Curr Neurol Neurosci Rep (2015) 15:520
DOI 10.1007/s11910-014-0520-2

SLEEP (M THORPY, M BILLIARD, SECTION EDITORS)

Tension-Type Headache and Sleep

Jeanetta C. Rains · Rachel E. Davis · Todd A. Smitherman

Published online: 5 December 2014

Abstract This review describes empirical evidence for a bidirectional relationship between tension-type headache (TTH) and sleep. In its most severe form, chronic TTH (CTTH) affects 2–3 % of the population and can be very disabling. Sleep dysregulation triggers episodic TTH, and sleep disorders may complicate and exacerbate headache. The majority of CTTH sufferers also have insomnia, and longitudinal data suggest that insomnia is a risk factor for new-onset TTH. Similarly, observational studies suggest that sleep disturbance is a risk factor for new-onset TTH and for progression from episodic to chronic TTH (i.e., headache "chronification"). CTTH is the most common headache secondary to sleep apnea and other sleep-related breathing disorders. Psychiatric disorders are comorbid with both TTH and insomnia and may further complicate diagnosis and treatment. Developments in diagnostic classification of sleep-related headache are presented.

Keywords Tension-type headache · Headache · Awakening headache · Morning headache · Sleep-related headache · Sleep apnea headache · Snoring · Insomnia · Psychiatric comorbidity · Chronification

This article is part of the Topical Collection on *Sleep*

J. C. Rains (✉)
Center for Sleep Evaluation at Elliot Hospital—River's Edge,
185 Queen City Avenue, Manchester, NH 03101, USA
e-mail: jrains@elliot-hs.org

R. E. Davis · T. A. Smitherman
Department of Psychology, University of Mississippi,
207 Peabody, University, MS 38677, USA

R. E. Davis
e-mail: redavis2@go.olemiss.edu

T. A. Smitherman
e-mail: tasmithe@olemiss.edu

Introduction

Tension-type headache (TTH) is the most common headache and, indeed, pain condition worldwide, but TTH historically has been a relatively neglected subject of research. TTH ranges from infrequent, mild variants to a chronic, severe, and disabling condition. TTH has received much less attention than its migraine counterpart, although the two often co-occur, especially in more severe and complicated headache presentations. Likely, the relative neglect of TTH stems from the historical view of TTH as psychogenic and perhaps also from the fact that milder forms are almost ubiquitous and relatively innocuous. Though less is known about the pathophysiology of TTH compared to migraine, the past decade has yielded important diagnostic, epidemiological, observational, clinical, and experimental evidence advancing our understanding of TTH and interrelated sleep disorders and processes.

The association between sleep and headache has been recognized in the medical literature for over a century and has been described extensively in earlier literature reviews [1, 2•, 3, 4]. Earlier studies often lacked diagnostic precision, frequently reporting on headaches proximally related to sleep (e.g., morning headache, awakening headache) rather than formal headache diagnoses. Diagnostic precision has improved in recent studies and may be facilitated by new developments in classification. The diagnostic manual for sleep disorders recently appended the manual with a section for sleep-related headaches. Thus, as described below, classification of sleep-related headache is now recognized to some extent in the diagnostic manuals for both headache and sleep.

TTH is the most common headache and the most common headache symptom pattern associated with sleep disorders [4]. Empirical research discussed below demonstrates the bidirectional relationship between sleep and headache. Sleep loss is an acute "headache trigger" confirmed in prospective time series analysis, and going to sleep is one of the most frequent

and effective self-management responses to headache. Interestingly, a recent biobehavioral model has proposed that short-term headache relief of sleeping may ultimately undermine basic processes involved in sleep-wake regulation. Sleep dysregulation has been shown to lower pain threshold in the pain literature, a finding consistent with literature on sleep-related headache triggers. Sleep dysregulation, then, may increase propensity to headache and further sleep problems in a vicious cycle.

Classification, Epidemiology, and Impact

Tension-Type Headache

Headache diagnosis follows the International Classification of Headache Disorders, 3rd edition, beta version (ICHD-3β) [5], which was released to facilitate integration with the forthcoming 11th edition of the International Classification of Diseases (ICD-11) by the World Health Organization.

TTH is distinguished from migraine by its bilateral location, mild-to-moderate pain intensity, and non-pulsatile (e.g., pressing, tightening, or band-like) pain characteristics (Table 1). Unlike migraine, TTH is not exacerbated by physical activity and not associated with significant nausea or vomiting, although mild nausea, photophobia, or phonophobia may be present. TTH is subclassified by frequency and the presence or absence of tenderness of the pericranial muscles. Episodic TTH (ETTH) episodes last 30 min to 7 days and may be characterized as infrequent (<1 day per month or <12 days per year) or frequent (1–14 days per month or <180 days per year). Chronic TTH (CTTH) includes high-frequency headache attacks (>15 days per month or >180 days per year) that, in some cases, may be unremitting.

TTH Prevalence and Impact Tension-type headache is the second most prevalent medical condition in the world [6]. A recent review of global headache prevalence concluded that 1-year prevalence of episodic TTH is 42 % among adults, with 2–3 % of adults experiencing chronic TTH [7]. Among those with ETTH, 32 % report experiencing headache several times a month and 10 % report experiencing TTH weekly [8]. Tension-type headache is slightly more common among women than men, and prevalence peaks between ages 30 and 39 [9].

