

Assessment and Psychological Management of Recurrent Headache Disorders

Kenneth A. Holroyd
Ohio University

This article updates earlier reviews of recurrent headache disorders published in 1982 and 1992, selectively reviewing research published since 1990. Current issues in assessment (headache diagnosis, psychophysiology, comorbid psychopathology, quality-of-life assessment, and new assessment technologies) and psychological treatment (efficacy, therapeutic mechanisms, treatment delivery, and integration with drug therapy) are addressed. The author emphasizes the need to adapt psychological treatments to the severity of the headache disorder and to developments in drug therapy. Opportunities for the integration of biological, medical, and psychological science are highlighted.

Consistent with the theme of this special issue, this article selectively reviews research on recurrent headache disorders published since 1990, updating two earlier reviews (Blanchard, 1992; Blanchard & Andrasik, 1982). Space limitations mandate a selective review. I have thus chosen to give particular attention to the topics that have not been updated since the 1982 review.

Diagnosis and Assessment of Headaches

In the two decades since this topic was last reviewed, a new system for classifying headache disorders has been widely adopted, the assessment of headaches has broadened to include the assessment of the impact of a recurrent headache disorder on a person's life, and new technologies, such as handheld computers, have begun to influence the way assessments are conducted.

The International Headache Society (IHS; Olesen, 1988) classification system included three changes of particular relevance to psychologists: (a) the addition of operational diagnostic criteria modeled after those in the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (*DSM-IV*; American Psychiatric Association, 1994); (b) the addition of criteria to distinguish episodic (ETTH) from chronic (CTTH) tension-type headache and to distinguish analgesic-abuse headache from other headache disorders; and (c) the elimination of mixed (tension and migraine) headache, a notoriously unreliable diagnosis. Diagnostic reliability appears to have improved (e.g., Granella et al., 1994; Leone, Filippini, D'Amico, Farinotti, & Bussone, 1994; Rasmussen, Jensen, & Olesen, 1991), and the adoption of the IHS classification system in epidemiological studies has contributed to a dramatic increase of knowledge about the epidemiology and impact of

recurrent headache disorders (cf. Edmeads et al., 1993; Hu, Markson, Lipton, Stewart, & Berger, 1999; Lipton, Stewart, & Von Korff, 1997; Lipton, Stewart, & Simon, 1998; Pryse-Philips et al., 1992; Rasmussen, Jensen, & Olesen, 1992; Schwartz, Stewart, & Lipton, 1997; Schwartz, Stewart, Simon, & Lipton, 1998; Solomon & Price, 1997; W. F. Stewart, Lipton, & Simon, 1996; van Roijen, Essink-Bot, Koopmanschap, Michel, & Rutten, 1995).

Nonetheless, the current IHS classification system is not without problems. In fact, controversy surrounds the diagnosis of three headache disorders that are frequently referred for psychological management: (a) nearly continuous headaches that currently receive multiple headache diagnoses (e.g., migraine, tension-type, and analgesic-abuse headache), where the debate concerns the nature and classification of these headaches (e.g., Guitera, Munoz, Castillo, & Pascual, 1999; Lipton, Stewart, Cady, et al., 2000; Olesen & Rasmussen, 1996; Silberstein, Lipton, & Sliwinski, 1996; Silberstein, Lipton, Solomon, & Mathew, 1994); (b) analgesic and other drug-induced chronic headache problems, where more accurate criteria are needed to identify headaches that are induced or aggravated by medications used to treat headaches (Diener, 1993; Diener & Wilkinson, 1988); and (c) pediatric headaches, for which more appropriate diagnostic criteria are needed (Maytal, 1997; Raieli et al., 1996; Winner, Martinez, Mate, & Bello, 1995). Periodic revisions of the IHS classification system are planned as new research findings become available, and the first revision is now underway. *I encourage psychologists to contribute to improvements in the classification and diagnosis of these headache disorders. For example, the assessment of behavior might be incorporated into the diagnosis of pediatric headache where the accurate communication of symptoms can be a barrier to diagnosis, possibly with a checklist that enables parents to monitor avoidance behaviors indicative of the presence of key diagnostic criteria (e.g., photophobia, phonophobia, nausea).*

It is widely recognized that pain measures provide only limited information about the impact of a pain disorder on a person's life. Attention has thus been devoted to the development of disability and quality of life measures to more adequately capture the impact of headaches on the individual (Dahlof, 1993; Solomon, 1997). Characteristics of measures used in recent studies are summarized in Tables 1 and 2. It can be seen that these measures vary in their

