Behavioral and Psychologic Aspects of the Pathophysiology and Management of Tension-type Headache

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Behavioral and psychologic factors in tension-type headache are reviewed with reference to pathophysiology, comorbid psychiatric disorders, headache triggers, and behavioral treatment, including the efficacy of behavioral treatments, brief minimal contact administration of behavioral treatment, therapeutic mechanisms underlying the effectiveness of behavioral treatments, and the integration of drug and behavioral treatments. Anxiety or depression may contribute to central sensitization that underlies frequent tension-type headaches. Excessive analgesic use, comorbid psychiatric disorders, or persistent unaddressed headache triggers may limit the effectiveness of headache treatment. Available drug and behavioral treatments are effective for episodic tension-type headache, but are only moderately effective for chronic tension-type headaches. The combination of behavioral and preventive drug therapies may improve outcomes for patients with chronic tension-type headache.

Introduction

Episodic tension-type headache is often readily managed with simple behavioral prescriptions and nonprescription analgesics or nonsteroidal anti-inflammatory agents. However, chronic tension-type headaches (CTTHs), which occur at least 15 days each month for at least 6 months [1] and often every day or nearly every day for patients seeking treatment, present a more difficult management problem [2–4]. This review examines psychologic and behavioral factors in the pathophysiology and management of tension-type headaches, emphasizing CTTH.

Pathophysiology

Frequent tension-type headaches are currently thought to be maintained by a central nervous system (CNS) dysfunction, rather than by input from peripheral nerves in contracted facial, neck, and shoulder muscles [5,6]. This CNS dysfunction may involve a sensitization of pain transmission circuits in the trigeminal nucleus [7•,8] that lowers the threshold of these circuits for the transmission of nociceptive signals. A dysfunction in supraspinal (limbic) pain modulation circuits also has been postulated to maintain pain by permitting or facilitating the transmission of nociceptive information [3,9]. However, research has failed to provide support for the latter possibility, at least when the putative dysfunction is indexed by abnormalities in the exteroceptive suppression of temporalis or masseter muscle activity [10–13].

In response to standardized pressure stimuli, pericranial muscle tenderness has been observed consistently in tension-type headache, particularly in CTTH, even when the patient is in a headache-free state [10–12,14,15]. From the shape of pressure-pain curves, Bendtsen *et al.* [16] have concluded that this muscle tenderness results from a sensitization of second-order neurons in the trigeminal nucleus and dorsal horn [7•,8]; nitric oxide appears to be involved in this sensitization process at the molecular level [17].

Anxiety or depression may contribute to the sensitization of pain transmission circuits [12,18]. For example, depression was associated with elevations in pericranial muscle tenderness, reductions in peripheral pain tolerance, and an increased likelihood that patients would experience tension-type headaches after laboratory psychologic stress [18]. Thus, depression or anxiety may increase vulnerability to or maintain frequent tension-type headaches by aggravating the central sensitization in pain transmission pathways, and through associated muscle tension that elevates peripheral input. As the pathophysiology of central sensitization becomes better understood, it should be possible to investigate the role of stress and emotion in tension-type headache more precisely.

Comorbid Psychiatric Disorders

The prevalence of anxiety or mood disorders does not appear to be elevated in episodic tension-type headache [19,20]. However, it is doubtful this finding can be generalized to CTTH. In clinical samples, the prevalence of anxiety and mood disorders is high in CTTH; more than 40% of patients with CTTH in primary care settings and higher percentages of

Table 1. Precipitating factors for headache as identified by patients

Lifestyle factors	Hormonal and physical factors	Environment
Fatigue	Head or neck injury	Altitude changes
Inconsistent eating patterns	Hormone replacement therapy	Air pollution
fasting	Medications	Bright or flickering lights; glare
missing meals	Menstrual period (before, during, after)	Exposure to vapors or chemicals (eg, gasoline, industrial fumes, cleaning products)
insufficient food	Oral contraceptives	Motion
Sleep	Physical exertion	Noise
excessive sleep	Postmenopausal changes	Perfumes, colognes
unrefreshing sleep	Pregnancy	Smoke
insufficient sleep	Unergonomic posture	Strong odors
change in sleep schedule		Weather changes (eg, barometric changes)
sleep problems (eg, delayed onset, restless sleep)		
Stress		
during stress		
after stress (eg, let-down headache)		
Travel		
Smoking or passive smoke		

patients with CITH who are seen in specialty settings are diagnosed with an anxiety or mood disorder by standardized diagnostic assessments [4,21–23]. Moreover, a comorbid anxiety or mood disorder appears to increase the disability associated with CITH, suggesting that the identification and effective management of comorbid psychiatric disorders plays an important role in reducing disability for patients with headache who also have a comorbid psychiatric disorder [4].