Tension-type headache is associated with numerous negative consequences including significant reductions in quality of life, decreased work performance, and increased healthcare costs both to the individual and society. Because it is much more prevalent than migraine, the societal burden resulting from disability caused by TTH is greater than that caused by migraine [7]. Specifically, the cost of medical services and medications are 54 % higher for TTH than migraine, and TTH results in three times more missed workdays across the population than migraine [9].

Table 1 International Headache Society diagnostic criteria for chronic tension-type headache and sleep apnea headache [5]

Chronic tension-type headache[a]

- A. Headache occurring on ≥15 days per month on average for >3 months (≥180 days per year), fulfilling criteria B–D
- B. Lasting hours to days or unremitting
- C. At least two of the following four characteristics:
  1. Bilateral location
  2. Pressing or tightening (non-pulsating) quality
  3. Mild or moderate intensity
  4. Not aggravated by routine physical activity such as walking or climbing stairs
- D. Both of the following:
  1. No more than one of photophobia, phonophobia, or mild nausea
  2. Neither moderate or severe nausea nor vomiting
- E. Not better accounted for by another ICHD-3 diagnosis

Sleep apnea headache

- A. Headache present on awakening after sleep and fulfilling criterion C
- B. Sleep apnea (Apnea-Hypopnea Index ≥5) has been diagnosed
- C. Evidence of causation demonstrated by at least two of the following:
  1. Headache has developed in temporal relation to the onset of sleep apnea
  2. Either or both of the following:
     a. Headache has worsened in parallel with worsening of sleep apnea
     b. Headache has significantly improved or remitted in parallel with improvement in or resolution of sleep apnea
  3. Headache has at least one of the following three characteristics:
     a. Recurs on >15 days per month
     b. All of the following:
        (i) Bilateral location
        (ii) Pressing quality
        (iii) Not accompanied by nausea, photophobia, or phonophobia
     c. Resolves within 4 h
- D. Not better accounted for by another ICHD-3 diagnosis

[a] Criteria A (headache frequency) and B (headache duration) vary by diagnosis for infrequent ETTH, frequent ETTH, and CTTH. Each diagnosis may be subclassified by the presence or absence of pericranial tenderness. "Probable" is added to the diagnosis when all criteria but one are met

When TTH Is Preempted by Other Headache Diagnoses Some TTH patterns are better characterized by other primary headache diagnoses, depending on historical features and precipitating factors [5]. Examples include the following: CTTH that emerges abruptly or de novo is diagnosed as new daily-persistent headache, and frequent headaches resembling TTH that occur exclusively during sleep (and usually among older adults) are diagnosed as hypnic

headache. Secondary headaches are those attributable to another condition such as overuse of acute headache medications (*medication overuse headache*), trauma to the head or neck (*posttraumatic headache*), or another medical problem [e.g., infection, other cranial or cervical disorders, substance use, psychiatric disorders (in very rare cases)]. Headache secondary to sleep apnea is most relevant to this review.

Sleep Apnea Headache

Across studies of patients with obstructive sleep apnea (OSA), the prevalence of any headache ranges from 18 to 60 % (across 12 studies) and that of morning headache from 11 to 58 % (5 studies) [10]. Sleep apnea headache (SAH) is defined by proximity of headache to sleep in the context of a positive diagnosis for sleep apnea (Table 1) [5]. Headache is present in the morning upon awakening from sleep. Pain characteristics usually resemble those of TTH. Alberti et al. [11] demonstrated that SAH presents commonly as bilateral (53 %) or unilateral (47 %); frontal (33 %), frontotemporal (28 %), or temporal in location (16 %); usually with pressing or tightening pain (79 %); and mild (47 %) or moderate intensity (37 %) but can be severe (16 %). However, unlike ETTH and CTTH, SAH predictably remits within 4 h. Successful resolution of sleep apnea resolves these headaches. When SAH represents an exacerbation of preexisting headache (e.g., tension-type headache, migraine, cluster), both primary and secondary headache diagnoses would be conferred.

Mechanisms of Sleep Apnea Headache Whether headache related to sleep apnea [considered a secondary headache attributable to a disorder of homeostasis (ICHD-3β)] is a function of "hypoxia, hypercapnia, or disturbance in sleep" [5, p. 751] has been a source of considerable debate. In their review of the literature, Provini et al. [10] concluded that it was unclear whether the mechanisms underlying SAH were hypoxemia or hypercapnia or, instead, another non-respiratory consequence of sleep apnea (i.e., sleep disturbance, autonomic arousal, cervical/cranial muscle tension, intracranial cerebrospinal fluid pressure changes). Perhaps, the most compelling research supporting metabolic mechanisms of SAH was polysomnographic research by Loh et al. [12], who observed a dose-response relationship between apnea severity (e.g., number of apneic events, degree of oxygen desaturation) and morning headache severity. Contradicting studies found that hypoxemia or other respiratory measures did not account for SAH [13–16]. Finally, evidence supporting the importance of both sides came from Alberti et al. [11], who compared a series of apneic versus insomniac patients; roughly half of patients in each group exhibited awakening headache, although likelihood of awakening headache was greater in apneic patients, particularly those with more severe sleep apnea.