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Correspondence concerning this article should be addressed to Kenneth A. Holroyd, Psychology Department, Ohio University, Athens, Ohio 45701. E-mail: holroyd@ohio.edu

specificity (for migraine, for headache in general, or for medical conditions in general), in the dimensions they assess (primarily disability or other impact dimensions as well), in the time frame they address (a single migraine episode, or a month or longer period of time), and in the availability of supportive psychometric data. Criteria used to evaluate the published psychometric data for each measure can be seen in a key at the bottom of Table 2. *Psychologists have much to contribute to the development and validation of disability and quality of life measures because these instruments purport to assess the behavioral and affective impact of recurrent headache disorders. Appropriate disability and quality of life measures should be included in future studies that evaluate psychological treatments.*

The availability and low cost of handheld computers have led investigators to consider computerizing the headache diary (Hermann, Peters, & Blanchard, 1995; Holroyd & Chen, 2000; Honkoop, Sorbi, Godaeri, & Spierings, 1999). Potential advantages of the handheld computer include the abilities to monitor the time of each diary entry, to efficiently collect large amounts of data without unduly burdening the patient, and to transmit data directly to the investigator over a wireless network or via modem, and the possibility of two-way communication between the patient and either a host Web site or a health professional. *Handheld computer technology has the potential to both enhance the quality and quantity of data that can be collected and, by providing two-way electronic communication between the patient and health care professional, to change the way educational and behavioral interventions are administered. We are likely to see this new technology effectively used in both data collection and the administration of educational and psychological interventions in the next decade.*

Psychophysiology of Headache

When this topic was last reviewed (in 1982), the muscle contraction model of tension headache (postulating that tension headaches result from a prolonged contraction of pericranial muscles, which stimulates peripheral nerves or nociceptors and reduces blood flow in affected muscles) and the vascular model of migraine (postulating that migraines result from paroxysmal vasodilatation in the cranial circulation) constituted the accepted orthodoxy. Although empirical data had presented problems for both models at the time of the 1992 review (e.g., Andrasik, Blanchard, Arena, Saunders, & Barron, 1982; Langemark, Jensen, & Olesen, 1990; Olesen, Larsen, & Lauritzen, 1981; Pikoff, 1984), no alternative model had received widespread acceptance. In the past decade this orthodoxy underwent fundamental revision.

Tension-Type Headache

Frequent tension-type headaches are now thought to be maintained primarily by a central nervous system (CNS) dysfunction, not solely by input from peripheral nerves in contracted facial, neck, and shoulder muscles (Olesen & Schoenen, 2000). This CNS dysfunction may involve the sensitization of pain transmission circuits in the trigeminal nucleus, where input from nerves in the face and head is first integrated and relayed toward the brain (Bendtsen & Ashina, 2000; Bendtsen, Jensen, & Olesen, 1996; Olesen, 1991). Such sensitization would lower the threshold of these circuits for the transmission of pain signals, so that little or

no input from peripheral nerves (nociceptors) is required for the transmission of pain signals to the brain. A dysfunction in supraspinal (limbic) pain modulation circuits may also maintain pain by permitting, or even facilitating, the transmission of pain signals in the brain (Schoenen, 1993; Schoenen, Jamart, Gerard, Lenarduzzi, & Delwaide, 1987; Schoenen & Wang, 1997).

This shift from peripheral to central mechanisms has stimulated a resurgence of interest in the psychophysiology of tension-type headache. Whereas earlier research focused primarily on muscle tension as reflected in electromyographic (EMG) activity, or measures of blood flow in pericranial muscles, recent research is focusing on measures that are likely to be sensitive to the CNS dysfunctions of interest. For example, elevated levels of pericranial muscle tenderness in response to standardized pressure stimuli have been observed consistently in tension-type headache—particularly CTTH—even when the subject is in a headache-free state (e.g., Jensen, Rasmussen, Pedersen, & Olesen, 1993; Langemark & Olesen, 1987; Lipchik et al., 1996; Lipchik et al., 2000; Lipchik, Holroyd, Talbot, & Greer, 1997). Bendtsen and colleagues (Bendtsen et al., 1996) have concluded from the shape of pressure-pain curves that this muscle tenderness results from a sensitization of second-order neurons in the trigeminal nucleus and dorsal horn. *These and other findings point to central sensitization as an important psychophysiological mechanism in frequent tension-type headache (Bendtsen & Ashina, 2000) and possibly in chronic daily headache or chronic migraine (Burstein & Cutrer, 2000) as well. Thus, psychophysiological measures of sensitization are likely to receive increased attention in the next decade.*

