBT, but may be accompanied by side effects, drug-drug interactions, short-term efficacy and a risk of addiction[3]. Medications that can be used to treat sleep but with less evidence of their effects on pain are the atypical antidepressant trazodone, the hypnotic agent zolpidem or the orexin receptor antagonist suvorexant[107–109]. Evidence indicates that benzodiazepines such as diazepam or clonazepam, can be beneficial for improving sleep and reducing pain outcomes in chronic pain populations including TMD[110]. The use of tricyclic antidepressants (TCAs) in low doses such as amitriptyline or nortriptyline has shown to be effective in reducing pain levels in TMD[111] and also in improving sleep quality in different chronic pain populations[112,113]. Anticonvulsant medications such as pregabalin and gabapentin may also be good options for managing pain and sleep[113,114] in chronic pain and in TMD. Other medications that may be effective in improving sleep and pain in TMD are the muscle relaxant cyclobenzaprine[58], melatonin[115] and the selective serotonin and norepinephrine reuptake inhibitors (SSRNI) duloxetine[113,116]. In general, opioid should be avoided as much as possible, due to the risk of addiction, central sleep apnoea and deleterious effect on insomnia[117,118].

Combination of CBT-I and short-term pharmacotherapy, hypnosis, physical exercise, music therapy, yoga, mindfulness or traditional Chinese medicine are other alternatives to improve pain and sleep disturbances in chronic pain[3,102]. Other alternative strategies may include the use of non-invasive neurostimulation techniques such as transcranial magnetic stimulation (TMS) and transcranial direct current stimulation, which have shown promising results in chronic insomnia as a main treatment and as an adjuvant therapy, and also in chronic pain including orofacial pain conditions[119–122].

### Sleep breathing disorders

Depending on the AHI, OSA's severity can be classified into mild (AHI> 5 but < 15), moderate (AHI> 15 but < 30) and severe (AHI> 30). For all of them, the gold standard treatment is the continuous positive airways pressure device (CPAP), which has been proved to reduce AHI as well as morbidity and mortality associated with it[123,124]. There is also less evidence about CPAP efficacy reducing experimental pain sensitivity, perhaps as an indirect improvement of OSA. However, this was not the case in chronic pain populations[125,126]. Adherence to the CPAP treatment remains challenging, and in these cases, other treatment options are available[127].

In a recent meta-analysis, exercise therapy (general exercise to oropharyngeal programs) was found to be the second most effective treatment option in reducing AHI. These findings highlight the use of exercise therapy not only as an adjuvant therapy but also as a first option in indicated cases[124]. Moreover, physical exercise can improve sleep quality and pain-related outcomes in chronic pain populations, thus becoming a very relevant option in TMD patients with sleep disturbances[128,129].

Oral appliances (OA) are indicated in mild and moderate cases of OSA and in cases where patients are unable to tolerate CPAP. OAs also become a good option in cases where OSA and sleep bruxism (SB) coexist, as it can also help to decrease bruxism effects. As OA work moving the jaw forward in order to open the airway, its use can produce jaw discomfort, bite changes and sometimes aggravate

TMD cases[130]. Therefore, the monitoring of TMD symptoms and the performance of more conservative advancements is preferable in these cases. Nevertheless, they are considered as a good alternative to CPAP, having higher adherence and being preferred by OSA patients[131].

Other treatment alternatives include other behavioural therapies such as weight loss, positional therapy or alcohol avoidance, adjunctive therapies such as bariatric surgery or medications, surgical options such as orthognathic surgery and uvulopalatopharyngoplasty and more experimental ones such as upper airways stimulation or TMS[127,132].

Additional consideration in patients where SB (frequently comorbid with TMD) coexists with OSA, is the election of oral devices to manage SB. Empirically, it appears that mandibular flat planes in maximal intercuspation rather than in a retruded position may be a reasonable alternative do diminish the risk of airway obstruction.

Moreover, the dentist should know that a personalised management of OSA, individualising treatments according to different phenotypes/traits is now considered the best management strategy for these patients[133,134]. A multidisciplinary approach where dentists collaborate with physicians, health psychologists and physical therapists among others emerges as an ideal approach to manage TMD patients OSA and also with sleep disturbances.

## Conclusions

Sleep disturbances are present in TMD patients when measured subjectively and objectively. Dentists need to screen for behavioural and somatic sleep-related problems (e.g., sleepiness, fatigue, mood instability) that may guide them to identify sleep disorders such as insomnia, sleep apnoea and sleep bruxism. In the presence of a suspected sleep disorder, collaboration with a sleep physician is mandatory. Individualised management and a multidisciplinary approach must be pursued when managing patients with TMD and comorbid sleep disturbances and/or sleep disorders, such as insomnia or OSA.

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