Definitive evidence has recently confirmed that nocturnal intermittent hypoxemia is an independent risk factor for morning headache as well as other pain. Doufas et al. [17•] utilized data from the Cleveland Family Study, the largest longitudinal cohort study to date, to examine spontaneous pain reports in association with recurrent nocturnal hypoxemia versus sleep. Nocturnal hypoxemia was associated with an increased risk for headache and chest pain, independent of sleep fragmentation, and the relationship was maintained after controlling for systemic inflammation. When comparing the lowest quartile of average minimum oxygen saturation (<75 %) with the highest quartile (>92 %), the odds ratios (ORs) for self-reported morning headache (OR=2.08; 1.21–3.69), headache disrupting sleep (OR=1.83; 1.26–2.73), and chest pain in bed (OR=2.15; 1.27–3.62) are approximately doubled.

Other Forms of Sleep-Related Headache

Sleep-related headaches were recently included in the International Classification of Sleep Disorders, 3rd edition (ICSD-3) [18•]. Under "sleep-related medical and neurological disorders," the five-page appendix describes the association of several headache diagnoses including migraine, cluster, and hypnic headache. Although this addition represents a significant step forward in acknowledging the relationship between sleep and headache disorders, the most common sleep-related headache—TTH—was not addressed. Likewise, ICSD-3 includes no diagnosis for sleep apnea headache; only the narrow and highly specific diagnosis "central sleep apnea due to high altitude periodic breathing" lists "awakening headache" among the symptoms, although awakening headache is an empirically supported diagnostic symptom for obstructive sleep apnea and other sleep-related breathing disorders. Perhaps, further research and refinement of diagnosis of sleep-related headaches will follow.

Evidence of a Bidirectional Relationship Between Sleep and TTH

Sleep-Related Headache Triggers

In TTH (as well as migraine), sleep dysregulation (e.g., sleep disturbance, sleep loss, oversleeping) is a frequently reported trigger of acute headache attacks. In a cross-sectional study of 1219 patients in a neurological clinic in China, Wang et al. [19] reported that the most common headache triggers for patients with TTH were negative affect (32 %) and sleep disturbance (29 %). A literature review identified *lack of sleep* as a trigger in 26 to 72 % of TTH patients (two studies) and *oversleeping* as a trigger in 13 % (one study) [20]. Other common triggers include menstruation and fasting. Among adolescents, the most common headache triggers were stress

(42 %) and several sleep-related variables including tiredness (72 %), too little sleep (42 %), and too much sleep (10 %) [21].

Time Series Analysis Confirms that Sleep Triggers Headache Sleep-related triggers of TTH were confirmed prospectively using individual time series analysis. Houle et al. [22•] examined the relationship between daily stress, sleep duration, and headache (sum of headache severity four times daily) in patients diagnosed with TTH (n=22) who completed 28 days of self-monitoring. The relationship between sleep and headache intensity was non-linear, with both extremes of the sleep period distribution (short=<6 h; long=>8.5 h) associated with increased headache intensity. The lowest headache activity was associated with traditional sleep periods (7–8 h). An interaction with stress was found, even among those with near-daily headache, suggesting that sleep may be a moderating variable in the stress-headache relationship. Headache activity was highest when high stress and low-duration sleep occurred in conjunction over a 2-day period. These data suggest indirectly that consistent sleep patterns may provide a buffer against stress, supporting the importance of sleep regulation in headache management, especially during periods of stress.

Actigraphy Confirms that Oversleeping Triggers Headache Kikuchi et al. [23] examined sleep parameters objectively using actigraphy over a 7-day period and collected headache intensity ratings provided at 6-h intervals (four times daily) in 27 TTH patients, most of whom were female (74 %) and had CTTH (74 %). Headache intensity was recorded on electronic diaries in a watch-like microcomputer. Sleeping for longer than usual duration, termed by authors as oversleeping, was associated with more intense headache, although insufficient sleep, which earlier studies identified as a frequent headache trigger, was not associated with subsequent headache. Potentially, a longer assessment period, such as the 28-day interval used by Houle et al. [22•] and recommended by guidelines for headache research [24], would have increased power to identify an effect for sleep loss.

Headache Prompts Sleeping, Napping, or Resting

Headache also triggers sleep disturbance. Prospective research reviewed above confirms that sleep dysregulation may precede and precipitate headache, but headache can also beget dysregulated sleep. Headache sufferers often sleep, nap, or rest as a palliative response to pain. Haque et al. [25] examined headache triggers and relieving factors in 500 patients presenting to a neurological practice for treatment, half with TTH and half with migraine. Sleeping (52 % TTH and 58 % migraine) and medication taking (50 % TTH and 61 % migraine) were the most commonly endorsed means of headache relief. Stress was identified as the most common trigger for TTH. Migraineurs were more likely than TTH patients to endorse fatigue (13 vs. 7 %) and sleep deprivation (20 vs. 14 %) as a headache trigger. Similarly, Ong and colleagues [26] demonstrated in a young non-clinical sample that those with TTH strongly endorsed sleep problems (56 %) and stress (94 %) as headache triggers and sleeping (81 %) and medication taking (75 %) as the most common responses to pain. Longitudinal studies are needed to clarify the impact of headache on future sleep cycles.