Migraine

Migraine has been reconceptualized as primarily a neuronal disorder rather than a vascular disorder (Goadsby, 1997; Olesen & Goadsby, 2000). Imaging studies showing the activation of brain stem regions involved in the control of sensory, nociceptive (pain), and vascular functions during spontaneous migraine provide support for the existence of a brain stem “migraine generator” (Weiller et al., 1995). The sensory disturbances, or *aura*, that can precede migraine now are believed to result from a *spreading depression*, or a transient inhibition of neuronal activity, that passes over the cerebral cortex (Lauritzen, 1994). Pain and other phenomena of migraine are postulated to result from activation of trigeminal innervation of the vasculature; it is not yet clear whether it is the spreading depression that induces trigeminal activation or whether spreading depression is simply a parallel phenomenon (Goadsby, 2001). Nonetheless, activation of trigeminal nerves induces neurogenic inflammation (dilation and leakage of plasma protein) from arteries surrounding the brain, sensitization of nerve endings at these arteries, and sensitization of pain transmission circuits in the trigeminal nucleus (Moskowitz, 1992; Olesen & Goadsby, 2000). Pain then results when sensitized nerves are stimulated by dilated arteries sending pain signals through highly sensitized pain transmission circuits, but it may also be generated by a dysfunction in supraspinal (limbic) pain modulation systems.

At the time of the 1982 review, psychophysiological research was still guided primarily by the vascular model of migraine, focusing primarily on blood flow in extracranial arteries, or, peripherally, as reflected in finger temperature. A decade later, psychophysiological studies were actively examining abnormali-

Table 1
Quality of Life and Disability Measures: Description

| Measure | Description and development |
|---|---|
| Headache-specific measures | |
| 1. HImQ: Headache Impact Questionnaire or MIDAS: Migraine Disability Assessment (W. F. Stewart, Lipton, Simon, Von Korff, & Liberman, 1998; W. F. Stewart, Lipton, Kolodner, et al., 1999; W. F. Stewart, Lipton, Simon, et al., 1999; W. F. Stewart et al., 2000; Lipton, Stewart, Sawyer, & Edmeads, 1998; Holmes, MacGregor, Sawyer, & Lipton, 2001) | <ul style="list-style-type: none"> ■ Measures pain and activity limitations due to headache. Focuses on disability. Most recent version consists of five easy-to-quantify items. ■ No subscales. ■ Items based on previous population-based and managed care-based studies of migraine. Reviewed by international group of headache clinicians and researchers. |
| 2. MSQ: Migraine-Specific Quality of Life Questionnaire (Jhingran, Osterhaus, Miller, Lee, & Kirchdoerfer, 1998; Dahlof et al., 1997; Martin et al., 2000) | <ul style="list-style-type: none"> ■ Measures general QoL among people with migraines. ■ Three subscales: Role Function-Restrictive, Role Function-Preventative, and Emotional Function. ■ Items included only if meaningful to both patients and clinicians. |
| 3. HDI: Headache Disability Inventory (Jacobson, Ramadan, Aggarwal, & Newman, 1994; Jacobson, Ramadan, Norris, & Newman, 1995) | <ul style="list-style-type: none"> ■ Measures functional and emotional impact of headache on everyday life. ■ Two subscales: Functional Impairment and Emotional Impairment. ■ Derived from patient perceptions of headache-related disability and based on existing scales for hearing and dizziness disability. |
| 4. MQoLQ: Migraine Quality of Life Questionnaire (24-hr) (Santanello, Hartmaier, Epstein, & Silberstein, 1995; Hartmaier, Santanello, Epstein, & Silberstein, 1995) | <ul style="list-style-type: none"> ■ Measures 24-hr QoL changes associated with an acute migraine attack. ■ Five subscales: Work, Social, Energy/Vitality, Feelings/Concerns, and Symptoms. ■ Items based on general health-related QoL literature, clinical migraine experts, and patients. |
