Chapter 15
Psychological and Psychiatric Treatment of Chronic Head and Face Pain

Taylor E. Rush and Harold W. Goforth

Introduction

Head and facial pain can affect many aspects of a patient's daily life. Chronic pain can affect a variety of ways in which people function, including sleep behaviors, activity patterns, and emotional experiences. As a result, they often require a multidisciplinary approach for successful comprehensive management. Behavioral interventions and psychotropic medication can serve as effective adjuncts to treatment in order to help meet this need and to enhance quality of life. Cognitive-behavioral strategies, relaxation techniques, biofeedback, and operant learning interventions have all been shown to be helpful techniques for behavioral headache management. This chapter will review the central theoretical tenets of cognitive and behavioral interventions, the research that supports their use, as well as a review of psychotropic modalities of treatment for headache prevention and mood management.

T. E. Rush (✉)
Center for Neurological Restoration, Neurological Institute, Cleveland Clinic, Cleveland, OH, USA
e-mail: rusht@ccf.org

H. W. Goforth
Center for Neurological Restoration, Neurological Institute, Cleveland Clinic, Cleveland, OH, USA

Lerner College of Medicine, Cleveland Clinic Foundation, Center for Neurological Restoration, Section of Headache Medicine, Cleveland, OH, USA
e-mail: goforth@ccf.org

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Non-pharmacological Headache Interventions

Cognitive and Behavioral Therapy Interventions

Cognitive-Behavioral Therapy Model Cognitive-behavioral therapy (CBT) has been considered a gold standard psychological intervention for a wide variety of conditions, including depression, anxiety, as well as pain. Psychiatric comorbidities are fairly common in headache disorders. For instance, those with migraine headaches have a 2–4 times higher likelihood of exhibiting co-occurring depression, a 2–7 times higher likelihood of exhibiting anxiety symptoms, and a 3–4 times higher likelihood of experiencing panic episodes [1]. This makes CBT interventions an ideal choice to help successfully manage the behavioral and affective components of patients' pain experiences.

The underlying assumption of CBT is that a person's thoughts, feelings, and emotions are strongly connected and maladaptive appraisals of situations can contribute to dysfunctional coping styles. Over time, this can lead to entrenched patterns of poor coping which can negatively affect a person's mood, relationships, and overall quality of life. For those with chronic head pain, these symptoms may also have deleterious effects on health-promoting behaviors (e.g., exercise, stress-reducing activities, healthy eating, adequate sleep), which can then increase the likelihood of more frequent and/or severe headache episodes.

The research on CBT for headache has provided strong evidence of treatment efficacy. It has been associated with decreased headache frequency by up to 50%, higher reported self-efficacy and internal locus of control, less pain-focused cognitions, improved mood, and increased medical treatment adherence [2]. In addition, CBT has been shown to be cost-effective and can reduce health-care utilization in chronic pain populations [3].

Physiologically, CBT appears to help activate parasympathetic nervous system activity, which can include normalized heart rate and blood pressure, increased peripheral blood flow, and decreased muscle tension. In addition, it can assist with HPA axis and nociceptive regulation by decreasing activity of pro-inflammatory cytokines [4].

Common Negative Thought Patterns Part of CBT treatment involves having patients identify maladaptive appraisals and thought patterns in order to challenge and reframe them in a more realistic, helpful way. This can help to attenuate strong negative emotional reactions that can also feed into poor behavioral coping reactions. When patients can identify negative thought patterns, it becomes easier for them to dispute them and minimize the associated threat.

Catastrophizing is a common negative thought pattern observed in chronic pain conditions. Pain is appraised as a high-level threat and often associated with feelings of helplessness and the inability to stop thinking about pain sensations [5]. The experience of pain is often associated with the belief that physical harm or damage to the body may occur, which can deter a person from engaging in normal activity. Instead, they will pursue safety behaviors, such as rest, isolation, avoidance of potential triggers (e.g., loud noises, bright lights, etc.), and excessive or inappropriate medication use.