Sleep Disorder Linked to More Frequent/Severe Headache

Population Studies Observational studies demonstrate a compelling relationship between chronic headache and sleep disturbance, especially insomnia. Using the Nord-Trøndelag Health Study (HUNT)-3 data described above, a cross-sectional analysis of 297 individuals found that severe sleep disturbance was threefold more likely to occur among those with TTH (n=135) and fivefold more likely to occur among those with migraine (n=51) than those without headache [27]. Those with chronic headache were 17-fold more likely to have severe sleep disturbance [28•].

In a UK cross-sectional study, Boardman et al. [29] identified a relationship between headache severity and sleep (i.e., trouble falling asleep, waking up several times, trouble staying asleep, or waking after a usual amount of sleep feeling tired or worn out). Among 2662 respondents, headache frequency was associated with slight (age/gender adjusted OR=2.4; 1.7–3.2), moderate (OR=3.6; 2.6–5.0), and severe (OR=7.5; 4.2–13.4) sleep complaints. Likewise, a European study using 18,980 telephone interviews found that "chronic morning headache" was associated with increased rates of insomnia (OR=2.14; 1.79–2.57) [30].

In a cross-sectional study of 1000 Copenhagen residents, Rasmussen et al. [31] found that *sleep problems* were more common in those with TTH than migraine in the general population; waking non-refreshed was more common among women with TTH than migraine in the population. The 12-year follow-up survey of the Lyngberg et al. cohort [8] determined that sleep problems were associated with a poorer headache outcome (i.e., at least 180 headache days per year at follow-up due to increased frequency from episodic to chronic or unremitting headache) in those individuals with sleep complaints (OR=2.7; 1.1–6.3) for TTH but not migraine. Poorer prognosis for TTH was predicted by fewer hours of sleep (OR=1.4; 1.1–2.0), waking non-refreshed (OR=2.0; 1.1–3.7), and fatigue (OR=2.5; 1.3–4.6) [8].

Clinical Studies Sancisi et al. [32] matched chronic headache patients with episodic headache controls drawn from an Italian neurology service. The chronic headache sample (n=105) included probable migraine or probable TTH with medication overuse (n=50 and n=30, respectively), chronic migraine (n=

4), and CTTH (n=21). Compared to episodic headache controls, chronic headache patients had a higher risk for insomnia every day (54 vs. 24 %; OR=2.71; 1.15–6.36), sleep apnea and/or snoring (49 vs. 37 %; p<0.01), daytime sleepiness (49 vs. 24 %; p<0.05), and anxiety and/or depression (43 vs. 27 %; OR=2.11; 1.17–3.77). Insomnia results were particularly notable: 68 % of the chronic headache group complained of insomnia, with 23 % reporting the daily use of hypnotics (vs. 10 % of episodic). Further multivariate analysis showed that the presence of insomnia was independently associated with chronic headache (OR=5.01; 2.30–10.91). Other variables independently associated with chronic headache were lower education, lower age of headache onset, and antidepressant therapy. While antidepressant use is probably a marker for more severe psychiatric comorbidity, it is also commonly employed in headache prophylaxis.

Insomnia Is a Risk Factor for New-Onset Headache

Longitudinal research has identified insomnia as a risk factor for TTH. The HUNT was one of the largest longitudinal population health studies in history, and headache was included in the collection of the health histories of 75,000 individuals in Norway beginning in 1984–1986 (HUNT-1) and assessed at regular intervals. Insomnia (HUNT-2, 1995–1997) was found to predict new-onset headache 11 years later (HUNT-3, 2006–2008) [28•]. Of 15,268 headache-free individuals in HUNT-2, a total of 2323 individuals developed new-onset headache in HUNT-3, most of which was TTH (n=1299 vs. n=388 for migraine, n=251 for probable migraine, and n=370 for unclassifiable headache). After controlling for gender, age, and sleep medication, insomnia was associated with increased risk during the 11-year interval for TTH [relative risk (RR)=1.4; 1.2–1.8] and both migraine (overall RR=1.4; 1.0–1.9) and unclassifiable headache (RR=1.4; 1.0–2.0). For those with insomnia who reported impairment in ability to work, there was a 60 % increased risk (RR=1.6; 1.3–2.1) for developing headache compared to those with insomnia but without work impairment.

Biobehavioral Model of Bidirectional Sleep-Headache Relationship

A novel biobehavioral model was recently proposed to explain the vicious cycle of sleep dysregulation and chronic headache [33•]. Using a prevailing theory of basic sleep processes (two-process model of sleep-wake regulation, depicting interplay of homeostatic sleep drive and circadian processes) and conceptual understanding of the development of chronic insomnia ("3-P model" of predisposing, precipitating, and perpetuating factors for insomnia), Ong and Park [33•] proposed three tenets: (1) The palliative behavioral response to headache of sleeping/resting can precipitate and perpetuate sleep disturbance over time, (2) the subsequent disruption in sleep physiology increases propensity for headache, and (3) over time, this vicious cycle may serve to transform episodic headache into chronic headache. These tenets parallel the well-known phenomenon in which daytime napping undermines nocturnal sleep drive and causes increased night awakenings, which in turn leads to increased daytime tiredness and more napping, which further disintegrates nocturnal sleep. This cycle undermines the basic physiological sleep-wake processes, which also happens to be critical in the regulation of pain and headache.