| 5. MSQOL: Migraine-Specific Quality of Life Measure (Wagner, Patrick, Galer, & Berzon, 1996; Patrick & Hurst, 1998; Patrick, Hurst, & Hughes, 2000) | <ul style="list-style-type: none"> ■ Measures general QoL among people with migraines. ■ No subscales. ■ Developed using Hunt and McKenna's needs-based model (Hunt & McKenna, 1992). Based on ethnographic interviews with migraine patients and literature reviews. |
| 6. QLH-Y: Quality of Life Headache in Youth (Langeveld, Koot, & Loonen, 1996; Langeveld, Koot, & Passchier, 1997; Langeveld, Koot, & Passchier, 1999) | <ul style="list-style-type: none"> ■ Measures QoL for adolescents between ages 12 and 18 who experience chronic headaches or migraines. ■ Five subscales: Psychological Functioning, Functional Status, Physical Status, Social Functioning, and Satisfaction With Life in General. ■ Derived from existing measurements of psychological functioning, social functioning, and satisfaction with life and health. |
| 7. HANA: Headache Needs Assessment (Cramer, Silberstein, & Winner, 2001) | <ul style="list-style-type: none"> ■ Brief assessment of health status and migraine impact on daily activities. ■ Seven Subscales: Anxiety/Worry, Depression/Discouragement, Self-Control, Energy, Function/Work, Family/Social Activities, and Overall Impact. ■ Items derived from interviews with migraine patients, headache specialists, and literature review of existing migraine quality of life measures. |
| 8. RIIP-HA: Recurrent Illness Impairment Profile (Headache Version) (Wittrock, Penzien, Moseley, & Johnson, 1991; Penzien, Rains, Dawson, Wirth, & Turkewitz, 1992; Holroyd, Malinoski, Davis, & Lipckik, 1999) | <ul style="list-style-type: none"> ■ Measures functional impairment associated with episodic or recurrent illnesses. ■ Two subscales: General Functional Impairment and Employment-Specific Functional Impairment. ■ Development not reported. |
| 9. MWPLQ: Migraine Work and Productivity Loss Questionnaire (Davies, Santanello, Gerth, & Lerner, 1999) | <ul style="list-style-type: none"> ■ Measures impact of migraine and migraine therapy on paid work. ■ Seven subscales: Time Management, Work Quality, Work Quantity, Bodily Effort, Interpersonal Demands, Mental Effort, and Environmental Factors. ■ Development not reported. |
| 10. HIT: Headache Impact Test (Ware, Bjorner, et al., 2000; Ware, Kosinski, et al. 2000; Ware, Bayliss, Kosinski, et al., 2000) | <ul style="list-style-type: none"> ■ Computerized dynamic tool designed to assess impact of headache on patient functional health and well-being. ■ No subscales. ■ Items adapted from four widely used surveys (HDI, HImQ, MIDAS, MSQ) and from clinician recommendations. Adaptive dynamic assessment based on item response theory analysis. |
| 11. HIT-6: Headache Impact Test-6 (short, static assessment) (Ware, Bayliss, Dahlof, et al., 2000) | <ul style="list-style-type: none"> ■ Short, static assessment of headache impact on patient functional health and well-being. ■ No subscales. ■ Six items chosen from larger HIT item pool to assess six domains: pain, role functioning, social functioning, energy/fatigue, cognition, and emotional distress. |
| 12. QoLQ: Quality of Life Questionnaire (Cavallini, Micieli, Bussone, Rossi, & Napi, 1995) | <ul style="list-style-type: none"> ■ Measures the impact of headache on QoL during and between headache attacks for both episodic and chronic headache. ■ Six subscales: Physical Functioning, Role Functioning, Social Functioning, Mental Health, Symptoms, and Health Perception. ■ Based on international guidelines including functional state, physical symptoms, psychological conditions, and social interactions. |