Headache Triggers

If patients are alerted to common triggers and early warning signs of headache and are encouraged to record these prospectively, most of them can learn to identify headache triggers and early headache warning signs. Table 1 presents the most frequently reported headache triggers. Unergonomic posture (*eg,* head forward), working position (*eg,* at desk, using industrial equipment), or sleeping position often are not spontaneously reported, but may be evident during a physical examination, through careful inquiry, or in the pattern of headaches that are recorded in a headache diary.

Identification of previously unrecognized triggers opens up new behavioral options for the prevention of headaches. Conversely, the failure to address persistent headache triggers may limit the effectiveness of other treatments. Identification of early signs (*eg,* muscle tension, environmental cues associated with headache onset) can provide warnings to employ behavioral headache management skills or to use medication abortively.

Excessive Medication Use

Headaches aggravated by excessive medication use (analgesic, ergotamine, triptan), often termed rebound headaches, are notably refractory to drug and behavior therapies [24]. In one retrospective study [25], fewer than one third of patients who were administered high amounts of medication achieved clinically significant reductions (50% or more) in headache activity with behavioral treatment; more than 50% of patients who were administered lower levels of medication showed this same level of improvement. High medication consumption in this study was defined by a score of 40 or higher on a commonly used weighted medication index [26], the equivalent of approximately six aspirin or three Fiorinal (Novartis, East Hanover, NJ) daily.

Medication withdrawal combined with behavioral therapy or with behavior therapy and prophylactic pharmacotherapy together has been found to be effective in managing rebound headaches in several case series. At a 17-month follow-up evaluation, Baumgartner *et al.* [27] reported that medication withdrawal accompanied by prophylactic pharmacotherapy and relaxation training produced significant reductions in headache activity in 61% of patients who had been using high levels (mean 39 doses weekly) of medication. However, the benefits of behavior therapy or prophylactic medication in facilitating medication withdrawal have not been evaluated in controlled trials.

It is important to help patients understand rebound headaches and to provide clear guidelines for using problem medications. In addition, the behavioral factors that can influence analgesic consumption need to be addressed. For example, some patients take analgesics in anticipation of a headache, although they are unable to accurately predict the onset of headaches. It is often anxiety and fear of headaches rather than a valid signal of the onset of a headache that cues the patient to take medication. For other patients, it is the mood-altering effects of medications containing barbiturates or caffeine that reinforce excessive use of medications. Behavioral interventions can be used to help patients distinguish

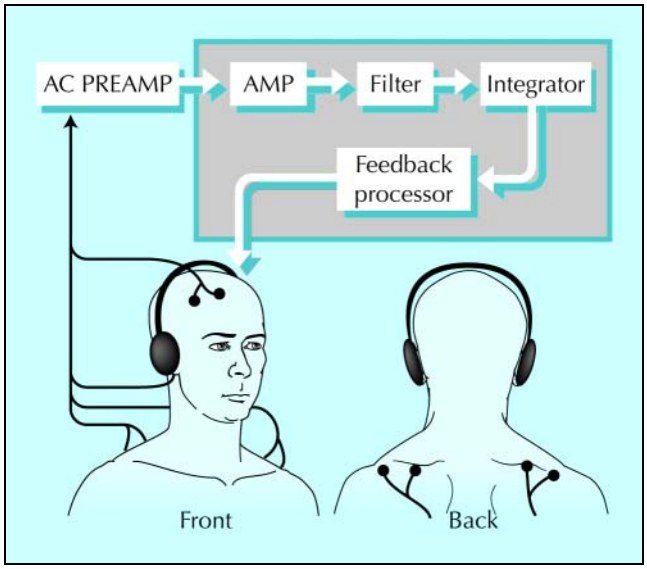

Figure 1. Schematic of electromyography biofeedback training. Frontal area and trapezius muscles are being monitored with ground on the ear lobe.

valid signs of headache onset from anxiety or fear of headaches and can teach alternative skills for managing anxiety.

Behavioral Management

Behavioral treatments can be efficacious if used as an alternative or as an adjunct to medication in the treatment of frequent tension-type headache [28•,29–31]. The most commonly used behavioral interventions for tension-type headache can be grouped into three categories: relaxation training, biofeedback training, and cognitive-behavior (stress-management) therapy. Although these treatments attempt to influence the frequency and the severity of headaches, they emphasize the prevention of headache episodes. Behavioral treatments may also reduce psychologic symptoms, affective distress, or somatic complaints.