Psychiatric Comorbidity

The symptom constellation including headache-sleep-affective disorders is a common and challenging clinical case for a headache specialist. Psychiatric disorders are comorbid with both headache and sleep disorders, especially insomnia [2•]. Rates of psychiatric disorders among CTTH patients are similar to those observed in migraineurs [34], although non-migrainous headache in general (including ETTH and other headache conditions) is not as strongly associated with psychiatric disorders as migraine (odds ratios vs. no headache= 2.0 and 3.1, respectively) [35]. In their review of studies using validated measures of both psychiatric disorders and headache, Heckman and Holroyd [34] found that CTTH was frequently comorbid with affective disorders. Anxiety disorders were more common than mood disorders (mean prevalence=48 vs. 30 %, respectively). Not surprisingly, clinic patients had higher rates of comorbid psychiatric disorders than headache sufferers identified in population studies. Among headache patients with comorbid disorders, these and other comorbidities account for the majority of disability associated with headache [35], suggesting that clinical attention to these comorbid conditions is essential.

Disturbances in sleep are often present during periods of depression and anxiety, and insomnia is the sleep disturbance most commonly associated with these psychiatric disorders [36, 37]. Whether insomnia is a consequence of depression/anxiety (secondary insomnia) remains a source of debate in the sleep medicine community [38, 39]. Data from studies of migraineurs, however, suggest that the association between sleep disturbance and headache is not fully attributable to comorbid psychiatric symptoms [40, 41] and thus deserves independent attention.

Sleep Dysfunction as a Risk for Headache Chronification

The pattern of progression or transformation from episodic TTH (or migraine) to chronic headache sub-forms (i.e., "chronification") has been documented [42]. Risk factors for

this phenomenon include medication overuse, psychiatric comorbidity, stress, obesity, other pain, and sleep variables—habitual snoring and sleep disturbance [43]. Regulation of sleep and early detection of sleep disorders could eventually be used as tools of primary or secondary prevention to halt progression, although research is needed to explore this possibility. At this time, however, there are few drawbacks in headache patients pursuing efforts to regulate sleep, as these strategies are highly compatible with and easily integrated into usual headache care [44].

Pathophysiology

The convergence of sleep and headache disorders is believed to be based on common neuroanatomy primarily in the brainstem and hypothalamus, influenced by melatonin, adenosine, and orexins. Evers [45] recently reviewed the biological basis and processes thought to underlie the relationship between sleep and trigeminal pain. Hypothetical models have primarily considered migraine, cluster, and hypnic headache. Central sensitization is thought to be a key process in the pathophysiology of CTTH [46, 47]. Sleep disturbance or deprivation and resulting fatigue lead to sympathetic activation to boost metabolic process for availability of energy, and the subsequent sympathetic activation may precipitate and perpetuate TTH.

Clinical Implications

Because of their prevalence and impact, comorbid sleep and psychiatric disorders warrant consideration in the diagnosis and treatment for TTH.

Sleep Apnea Headache In the case of SAH, the identification of OSA can be accomplished using a brief screening tool (STOP-Bang in Fig. 1) or other screeners