Table 1 (continued)

| Measure | Description and development |
|---|---|
| General measures used with headache | |
| 1. MOS-36: Medical Outcomes Study Short Form Health Survey (36) (Solomon et al., 1993; Ware & Sherbourne, 1992; Osterhaus et al., 1994; Essink-Bot, van Royen, Krabbe, Bonsel, & Rutten, 1995; Weinberger, Oddone, Samsa, & Landsman, 1996; Durham et al., 1998; Monzon & Lainez, 1998) | <ul style="list-style-type: none"> ■ General measure of disease impact. ■ Eight subscales: Physical Functioning, Role Functioning–Physical, Role Functioning–Emotional, Social Functioning, Bodily Pain, Mental Health, Vitality, General Health. ■ Developed as a brief instrument for general assessment of QoL. |
| 2. MSEP: Minor Symptom Evaluation Profile (Dahlof, 1993; Dahlof & Dimenas, 1995; Dahlof, 1995) | <ul style="list-style-type: none"> ■ General measure of subjective CNS-related symptoms that affect well-being. ■ Three subscales: Contentment, Vitality, and Sleep. ■ Developed as a general assessment of CNS-related symptoms and QoL. |
| 3. NHP: Nottingham Health Profile (Hunt, McEwen, McKenna, Brackett, & Pope, 1982; Jenkinson, Fitzpatrick, & Argyle, 1988; Jenkinson, 1990; Essink-Bot et al., 1995; Essink-Bot, Krabbe, Bonsel, & Aaronson, 1997) | <ul style="list-style-type: none"> ■ General measure of health-related QoL. ■ Six subscales: Energy, Pain, Emotional Reactions, Sleep, Social Isolation, and Physical Mobility. ■ Developed as a general measure of perceived health. |
| 4. QWB-SA: Quality of Well-being Scale—Self-Administered (Sieber, Ganiats, & Kaplan, 1997; Seiber, David, Adams, Kaplan, & Ganiats, 2000) | <ul style="list-style-type: none"> ■ General measure of health-related QoL that is self-administered. Includes assessment of death. ■ Four subscales: Symptoms, Mobility, Physical Activity, and Social Activity. ■ Developed as a less expensive measure of health-related QoL than the QWB and could still be used for cost-effectiveness analysis. |
| 5. SSAP: Subjective Symptom Assessment Profile (Dimenas, Dahlof, Olofsson, & Wiklund, 1990; Dahlof & Dimenas, 1995) | <ul style="list-style-type: none"> ■ General measure of subjective symptoms. ■ Six subscales: Emotional Distress, Gastrointestinal Symptoms, Peripheral Vascular Symptoms, Cardiac Symptoms, Sex Life, and Dizziness. A subset of these questions is considered migraine specific. ■ Developed as a general measure of QoL. |
| 6. PGWB: Psychological General Well-Being (Dupuy, 1984; Dahlof & Dimenas, 1995) | <ul style="list-style-type: none"> ■ General measure of changes in well-being. ■ Six subscales: Anxiety, Depressed Mood, Positive Well-Being, Self-Control, General Health, and Vitality. ■ Developed as a general measure of QoL. |
| 7. QWB: Quality of Well-being Scale (interviewer administered) (Kaplan, Bush, & Berry, 1976; Sieber et al., 2000) | <ul style="list-style-type: none"> ■ General measure of health-related QoL administered by an interviewer. Includes assessment of death. ■ Four subscales: Symptoms, Mobility, Physical Activity, and Social Activity. ■ Developed as a general measure of health-related QoL that could be used for cost-effectiveness analysis in determining quality-adjusted life-years. |
| 8. MOS SF-20: Medical Outcomes Study Short Form Health Survey (20) (A. L. Stewart, Hays, & Ware, 1988; Solomon et al., 1993; Skobieranda, Solomon, & Gregg, 1993) | <ul style="list-style-type: none"> ■ General measure of disease impact. ■ Six subscales: Physical Functioning, Role Functioning, Social Functioning, Mental Health, Health Perceptions, and Pain. ■ Developed as a brief version of the MOS for general assessment of QoL. |

Note. QoL = quality of life; CNS = central nervous system.

ties in central neuronal function in migraine (Schoenen, 1996, 1997, 1998). One particularly active area of research focuses on altered thresholds for firing and the failure of habituation in neurons in the brain vulnerable to migraine.

The most rapidly growing literature examines abnormalities in slow negative cortical potentials in electroencephalic activity (EEG), because these potentials are thought to provide a measure of the excitability of cortical neuronal networks (Elbert, 1993). Abnormalities in slow cortical potentials, particularly increased amplitudes and reduced habituation in migraine relative to healthy controls, have been reported to both visual and auditory stimuli, as well as during signaled reaction-time tasks (e.g., Afra, Proietti-Cecchini, DePasqua, Albert, & Schoenen, 1998; Bocker, Timsit-Berthier, & Schoenen, 1990; Evers, Bauer, Husstedt, & Grote-meyer, 1997; Evers, Quibelday, Grote-meyer, Suhr, & Husstedt, 1999; Kropp & Gerber, 1995; Kropp et al., 1999; Lahat, Nadir, & Barr, 1997; Maertens de Noordhout, Timsit-Berthier, & Schoenen, 1986; Schoenen, 1998; Schoenen, Maertens de Noordhout, &

Timsit-Berthier, 1986; Schoenen, Wang, Albert, & Delwaide, 1995); in at least some studies, these abnormalities have differentiated migraine from tension-type headache (Evers et al., 1997; Lahat et al., 1997; Maertens de Noordhout et al., 1986; Wallasch, Kropp, Weinschutz, & Konig, 1993). Increased amplitudes and reduced habituation of responses also appear temporally related to the onset of migraine, being most pronounced a few days before a migraine episode and normalizing during the attack itself (Evers et al., 1999; Judit, Sandor, & Schoenen, 2000; Kropp & Gerber, 1995). Finally, several small studies have indicated that these abnormalities are normalized by preventive migraine medication (Diener et al., 1989; Maertens de Noordhout, Timsit-Berthier, Timsit, & Schoenen, 1987; Siniatchkin, Gerber, & Vein, 1998). On the other hand, negative findings also have been reported (Palmer & Chronicle, 1998; Sand & Vanagaite Vingen, 2000), observed abnormalities could be a consequence of repeated migraine attacks rather than reflect the primary mechanisms in migraine (Chronicle & Mulleners, 1994), and it is not yet clear whether positive