Another common negative thought patterns include fear-avoidance cognitions, characterized by strong beliefs that activity will cause pain to worsen [6]. Threatening activities can range from high-impact physical activity, such as rigorous exercise, to minimal impact daily activities such as sitting in front of a computer or driving. This can result in specious reasoning that safety behaviors such as rest and activity avoidance will keep head pain at bay. However, this pattern of activity avoidance can lead to physical deconditioning, which can then cause pain to worsen.

[Example starts] Jane experiences chronic migraines. When pain becomes severe, she thinks, "Here we go again- my day is ruined! This headache is only going to get worse, so I need to cancel my plans." Resultantly, she feels stressed, frustrated, irritable, and out of control of her situation. In reaction to these thoughts and emotions, she withdraws from planned activity, lies down, and takes medication. She may also possibly seek emergent medical help. Over time, Jane develops anticipatory anxiety about her headache episodes (e.g., "If I go somewhere with bright lights or loud noises it will make my headaches worse") and therefore avoids activities that she thinks may induce headaches. This can lead to an overall decrease in activity and functioning and increase in stress due to low quality of life and fear of worsening pain. Additionally, Jane may experience an increase in migraines despite all of her safety behaviors to avoid them [example ends].

Cognitive Restructuring CBT targets changing maladaptive cognitive and behavioral coping patterns in order to enhance adaptive coping and minimize mood and anxiety symptoms that may be comorbid with pain. This involves learning how to identify negative thought patterns and challenging them with more adaptive, realistic thoughts (see Table 15.1). Patients are also taught to acknowledge that pain is not harmful and accept that, while it may not be curable, it is manageable. Setting this expectation at the beginning of treatment is very important so that the patient understands that CBT is not a curative intervention for pain. If realistic expectations are not discussed at the beginning of treatment, successful outcomes are less likely.

Activity Pacing Another key component to effective behavioral management of headaches is moderating activity levels and including activities that promote health. Often, those experiencing chronic pain will demonstrate a boom-bust activity

Table 15.1 Examples of cognitive restructuring

Maladaptive cognition	Restructured cognition
This pain is going to get worse	The pain may get worse, but it will not stay that way forever
There is nothing I can do to stop it	I have tools that can help me manage this
I need to cancel my plans	I can continue to function despite the presence of pain
I need to go to the emergency room	I can manage this without acute medical intervention
Medication is the only thing that helps	I have other strategies that can be just as, if not more, helpful than medication
Activity will worsen my pain	Activity can help me cope more effectively and will not harm me

pattern, where they will overextend themselves on less severe pain days and then rest extensively on more severe days. Engaging in this pattern can result in worsening pain and lower functioning over time. Additionally, as their number of functional days decrease, patients will often begin to exclude pleasant activities, such as hobbies and socializing, and health-promoting activities, such as exercise and self-care.

Learning how to appropriately pace activity and incorporate salutatory behaviors can be a trial and error process. Patients are often asked to log their daily activity and keep their hours of activity per day equitable across high and low pain days. They are encouraged to break up activities that may have historically exacerbated pain, such as exercise, long drives, and computer work, into manageable stretches of time punctuated by breaks. Patients can determine what constitutes a manageable stretch by timing how long they can do an activity before pain begins to worsen. The next time they engage in the activity, they are advised to take a break prior to the point of pain exacerbation. Over time, patients are encouraged to slowly increase the intensity or duration of activity to improve overall functional capacity.

Sleep Hygiene Sleep dysregulation is often an associated symptom of chronic head pain. Insomnia issues are reported in up to two-thirds of chronic headache patients and sleep apnea reportedly co-occurs in up to 60% of this population [7]. For many, problematic sleep can then contribute to significant daytime fatigue, difficulties with concentration, mood dysregulation, and poor performance on daily activities. Behavioral strategies for improving sleep have been found to be more efficacious than medications [8] and therefore play an important role in the successful behavioral management of headaches.