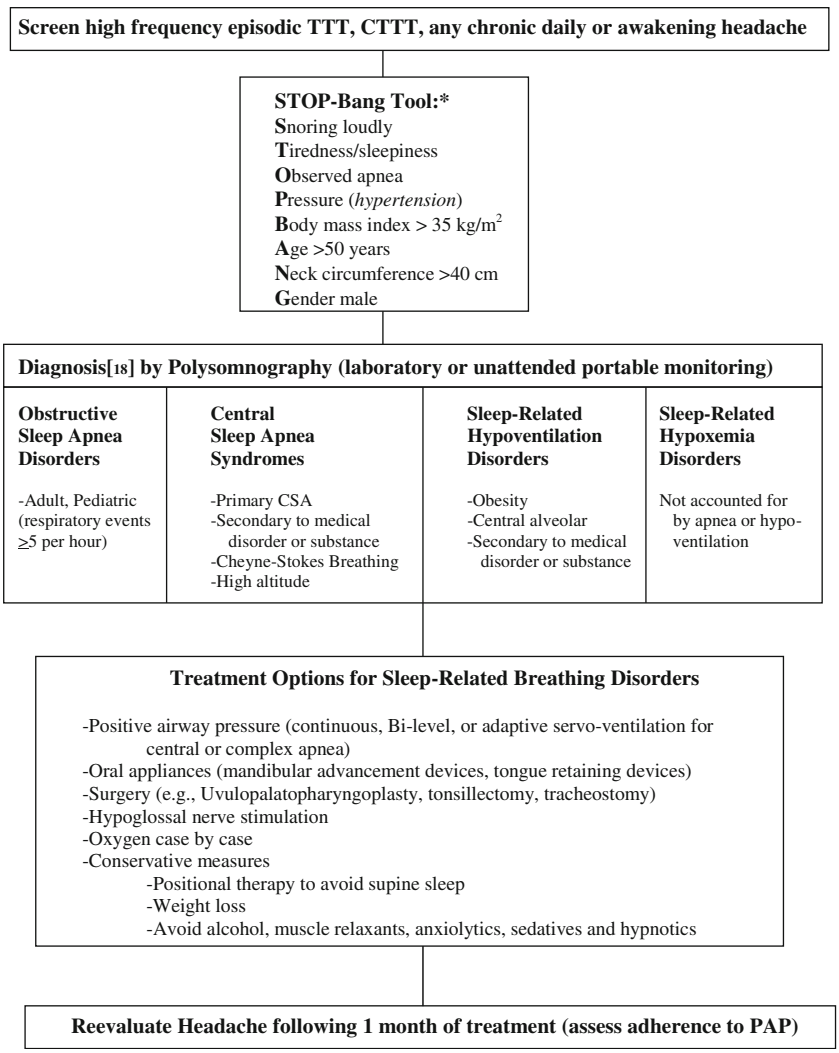

Fig. 1 Screening, differential diagnosis, and treatments of sleep apnea headache. *Asterisk* high risk for sleep apnea defined as approximately greater or equal to three affirmative STOP-Bang answers

described elsewhere [2•]. The STOP-Bang screener, initially developed for presurgical screening for sleep apnea risk, has since been validated in the general medical population [48]. Positive endorsement of three or more items on the STOP-Bang is highly predictive of moderate-to-severe OSA [49] such as that associated with morning headache [17•].

The goal for treatment of SAH is to improve or resolve headache by treating underlying apnea. No controlled trials exist, but anecdotal evidence indicates that SAH treated with continuous positive airway pressure (CPAP) improved or resolved headache in one third to one half of cases [1, 2•, 3, 4]. A month of CPAP treatment should be sufficient to demonstrate headache improvement in patients who are adherent to treatment. Concurrent treatment of headache, using the options articulated below, is usually needed. Opiates, muscle relaxants, and other medications known to exacerbate apnea or suppress respiration should be avoided in headache patients with untreated sleep apnea.

Prophylactic Medications Because patients with SAH usually have insomnia or daytime sleepiness, prophylactic (and acute) medications should be chosen based on their sedating versus neutral or alerting effects. The antidepressant amitriptyline is consistently recommended as the first choice with grade A evidence for prophylactic treatment of CTTH, and patients with insomnia may benefit from bedtime dosing. Mirtazapine, clomipramine, venlafaxine (each with grade B evidence) may also be considered [50, 51]. Psychiatric symptoms, especially anxiety and depression, may also influence selection of treatments. Antidepressants (e.g., amitriptyline, mirtazapine, trazodone, doxepin) are also often used off-label in the treatment of insomnia; thus, sedating antidepressants may be particularly appropriate in the treatment of TTH with insomnia but should be avoided in individuals with excessive daytime sleepiness. Muscle relaxants such as tizanidine [50] and cyclobenzaprine [51] have grade B evidence for TTH but are contraindicated for sleep apnea headache.

Cognitive and Behavioral Treatments Preventive nonpharmacologic treatments are consistently recommended for CTTH, especially biofeedback training (grade A evidence) and cognitive-behavioral therapy (CBT; grade B) [50, 51]. CBT includes self-management of headache triggers (sleep deprivation, oversleeping, stress, etc.). Although insomnia is the sleep complaint most often identified in clinical headache populations, published studies on the treatment of insomnia in TTH patients are lacking. There is some evidence that regulation of sleep may improve chronic migraine [52, 53]. The best empirical evidence supporting the treatment of insomnia in CTTH comes from parallel pain literatures that demonstrate that insomnia improves with behavioral sleep interventions, although it is not known if improvement in insomnia improves pain. Behavioral insomnia therapies are the preferred treatment for insomnia [grade A; 54] and have been adapted to headache patients, although these adaptations have not been validated [55]. Patients with TTH and chronic insomnia should be treated or referred for CBT [56].

Psychiatric Referral or Treatment As noted above, sleep disturbances are diagnostic symptoms of a number of psychiatric disorders, and they occur in the majority of patients with depression, anxiety, or substance abuse. Although there are no evidence-based algorithms to guide treatment, patients with insomnia, depression, or anxiety may benefit from headache prophylaxis using sedating antidepressants or anticonvulsants, while patients with hypersomnia warrant neutral or more alerting medication options. In many cases, treating the comorbid insomnia and psychiatric disorder will require the use of agents separate from those used to treat headache. Patients with severe psychiatric symptomatology should be referred to a mental health professional for concomitant treatment.

Conclusions

A strong association between headache and sleep has been recognized for many years. Conventional wisdom encouraged patients to avoid sleep variations that could trigger headaches (e.g., oversleeping, sleep deprivation) and to sleep off an existing headache. Recent research with sound methodology has certainly supported the bidirectional relationship between TTH and sleep processes. Prospective research demonstrates that sleep variables can trigger acute headaches, predict new-onset headache, and negatively impact prognosis and chronification of headache.

TTH is the most common headache phenotype presenting secondary to a sleep disorder—best established in the case of SAH. Although sleep apnea headache may present like other TTHs, compelling evidence shows underlying mechanisms to be at least in part attributable to hypoxemia, and screening and treatment of OSA is an essential component of headache care. Insomnia is the most common sleep disorder in clinical headache populations, and the majority of patients with CTTH suffer from insomnia. Insomnia can predict new-onset headache and more chronic and severe headache conditions and complicate treatment. Insomnia is also a symptom common to depression and anxiety, and emerging research suggests that treating insomnia directly may improve headache. Because of their impact on headache, in clinical settings, insomnia and psychiatric disorders should be assessed and considered in the selection of pharmacological and cognitive-behavioral treatments.