Table 2
Quality of Life and Disability Measures: Psychometrics

| Measure | Internal consistency ^a | Test-retest reliability ^b | Convergent validity ^c | Discriminative validity ^d | Sensitivity examples ^e |
|---|-----------------------------------|--------------------------------------|---|---|---|
| Headache-specific measures | | | | | |
| 1. HImQ: Headache Impact Questionnaire or MIDAS: Migraine Disability Assessment | ■ *** | ■ *** | ■ *** ■ Migraine diary ■ Physician's judgment of disability | ■ ** ■ Severe vs. less severe migraine as determined by physicians | ■ ** ■ Improves with withdrawal from drug overuse (D'Amico et al., 2001) |
| 2. MSQ: Migraine-Specific Quality of Life Questionnaire | ■ *** | ■ ** | ■ *** ■ Migraine severity ■ MOS SF-36 | ■ ? | ■ **** ■ Improves with sumatriptan treatment (Cohen et al., 1996; Dahlof et al., 1997; Jhingran et al., 1996; Loftland, Johnson, Batenhorst, & Nash, 1999) |
| 3. HDI: Headache Disability Inventory | ■ ** | ■ *** (long-term and short-term) | ■ *** ■ Migraine severity ■ Spouse version of HDI ■ HANA | ■ *** ■ Severe vs. moderate vs. mild headache ■ Not able to distinguish between headache types | ■ ** ■ Improves with guided imagery (Mannix, Chandurkar, Tusek, & Solomon, 1999) ■ Improves with amitriptyline vs. placebo (Holroyd et al., 2000) |
| 4. MQoLQ: Migraine Quality of Life Questionnaire (24-hr) | ■ ** | ■ ? | ■ ** ■ Migraine diary | ■ ** ■ Migraine with nausea/vomiting vs. migraine without nausea/vomiting ■ Migraine-free period vs. 24-hr. postmigraine | ■ **** ■ Improves with rizatriptan vs. placebo (Ahrens et al., 1999; Santanello et al., 1997; Pascual et al., 2000) |
| 5. MSQOL: Migraine-Specific Quality of Life Measure | ■ ** | ■ ** | ■ ** ■ Migraine severity ■ PGWB ■ MOS SF-36 | ■ ? | ■ *** ■ Sumatriptan vs. usual care (Mushet, Miller, Clements, Pait, & Gutterman, 1996) ■ Improves with zolmitriptan (Patrick, Hurst, & Hughes, 2000) |
| 6. QLH-Y: Quality of Life Headache in Youth | ■ ** | ■ ** | ■ ** ■ Diary QLH-Y ■ Parent ratings of child QLH-Y | ■ ** ■ Headache vs. headache-free subjects ■ Captured changes in headache status | ■ ? |
| 7. HANA: Headache Needs Assessment | ■ ** | ■ * | ■ ** ■ HDI | ■ * ■ Severe migraine vs. less severe migraine | ■ ** ■ Improves with Depakote treatment (Cramer, Silberstein, & Winner, 2001) |
| 8. RIIP-HA: Recurrent Illness Impairment Profile (Headache Version) | ■ ** | ■ ** | ■ * ■ Migraine severity | ■ * ■ Tension headache vs. migraine ■ Migraine vs. tension headache with migraine | ■ ? |
| 9. MWPLQ: Migraine Work and Productivity Loss Questionnaire | ■ ** | ■ ? | ■ ** ■ Migraine diary ■ MOS SF-36 ■ MQoLQ | ■ ? | ■ ** ■ More improvement with rizatriptan vs. usual care (Dasbach, Gerth, Pigeon, Santanello & Kramer, 1997) |
| 10. HIT: Headache Impact Test | ■ ** | ■ ? | ■ ? | ■ *** ■ Migraine vs. general headache ■ Mild vs. moderate vs. severe headache ■ Low frequency migraine vs. high frequency migraine | ■ ** ■ Pharmacological treatment vs. no treatment |
| 11. HIT-6: Headache Impact Test-6 (short, static assessment) | ■ ** | ■ ? | ■ ? | ■ ** ■ Migraine vs. general headache ■ Mild vs. moderate vs. severe headache | ■ ? |
| 12. QoLQ: Quality of Life Questionnaire | ■ ? | ■ ? | ■ ? | ■ ** ■ Episodic headache vs. chronic daily headache | ■ ? |