Behavioral strategies for insomnia can fall into three categories: sleep hygiene, stimulus control, and sleep restriction. Table 15.2 below gives a brief description of some of the techniques for each of these strategies.

Relaxation Strategies/Biofeedback Stress has been identified as a potential predisposing and exacerbating factor for chronic head and facial pain. Psychologically, stress typically occurs when the perceived demands of a situation exceed a person's perceived ability to meet those demands. If a stressor is appraised as uncontrollable and the person has few resources to help manage the stressor, physiological as well as psychological stress will likely result. Research has shown that the intensity of stress is correlated with headache frequency [9]. Additionally, pain can contribute to significant daily stress, as it can enhance normal stressors in addition to being a stressor itself. When the body is exposed to prolonged discomfort, it can lead to chronic activation of the sympathetic nervous system's "fight-or-flight" response. This can have a cascading effect on autonomic functioning, including rising blood pressure, heart rate, cortisol levels, immune and inflammatory responses, and muscle tension. Behaviorally, stress can lead to poor self-care; infrequent health-promoting activities, such as healthy eating, adequate hydration, and physical activity; and disengagement from adaptive coping strategies. Together, these biological and behavioral factors can exacerbate the experience of pain.

Table 15.2 Behavioral sleep interventions

Behavioral intervention	Techniques
Sleep hygiene	– Keep consistent bedtimes and wake times – Avoid caffeine, alcohol, nicotine, and spicy foods in the hours leading up to bedtime – Exercise daily – Create a bedtime routine to help wind down – Avoid naps – Increase exposure to natural light during day – Avoid screens (TV, tablets, phones) in the hour leading up to bed
Stimulus control	– Restrict activity in the bedroom (sleep and sex only) – Go to bed only when tired – Get out of bed if not asleep within 15 min; proceed to engage in boring, sedentary activity outside of bedroom – Avoid clock watching
Sleep restriction	– Keep a sleep journal logging total sleep time and actual time spent asleep, and calculate sleep efficiency (SE = time asleep/time in bed) – If SE ≤ 80–85%, reduce time in bed by 15 min increments each week SE stays ≤80% – Once SE > 85%, can slowly add 15 min to time in bed each week until normative bed and wake times are obtained

Lipton and colleagues [10] have examined how the dissipation of stress can specifically contribute to the development of migraine, described as the "let-down" hypothesis. The let-down hypothesis was tested in a small study, which interestingly showed that, while stress level was not associated with migraine frequency, a quick decline in stress over a 24 h period almost doubled the odds of migraine onset in the following 6–18 h. Physiologically, this may be due to lowered HPA activation and reduced glucocorticoid production, which can subsequently trigger a migraine.

Given how stress can potentially precipitate and exacerbate pain, learning how to successfully manage stress can be a critical skill set for effective pain management. By engaging in relaxation strategies, the body's stress response can be better regulated and allow restorative processes to work more effectively, including activating the body's parasympathetic or "rest and digest" response. Cognitively, relaxation techniques help to provide distraction from pain, increase awareness of emotional states, as well as understand the physiological signs of stress and tension on the body. Over time, this information can be used to better manage stress as well as pain sensations. A summary of common relaxation strategies for pain management can be found in Table 15.3.

Biofeedback is considered an objective way in which to monitor the body's response to various relaxation techniques. By receiving real-time feedback on their physiological functioning, such as heart rate, body temperature, muscle tension, and galvanic skin response, patients can become aware of physical responses to pain and stress. They can then use that information to target relaxation strategies to enhance physiological self-regulation. Biofeedback has been shown to be an effective behavioral strategy for head and facial pain management. Specifically, electromyographic