Relaxation training

Relaxation training skills are thought to enable patients with recurrent headache disorders to exert control over headache-related physiologic responses. The practice of relaxation exercises also assists patients in achieving a sense of mastery or self-control over their headaches, providing a brief hiatus from everyday stress and enabling patients to manage everyday stressors. Three types of relaxation training techniques are used in the treatment of headache: progressive muscle relaxation, which is alternately tensing and relaxing specific muscle groups throughout the body [32]; autogenic training, which is the use of self-statements of feelings of warmth and heaviness to achieve a state of deep relaxation[33]; and meditative exercises or passive relaxation techniques, which use silently repeated words or sounds to promote mental calm and relaxation [34]. These various relaxation training procedures often are combined in a treatment package (Fig. 1).

Biofeedback training

Biofeedback training refers to procedures that provide information about physiologic processes (usually through the use of electronic instrumentation) in the form of an observable display (typically an audio tone or visual display) [35]. This information, or feedback, is used by the patient to learn to self-regulate the response that is being monitored. Electromyographic (EMG) biofeedback, which is feedback of electrical activity from muscles of the scalp, neck, and sometimes the upper body, is typically used to teach patients to self-regulate muscle tension in the treatment of tension-type headache (Fig. 1). EMG biofeedback is typically administered in combination with relaxation training.

Stress-management training (cognitive-behavioral therapy)

The rationale for stress-management training derives from the observation that the way patients cope with everyday stressors can precipitate, exacerbate, and maintain headache episodes, or increase headache-related disability and distress [36,37•]. Stress-management therapy focuses on the cognitive and affective components of headaches. It is typically administered in conjunction with relaxation interventions. Stress-management interventions are used to teach patients to identify stressful circumstances that precipitate or aggravate headaches and to employ effective strategies for coping with these stressors. They also help patients cope more effectively with pain and distress associated with headache episodes, and limit negative psychologic consequences of recurrent headaches (*eg,* depression and disability).

Minimal contact treatment formats

Minimal contact behavioral treatment employs only a few (*eg,* typically 3 to 4 monthly) clinical visits to introduce relaxation or stress-management skills and to address problems that patients encounter in acquiring these self-regulation skills. Headache management skills are acquired primarily at home with guidance from home study materials (audiotapes and workbooks) and periodic (*eg,* biweekly) phone calls between patient and therapist. Relaxation and stress-management therapies have been effectively administered in a minimal contact or home-based treatment format [38–42]. Minimal contact treatment protocols can reduce the cost and increase the availability of behavioral treatment.

There are no absolute contraindications to minimal-therapist-contact treatment. However, Lipchik *et al.* [37•] proposed relative contraindications that incorporate existing empirical evidence.

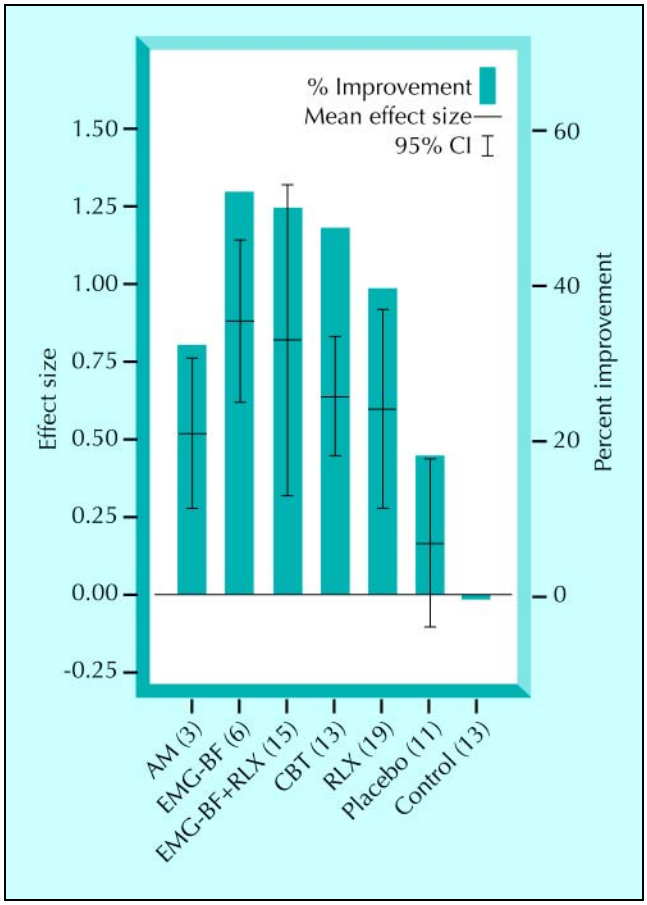

Figure 2. Effect size with 95% confidence interval (top axis) and percent reduction in tension-type headache activity (bottom axis). AM—amitriptyline HCl; EMG-BF—electromyographic biofeedback training; EMG-BF + RLX—electromyographic biofeedback training plus relaxation training; CBT—cognitive-behavior therapy; RLX—relaxation training; placebo—pseudotherapy or false biofeedback training; control— headache monitoring control. (*Adapted from* McCrory *et al.* [31])