Table 2 (continued)

| Measure | Internal consistency ^a | Test-retest reliability ^b | Convergent validity ^c | Discriminative validity ^d | Sensitivity examples ^e |
|--|-----------------------------------|--------------------------------------|---|---|--|
| General measures used with headache | | | | | |
| 1. MOS SF-36: Medical Outcomes Study Short Form Health Survey (36) | ■ *** | ■ ** | ■ ** ■ MOS SF-20 ■ NHP | ■ **** ■ Migraine vs. headache-free ■ Migraine vs. severe-headache ■ Migraine vs. chronic daily headache ■ Headache vs. chronic disease | ■ *** ■ Improves with sumatriptan treatment (Jhingran et al., 1996; Litaker, Solomon, & Genzen, 1997) ■ Improves with guided imagery (Mannix et al., 1999) ■ **** |
| 2. MSEP: Minor Symptom Evaluation Profile | ■ ** | ■ ** | ■ ** ■ NHP ■ PGWB | ■ * ■ Migraine vs. headache-free | ■ Improves with sumatriptan treatment (Dahlof, Edwards, & Toth, 1992) ■ Improves with diclofenac-K treatment (Dahlof, 1993) ■ No change with sumatriptan treatment (Dahlof, 1995) ■ ? |
| 3. NHP: Nottingham Health Profile | ■ * | ■ ? | ■ ** ■ MOS SF-36 | ■ *** ■ Headache vs. headache-free ■ Migraine vs. migraine-free ■ Migraine vs. rheumatoid arthritis | ■ ? |
| 4. QWB-SA: Quality of Well-being Scale—Self-Administered | ■ Not headache | ■ ** | ■ ** ■ QWB ■ MOS SF-36 ■ Pain intensity and disability ratings | ■ * ■ Migraine vs. migraine-free days | ■ ? |
| 5. SSAP: Subjective Symptom Assessment Profile | ■ ** | ■ ? | ■ ? | ■ ** ■ Migraine vs. headache-free | ■ * ■ Some improvement on three dimensions after stepped care drug treatment (Dahlof, 1995) ■ ? |
| 6. PGWB: Psychological General Well-Being | ■ ** | ■ ? | ■ ? | ■ ** ■ Migraine vs. headache-free | ■ ? |
| 7. QWB: Quality of Well-Being Scale (Interviewer) | ■ Not headache | ■ Not headache | ■ ** ■ QWB-SA | ■ * ■ Migraine vs. migraine-free days | ■ ? |
| 8. MOS SF-20: Medical Outcomes Study Short Form Health Survey (20) | ■ ** | ■ ? | ■ - - ■ Poor in one study (Holroyd et al., 2000) | ■ **** ■ Headache vs. headache-free with other chronic diseases ■ Tension headache vs. headache-free | ■ - - ■ Poor in one study of 6 month outpatient treatment (Skobieranda, Solomon, Gregg, 1993) |

Note. Rating system: ? = no research evidence found; - - = negative evidence; * = low support from one source; ** = moderate to high support from one source; *** = moderate to high support from multiple sources; **** = moderate to high support from multiple, independent sources.

^a The degree to which items within a scale measure the same construct. Determined by interitem correlations, usually Cronbach's alpha. ^b The degree to which an individual's scores on a scale are stable over time. Determined by correlating individuals' scores from two different testing times. ^c The degree to which a scale coherently relates to other measures of the same construct. Determined here by positive correlations with other related measures. ^d The degree to which a scale is able to differentiate individuals in groups that vary on the targeted construct. Determined by significantly different scores for different diagnoses or headache-related conditions. ^e The degree to which a scale is able to capture changes in the targeted construct due to treatment. Determined here by significant score changes after pharmacological or nonpharmacological treatment.

findings should be interpreted as reflecting low (Schoenen, 1996) or high (Welch & Ramadan, 1995) prestimulus cortical excitability in migraine. *Nonetheless, initial positive findings will ensure that slow cortical potentials receive attention from psychophysicologists as one methodology for examining deficits in the regulation of cortical excitability in migraine.*