Table 15.3 Summary of relaxation strategies

Relaxation strategy	Description	Mechanism of action
Diaphragmatic breathing	Engaging the diaphragm to breath, allowing the lungs to fill more effectively. Using the diaphragm to breath causes the belly to expand on the inhale, hence the nickname "belly breathing"	Slows the breath rate, allowing better oxygen intake
Progressive muscle relaxation	Systematically tensing and releasing different muscle groups in the body, anywhere from 8 to 32 groups in total	Increases awareness of areas of the body that tend to hold chronic tension and gives an opportunity to relax them, facilitating better awareness of tense and relax states
Autogenic relaxation	Systematically focusing on different areas of the body and imagining them becoming heavy and warm. This can be paired with an image that helps to enhance sensations of heaviness and warmth (e.g., immersing into a warm bath)	Can help to increase peripheral blood flow and decrease chronic muscle tension
Guided imagery	Creating a relaxing mental scene using all five senses	Can serve as a cognitive distraction from pain stimuli

(EMG) for tension-type headaches and thermal biofeedback for migraine have demonstrated efficacy in reducing pain severity and frequency [11]. It has also been found to help reduce medication overuse [12]. When compared to other behavioral interventions for headache, biofeedback and relaxation training has demonstrated to be as effective as CBT-based strategies and more effective than placebo [11].

Operant Behavioral Conditioning

In addition to the above described interventions, operant behavioral therapy has long been established as a way to help decrease pain experiences by targeting reduction of pain behaviors (e.g., talking about pain, grimacing, abnormal posturing, excessive rest). It was first introduced by Fordyce [13], who applied operant learning theory (which posits that learned behavior is a direct result of reinforcement and/or punishment) to pain. He believed that positive reinforcement (positive attention, caregiving) as well as negative reinforcement (e.g., encouraging medication use or utilization of emergency services to abate symptoms, making the environment dark and quiet, encouraging rest) in response to pain behaviors would increase them over time. Therefore, Fordyce recommended that pain behaviors be ignored, while more adaptive and functional behaviors be reinforced via verbal cues and positive attention. Various studies testing this theory have shown that participants who underwent interventions where these principles were applied exhibited less pain behavior, increases in activity, and lower pain ratings [14].

Role of Family Support

Relationship dynamics and interactions can play a significant role in whether a person is able to adaptively manage chronic pain. Research shows that if family members preferentially give attention and support in response to pain behaviors, those behaviors become more frequent and pain ratings are higher [15]. Conversely, family members who invalidate their loved one's experience with chronic pain can lead to increased risk of mood dysregulation and maladaptive coping [16]. There can be significant benefit from incorporating family into behavioral interventions. This can provide an opportunity for loved ones to learn ways in which they can be supportive without reinforcing pain behavior or minimizing their family member's struggle. These family-based interventions can focus on facilitating assertive communication, including verbalizing and acknowledging how pain has affected their respective roles in their relationship. It can also be helpful for family members to elucidate expectations moving forward for how to appropriately handle pain crises, so all involved understand how they can show support without being enabling or withholding.

Pharmacological Interventions

The use of psychotropic medication for headache prevention in addition to mood management has been more commonly accepted through the past several decades. These medications can be an effective way to address headache pain directly as well as any comorbid mood or anxiety symptoms. The following paragraphs will provide a brief overview of various medication classes and the research supporting their use.

MAOI Therapies

The monoamine oxidase inhibitor theory of migraines states that alterations of monoamines in the central nervous system produce dysregulated responses in the cerebral vasculature to produce migrainous headaches. An early study looking at the effect of treating migraine with beta-blockers and nonselective monoamine oxidase inhibitors (phenelzine) demonstrated a significant improvement in frequency and severity of migraine attacks. Anxiety and depression were also improved in both the phenelzine monotherapy group and the group receiving dual therapy with beta-blockers (atenolol) [17]. Other data looking at selective MAOIs have not been supportive of their use in the prevention of migraine headaches. Selegiline in particular failed to demonstrate improvement upon migraine without aura; however, the doses of oral selegiline used in the study did not reach therapeutic levels to allow the inhibition of MAO-A in this group. It may be more accurate with regard to this study to

note that selective MAO-B inhibitors are ineffective in the prevention of migraine rather than interpret this as a failure of MAOIs in general [18].