Limited reading comprehension, low intelligence, or significant cognitive impairment

Patients with reading comprehension below the eighth grade level, below average intelligence, deficits in concentration, attention, or memory, or organic brain damage typically will have difficulty making effective use of the written materials and audiotapes.

Misuse or overuse of medication

Patients need to reduce or eliminate offending medications if there is evidence of medication overuse (*eg,* use of acute medications 20 days or more monthly). Patients who have difficulty reducing their medication use on their own may benefit from additional treatment sessions during this period.

Chronic daily severe headache

Patients who have near-constant headaches that can be severe often respond poorly to relaxation and biofeedback therapies [43]. Patients with chronic daily headache may require multimodal therapy that includes prophylactic medications and behavioral intervention over a longer period of time than that afforded in a minimal contact treatment protocol.

Nonadherence

Nonadherence refers to the failure of patients to complete headache diaries or homework assignments. Some patients simply do not make the effort to learn or apply self-regulation skills without regular contact with a health professional

Psychiatric comorbidity

Patients who are significantly impaired by a psychiatric disorder or who have a personality disorder may require attention; they may also have a personality disorder. The presence of psychologic symptoms or a comorbid psychiatric disorder does not prohibit behavioral treatment; psychologic treatment may be indicated. However, additional treatment sessions may be required to address more severe psychologic problems.

Age

Some older patients have difficulties with attention, concentration, or other problems that affect the learning of novel material. Although these patients may not benefit from minimal contact treatment, they often can benefit from a clinic-based program [44,45] (Fig. 2).

Efficacy

The Agency for Health Care Research and Quality (AHCRQ) evaluated behavioral treatments for tension-type headache [31,46], concluding that a consistent body of research indicated that efficacy exists for the four behavioral treatments described previously. Figure 2 shows that these behavioral treatments have each yielded a 40% to 50% improvement in tension-type headache activity when results are averaged across trials; these improvements are at least equal to those reported with amitriptyline HCl.

Approximately one third (n = 35) of the identified behavioral treatment trials (n = 107) could be included in the formal AHCRQ meta-analysis because many trials failed to provide the information necessary to calculate the treatment effect sizes used in meta-analysis. Nonetheless, additional analyses suggested that overall findings would not materially change in the larger pool of trials. Analyses conducted for this evidence report are consistent with results from earlier, more inclusive meta-analyses that used different statistical techniques [47–49] (Fig. 3).

Clinical wisdom suggests that the presence of a comorbid psychiatric disorder reduces the effectiveness of treatment, but empirical data supporting this is limited [21,50,51]. A comparative trial of stress-management (cognitive-behavioral) and preventive drug (tricyclic antidepressant) therapies for chronic tension-type headache found that both active treatments significantly differed from

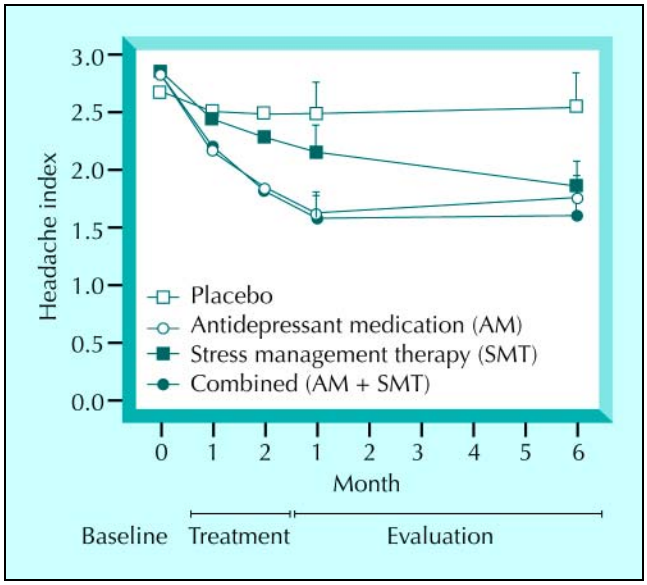

Figure 3. Mean Headache Index scores at baseline during treatment (dose adjustment) and (with standard error) at one-month and 6-month evaluations. Headache Index is calculated as a mean of daily headache ratings (range 0–10) over 1-month periods. AM—antidepressant medication; PL—placebo; SMT—stress-management therapy; AM + SMT—combined treatment. (*Adapted from* Holroyd *et al.* [56••] with permission.)