Compliance with Ethics Guidelines

Conflict of Interest Rachel D. Davis declares that she has no conflict of interest.

Jeanetta C. Rains has received consultancy fees from the National Institute of Health R21 for being a consultant of the study "Sleep regulation and circadian phase in chronic migraine position" and from the American Migraine Foundation for being a consultant of the study "Efficacy of behavioral treatment of insomnia for chronic migraine: a randomized controlled pilot study."

Todd A. Smitherman receives research support from Merck and the American Migraine Foundation.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:

- • Of importance

1. Poceta JS, Rains JC. Sleep and headache syndromes. Sleep Med Clin. 2008;3:427–41.
2.• Rains JC, Poceta JS. Sleep-related headache. In B Vaughn (Ed.) Neurologic clinics. 2012; 30:1285-98. Elsevier: Philadelphia, PA. *This paper provides a thorough literature review and detailed clinical recommendations to guide clinicians in screening for diagnosing and treating sleep-related headache.*
3. Rains JC, Poceta JS, Penzien DB. Sleep and headaches. Curr Neurol Neurosci Rep. 2008;8:167–75.
4. Rains JC, Poceta JS. Headache and sleep disorders: review and clinical implications for headache management. Headache. 2006;46:1344–63.
5. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia. 2013;33:629–808.
6. Vos T, Flaxman AD, Naghavi M, et al. Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012;380:2163–96.
7. Stovner L, Hagen K, Jensen R, et al. The global burden of headache: a documentation of headache prevalence and disability worldwide. Cephalalgia. 2007;27:193–210.
8. Lyngberg AC, Rasmussen BK, Jorgensen T, Jensen R. Incidence of primary headache: a Danish epidemiologic follow-up study. Am J Epidemiol. 2005;161:1066–73.
9. Jensen R, Stovner LJ. Epidemiology and comorbidity of headache. Lancet Neurol. 2008;7:354–61.
10. Provini F, Vetrugno R, Lugaresi E, et al. Sleep-related breathing disorders and headache. Neurol Sci. 2006;27:S149–52.
11. Alberti A, Mazzotta G, Gallinella E, Sarchielli P. Headache characteristics in obstructive sleep apnea and insomnia. Acta Neurol Scand. 2005;111:309–16.
12. Loh NK, Dinner DS, Foldvary N, et al. Do patients with obstructive sleep apnea wake up with headaches? Arch Intern Med. 1999;159: 1765–8.
13. Russell MB, Kristiansen HA, Kværner KJ. Headache in sleep apnea syndrome: epidemiology and pathophysiology. Cephalalgia. 2014;34:752–5.
14. Göder R, Friege L, Fritzer G, et al. Morning headaches in patients with sleep disorders: a systematic polysomnographic study. Sleep Med. 2003;4:385–91.
15. Greenough GP, Nowell PD, Sateia MJ. Headache complaints in relation to nocturnal oxygen saturation among patients with sleep apnea syndrome. Sleep Med. 2002;3:361–4.
16. Sand T, Hagen K, Schrader H. Sleep apnoea and chronic headache. Cephalalgia. 2003;23:90–5.
17.• Doufas AG, Tian L, Davies MF, Warby SC. Nocturnal intermittent hypoxia is independently associated with pain in subjects suffering from sleep-disordered breathing. Anesthesiology. 2013;119:1149–62. *Most conclusive evidence to date linking mechanisms of pain and awakening headache with recurrent nocturnal hypoxemia independent of sleep.*
18.• American Academy of Sleep Medicine. International Classification of Sleep Disorders, 3rd edition. Darien: American Academy of Sleep Medicine; 2014. *The first edition of the diagnostic manual for sleep disorders to recognize "sleep-related headaches" which describes primary headaches and relation to sleep in a five-page appendix.*
19. Wang J, Huang Q, Li N, et al. Triggers of migraine and tension-type headache in China: a clinic-based survey. Eur J Neurol. 2013;20: 689–96.
20. Martin PR, MacLeod C. Behavioral management of headache triggers: avoidance of triggers is an inadequate strategy. Clin Psychol Rev. 2009;29:483–95.
21. Tonini MC, Frediani F. Headache at high school: clinical characteristics and impact. Neurol Sci. 2012;33:S185–7.
22.• Houle TT, Butschek RA, Turner DP, et al. Stress and sleep predict headache severity in chronic headache sufferers. Pain. 2012;153: 2432–40. *Prospective time series analysis demonstrating a relationship between sleep and subsequent headache.*
23. Kikuchi H, Yoshiuchi K, Yamamoto Y, et al. Does sleep aggravate tension-type headache?: An investigation using computerized ecological momentary assessment and actigraphy. Biopsychosoc Med. 2011;5:10.
24. Penzien DB, Andrasik F, Freidenberg BM, et al. Guidelines for trials of behavioral treatments for recurrent headache. Headache. 2005;45:S109–31.
25. Haque B, Rahman KM, Hoque A, et al. Precipitating and relieving factors of migraine vs tension type headache. BMC Neurol. 2012;12:82.
26. Ong JC, Stepanski EJ, Gramling SE. Pain coping strategies for tension-type headache: possible implications for insomnia? J Clin Sleep Med. 2009;5(1):52–6.
27. Odegard SS, Engstrom M, Sand T, et al. Associations between sleep disturbance and primary headaches: the third Nord-Trøndelag Health Study. J Headache Pain. 2010;11:197–206.
28.• Odegard SS, Sand T, Engstrom M, et al. The long-term effect of insomnia on primary headaches: a prospective population-based cohort study (HUNT-2 and HUNT-3). Headache. 2011;51:570–80. *Compelling longitudinal data demonstrating that insomnia predicts later onset of TTH.*
29. Boardman HF, Thomas E, Millson DS, Croft PR. Psychological, sleep, lifestyle, and comorbid associations with headache. Headache. 2005;45:657–69.
30. Ohayon MM. Prevalence and risk factors of morning headaches in the general population. Arch Intern Med. 2004;164:97–102.
31. Rasmussen BK. Migraine and tension-type headache in the general population: precipitating factors, female hormones, sleep pattern and relation to lifestyle. Pain. 1993;53:65–72.
32. Sancisi E, Cevoli S, Vignatelli L, et al. Increased prevalence of sleep disorders in chronic headache: a case-control study. Headache. 2010;50:1464–72.
33.• Ong JC, Park M. Chronic headaches and insomnia: working toward a biobehavioral model. Cephalalgia. 2012;32:1059–70. *Novel*