Tricyclic Therapies

Following the advent of tricyclic antidepressants, these became one of the most widespread groups of medications used for migraine prevention in both pediatric and adult patients. While tricyclics are one of the most common medication classes prescribed for the prevention of migraine headache, there is a paucity of large-scale or randomized trials with respect to this class of agents. Amitriptyline is the most common of the tricyclics that are prescribed for migraines, although nortriptyline and desipramine are other potential agents that may have improved side effect profiles.

One large-scale trial of amitriptyline that was performed between 1976 and 1979 was not fully reported until 2011 but involved a placebo-controlled trial of amitriptyline of 20-week duration. Study participants included both intermittent migraine patients and chronic daily headache. Dropout rates were significant across the study with only 48% completing week 20. There was a significant improvement in headache frequency for amitriptyline over placebo at 8 weeks, but not at 12, 16, or 20 weeks. The amitriptyline effect was more pronounced for those subjects with chronic daily headache where superiority of amitriptyline was evident at 8 weeks and 16 weeks, but not 20 weeks.

A retrospective cohort study examining amitriptyline dose and treatment outcomes demonstrated that there is a significant range of amitriptyline dosing from 2.5 to 100 mg daily, which are considered lower doses in the dosing spectrum of amitriptyline. Interestingly, amitriptyline was well tolerated and approximately three-fourths of patients were found to derive significant clinical benefit in the reduction of migraine frequency [19].

Amitriptyline has also been demonstrated to be cost-effective in the prevention of migraine headaches among low- and middle-income countries. Amitriptyline was considered more cost-effective than either topiramate or propranolol [20]. Amitriptyline has also been combined with cognitive-behavioral therapy (CBT) for chronic migraines in adolescents and children. The data demonstrated a robust marginal reduction in headache frequency when CBT was added to amitriptyline treatment over amitriptyline treatment alone. These benefits were sustained at 12 months [21].

Amitriptyline has shown to be comparably efficacious to other well-established and FDA-approved medications for migraine such as divalproate over a 6-month period. Divalproex was more effective at 3 months with respect to headache frequency and visual analog scale of severity. However, the significant differences between the two treatment groups had dissipated by 6 months when no significance was found between the two groups. Hair loss, menstrual irregularities, polycystic ovaries, and weight gain were all more common in the divalproex group [22].

The Canadian Headache Society produced a set of guidelines that stratified available medications for migraine prevention according to available data. Amitriptyline received strong recommendation for use [23].

Other tricyclic depressants have also been identified as effective choices for migraine prophylaxis. Monotherapy with any one migraine preventative agent has been noted to improve only a minority of individuals. However, dual therapy with different classes of migraine preventatives has been shown to be more effective than monotherapy alone. One study examined the role of additive nortriptyline 30 mg daily to topiramate 100 mg daily in monotherapy nonresponders. Seventy-eight percent of patients receiving polytherapy demonstrated at least a 50% reduction in headache frequency compared to 37% of those assigned to the monotherapy groups. Combination therapy of topiramate and nortriptyline was effective in patients with inadequate improvement on monotherapy of either agent alone [24].

Beta-Blocker Therapies

A randomized double-blind controlled trial examined the efficacy of low-dose propranolol 40 mg daily, nortriptyline 20 mg daily, and combination therapy of the two agents. The period of treatment was 2 months. Treatment with propranolol alone or in combination was effective, but monotherapy with nortriptyline was not effective. This study was limited by a very small sample size that was underpowered for the number of groups and possible outcomes [25]. Another open-label study examining the role of amitriptyline in 25 and 50 mg dosages over a 2-month period also demonstrated a weak effect upon migraine prevention and reduced the number of monthly migraines from an average of 7 to 6 per month [26]. Thus, while most treatment algorithms embrace amitriptyline or other tricyclics in the prevention of migraine headaches, the data supporting this use is not uniformly consistent.