pill placebo only when a comorbid anxiety disorder was present or when headaches were severe (high headache index score); active treatment and placebo plus clinical management produced similar improvements for patients with less complicated headache problems [50]. This suggests that active (drug or behavioral) treatment may be beneficial for patients who have more complicated headache problems; however, active treatment may add little to education and clinical management for patients with less complicated headache problems. A relevant study of migraine [51] suggests that the presence of a comorbid psychiatric disorder may be associated with long-term (6 to 7 years) relapse, even if it is not associated with a poor initial response to drug therapy. Thus, long-term maintenance of improvements may be dependent on the effective management of headaches and co-morbid psychiatric disorders.

Therapeutic mechanisms

Efforts to understand how behavioral treatments produce improvements in headache activity have focused on EMG biofeedback training. The conventional wisdom that EMG biofeedback training reduces tension headache activity simply by enabling patients to control sustained contractions in pericranial muscles was challenged in the early 1980s [52,53]. An alternative cognitive-attributional model emphasizing changes in self-efficacy (the patients perception that they can take actions to manage their headaches) as the key therapeutic mechanism in EMG biofeedback has received some support [53–55]. However, the possibility that improvements result from the impact of EMG biofeedback training on central pain modulation deficits that have been postulated to underlie frequent tension-type headaches has not received support (at least when hypothesized CNS deficits were indexed by abnormalities in exteroceptive suppression periods in masseter activity) [55]. Improvements in tension-type headaches following EMG activity may be mediated by different therapeutic mechanisms in different patients, with each of the therapeutic mechanisms illustrated in Figure 2 being important for some patients.

Integrating Drug and Behavior Therapies

Trials comparing cognitive-behavioral therapy and (tricyclic) antidepressant medication for chronic tension-type headache suggest that these two treatments produce similar outcomes; however, they raise the possibility that combining the two treatments may enhance outcomes [40,56••]. In the largest study to date [56••], 203 patients were randomized to one of four of the following treatments: tricyclic antidepressant (100 mg/d of amitriptyline hydrochloride or 75 mg/d of nortriptyline hydrochloride), medication placebo, minimal-contact (three clinical sessions) stress-management (cognitive-behavior therapy) plus antidepressant medication, or stress-management therapy plus placebo. Antidepressant medication and stress-management therapy yielded similar reductions in CTTHs, analgesic medication use, and headache-related disability at a 6-month follow-up evaluation; improvements tended to be more rapid in the antidepressant medication conditions than with stress-management therapy alone (Fig. 3). However, the combined treatment was more likely to produce clinically significant (≥ 50%) reductions in CTTHs (64% of patients), than antidepressant medication alone (38% of patients) or stress-management therapy alone (35% of patients).

Reich and Gottesman [57] examined the long-term benefits of adding amitriptyline (75 mg/d) to an intensive (30 sessions) multiple-site EMG biofeedback protocol. The combination of amitriptyline HCl and EMG biofeedback yielded more rapid improvement in tension-type headache activity than EMG biofeedback alone; however, the combined treatment showed no advantage over EMG biofeedback alone beginning at month 8 and continuing through the 24-month evaluation period. In fact, patients who received EMG biofeedback alone at the 20- and 24-month observation period, after withdrawal from amitriptyline HCl, recorded significantly fewer hours of headache activity than patients who received combined treatment. Thus, EMG biofeedback training was associated with better long-term outcomes than the combined treatment, at least after medication was discontinued. It has been suggested that patients who attribute their improvement to medication may cease to employ the headache management skills learned in behavioral treatment over time and may lose the benefits of combined treatment.

Conclusions

Behavior may influence the pathophysiology of tension-type headache if it leads to persistent muscle strain that produces excessive peripheral input from pericranial nociceptors or exposure to headache triggers. Anxiety or depression may contribute to the sensitization of pain transmission pathways that underlie chronic tension-type headaches by elevating muscle tension and peripheral input from pericranial nociceptors and by other mechanisms. Available drug and behavioral treatments are effective for episodic tension-type headache, but are only moderately effective for chronic tension-type headache. However, the combination of behavioral and preventive drug therapies may improve outcomes with chronic tension-type headaches. Management of comorbid anxiety and depression headaches appears to be important in reducing headache-related impairment.

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