conceptualization of relationship and potential mechanisms between insomnia and chronic headache.
34. Heckman BD, Holroyd KA. Tension-type headache and psychiatric comorbidity. Curr Pain Headache Rep. 2006;10:439–47.
35. Saunders K, Merikangas K, Low NC, et al. Impact of comorbidity on headache-related disability. Neurology. 2008;70(7):538–47.
36. Baglioni C, Spiegelhalder K, Lombardo C, Riemann D. Sleep and emotions: a focus on insomnia. Sleep Med Rev. 2010;14:227–38.
37. Chorney DB, Detweiler MF, Morris TL, Kuhn BR. The interplay of sleep disturbance, anxiety, and depression in children. J Pediatr Psychol. 2008;33:339–48.
38. Lichstein KL. Secondary insomnia: a myth dismissed. Sleep Med Rev. 2006;10:3–5.
39. Stepanski EJ, Rybarczyk B. Emerging research on the treatment and etiology of secondary or comorbid insomnia. Sleep Med Rev. 2005;10:7–18.
40. Vgontzas A, Cui L, Merikangas KR. Are sleep difficulties associated with migraine attributable to anxiety and depression? Headache. 2008;48:1451–9.
41. Walters AB, Hamer JD, Smitherman TA. Sleep disturbance and affective comorbidity among episodic migraineurs. Headache. 2014;54:116–24.
42. Saper JR. Chronic daily headache: transformational migraine, chronic migraine, and related disorders. Curr Neurol Neurosci Rep. 2008;8:100–7.
43. Scher AI, Midgette LA, Lipton RB. Risk factors for headache chronification. Headache. 2008;48:16–25.
44. Rains JC. Chronic headache and potentially modifiable risk factors: screening and behavioral management of sleep disorders. Headache. 2008;48:32–9.
45. Evers S. Sleep and headache: the biological basis. Headache. 2010;50:1246–51.
46. Coppola G, Di Lorenzo C, Schoenen J, Pierelli F. Habituation and sensitization in primary headaches. J Headache Pain. 2013;14:65.
47. Jensen R. Peripheral and central mechanisms in tension-type headache: an update. Cephalalgia. 2003;23:S49–52.
48. Ong TH, Raudha S, Fook-Chong S, et al. Simplifying STOP-Bang: use of a simple questionnaire to screen for OSA in an Asian population. Sleep Breath. 2010;14:371–6.
49. Silva GE, Vana KD, Goodwin JL, et al. Identification of patients with sleep disordered breathing: comparing the four-variable screening tool, STOP, STOP-Bang, and Epworth Sleepiness Scales. J Clin Sleep Med. 2011;7:467–72.
50. Sarchielli P, Granella F, Prudenzano MP, et al. Italian guidelines for primary headaches: 2012 revised version. J Headache Pain. 2012;13:S31–70.
51. Bendtsen L, Evers S, Linde M, et al. EFNS guideline on the treatment of tension-type headache—report of an EFNS task force. Eur J Neurol. 2010;17:1318–25.
52. Calhoun AH, Ford S. Behavioral sleep modification may revert transformed migraine to episodic migraine. Headache. 2007;47:1178–83.
53. Smitherman TA, Walters AB, Ambrose CE, et al. Randomized controlled trial of behavioral insomnia treatment for chronic migraine with comorbid insomnia: preliminary results of a sham-controlled pilot study. Headache. 2014;54:33–4.
54. Schutte-Rodin S, Broch L, Buysse D, et al. Clinical guideline for the evaluation and management of chronic insomnia in adults. J Clin Sleep Med. 2008;4:487–504.
55. Rains JC. Optimizing circadian cycles and behavioral insomnia treatment in migraine. Curr Pain Headache Rep. 2008;12:213–9.
56. Tran DP, Spierings EL. Headache and insomnia: their relation reviewed. Cranio. 2013;31:165–70.