SSRIs and SNRIs

Other antidepressant classes of medications have been examined for migraine prevention and treatment of depression in migraine populations. A review of selective serotonin reuptake inhibitors (SSRI) for preventing migraine and tension headaches identified 13 studies utilizing 5 different SSRIs. SSRIs did not significantly lower headache index score in patient with migraine when compared to placebo after 2 months. SSRIs were more tolerable than tricyclic compounds [27].

A prospective study comparing venlafaxine to escitalopram demonstrated a clear advantage of venlafaxine over escitalopram in terms of headache frequency, duration, and severity. Daily work performance also improved in the venlafaxine group. The escitalopram group also showed reductions in frequency, duration, and severity, but the reductions were less robust than in the venlafaxine group. These effects were

independent of mood disorders. [28]. Other SSRI studies examining the role of fluoxetine have demonstrated small but significant effect sizes with respect to migraine frequency while improving mood in these populations [29, 30].

A randomized, double-blind, crossover study examining venlafaxine versus amitriptyline in the preventative treatment of migraine demonstrated that both drugs have benefits in pain parameters. Venlafaxine was more tolerable than amitriptyline in this study, and fewer patients dropped out of the venlafaxine arm. The study was limited by a small sample size ($n = 52$) [31].

Duloxetine is a selective serotonin-norepinephrine reuptake inhibitor that has received considerable attention for its use in pain syndromes. One recent prospective study examined the role of duloxetine 60–120 mg daily in the prevention of episodic migraine in persons without depression. The study was limited by a small sample size, but greater than 50% of participants receiving duloxetine (mean dose 110 mg daily) had a 50% or greater improvement in number of monthly headache days [32]. An older 8-week open-label trial of duloxetine 60 mg daily for comorbid major depressive disorder and chronic headache noted significant improvements in both depression scores and headache frequency [33]. Duloxetine appears well tolerated across both studies.

In summary, there appears to be generally good but not uniformly agreeable data for the use of antidepressant therapies in the prevention of migraine headaches as well as mood management. Data appear more robust for lower-dose tricyclic compounds and dual serotonin-norepinephrine reuptake inhibitors than for selective serotonin reuptake inhibitors. Amitriptyline appears to be the compound most commonly prescribed for migraine prevention, although more recent data suggest a growing role for venlafaxine and duloxetine as alternatives.

Placebo Effects

Placebo effects in headache treatment have demonstrated that patient expectations, as well as provider proclamations and attitude regarding the treatment, can play a role in pharmacological and behavioral treatment efficacy. In RCTs of acute headache medication that had treatment and placebo groups, reported pain improvements in placebo groups have been upward of 28% versus 58% in active treatment [34]. Unfortunately, no studies have been conducted that assign patients to a waitlist control as well as placebo and active treatment groups. For preventative pharmacotherapies, RCT meta-analyses show that 21% of those in placebo groups report a ≥50% reduction in number of headache days, in comparison to 41% in active treatment. Few RCTs of behavioral therapies have been conducted, and many studies are confounded by selection bias as well as facilitator variability. This makes replication of some interventions difficult, especially since level of engagement in therapeutic providers as well as patients can affect treatment gains and placebo effects. However, the research that has been done has shown that participants who underwent true biofeedback reported better symptom improvement than those who

participated in sham behavioral therapies. This has also shown to be true with relaxation training and cognitive-behavioral interventions.

Conclusion

Psychological and psychiatric treatment can be integral aspects of comprehensive head and facial pain management. Behavioral interventions can help equip patients with self-regulatory skills necessary to manage mood, pain exacerbations, physiological stress, activity levels, and social engagement. This can help to enhance a sense of control over symptom management and contribute to a higher quality of life. Psychotropic medications can be used in concert with behavioral strategies in order to help with headache prevention as well as mood dysregulation.

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