This "new" psychophysiology of migraine has also stimulated investigators to creatively use other techniques to examine cortical excitability—for example, drawing on basic research in psychophysics that has identified tasks where visual performance is

influenced by cortical excitability. Two recent studies have thus used performance on perceptual tasks involving high-contrast masking stimuli as an indirect measure of cortical excitability (McColl & Wilkinson, 2000; Palmer, Chronicle, Rolan, & Mulleners, 2000). Electromagnetic stimulation of the cortex also has been used to assess thresholds for the firing of cortical neurons; for example, the level of electromagnetic stimulation of the visual cortex that is required for the subject to report a visual response (phosphenes or lights in the visual field) is assessed. Thresholds for activation of the visual or motor cortex were decreased in

migraine in four studies (Agugia, Zibetti, Febbarano, & Mutani, 1999; Aurora, Ahmad, Welch, Bhardwaj, & Rhamadan, 1998; Aurora, Wasserman, Shultz, & Welch, 2001; Aurora & Welch, 1998); however, no difference in thresholds in migraine and control subjects (Afra, Marcia, Gerard, Maertens de Noordhout, & Schoenen, 1998) or even lower thresholds in migraine (Werhahn et al., 2000) have been reported in other studies. Thus, these interesting findings must be regarded as preliminary.

The promise that such psychophysiological measures (Schoenen, 1997, 1998) of CNS excitability can provide a window into central mechanisms in migraine appears likely to make this an active research area in the next decade. The use of biofeedback (neurofeedback) training to teach the self-regulation of cortical excitability (Siniatchkin et al., 2000) is also likely to stimulate considerable interest. However, the psychophysiological measures of current interest provide only global indices of neuronal activity in roughly defined cortical (and possibly subcortical) brain regions. Thus, the ability of these measures to assess migraine mechanisms with precision remains unproven. The history of clinical biofeedback also teaches us that therapeutic claims should await evidence that neurofeedback training produces benefits beyond those obtained with a suitable (pseudoneurofeedback) control procedure or simple relaxation training (Andrasik & Holroyd, 1980; Silver & Blanchard, 1978).

Headache Disorders and Comorbid Psychopathology

When this topic was last reviewed (in 1982), no adequately controlled epidemiological study had examined comorbid psychological disorders in individuals with a recurrent headache disorder. At that time information came primarily from convenience samples of patients seeking treatment for headache problems, where selection factors (e.g., Berkson's paradox¹) render samples unrepresentative of the larger population of headache sufferers. Also, psychological problems often were inferred from scores on self-report measures that lack adequate specificity for psychiatric diagnosis and fail to distinguish psychiatric symptoms from consequences of recurrent headaches such as fatigue and sleep problems (Breslau, Merikangas, & Bowden, 1994; Holm, Penzien, Holroyd, & Brown, 1994; Holroyd, France, Nash, & Hursey, 1993; Merikangas & Rasmussen, 2000).

Two decades later, a number of epidemiological studies, including two influential longitudinal studies (Breslau & Davis, 1993; Breslau, Davis, Schultz, & Peterson, 1994; Breslau, Merikangas, et al., 1994; Merikangas, Angst, & Isler, 1990; Merikangas, Merikangas, & Angst, 1993), have used structured interviews to diagnose both headache (IHS) and psychiatric (*DSM*) disorders in population studies. One can see in Table 3 that the prevalence of mood and/or anxiety disorders has been substantially elevated in migraine sufferers in all recent epidemiological studies.

Longitudinal data further argue that the association between mood disorders and migraine is bidirectional: For example, Breslau and colleagues (Breslau, Davis, et al., 1994; Breslau, Merikangas, et al., 1994) found that migraine increased the risk of a subsequent episode of major depression (adjusted relative risk = 4.8) but that the presence of major depression also increased the risk of subsequently developing migraine (adjusted relative risk = 3.3). Longitudinal data also have been interpreted as suggesting that there is a distinct syndrome whereby both an anxiety

Table 3
Association Between Migraine and Mood or Anxiety Disorders: Odds Ratios

| Study | N | Mood disorders | Anxiety disorders |
|---|--------|----------------|-------------------|
| Breslau, Davis, et al. (1994); Breslau and Davis (1993) | 1,007 | 3.6 | 1.9 |
| Merikangas et al. (1990, 1993) | 591 | 2.2 | 2.7 |
| Merikangas (1996) | 1,218 | 3.0 | 2.8 |
| Moldin et al. (1993) | 914 | 2.1 | 2.1 |
| Stewart, Celentano, and Linet, (1989) | 10,169 | | 5.3 |

disorder and a mood disorder are comorbid with migraine (Merikangas & Rasmussen, 2000). Interestingly, Merikangas et al. (1990) found that in this "syndrome," the onset of the anxiety disorder preceded the onset of migraine in about 80% of individuals whereas the onset of depression followed the onset of migraine in about 75% of individuals.

Prospective epidemiological data are more limited for tension-type headache. Epidemiological data suggest the prevalence of anxiety and mood disorders are not elevated in ETTH (Merikangas, 1994; Merikangas, Stevens, & Angst, 1993). However, it is doubtful this finding can be generalized to CTTH. In clinical samples the prevalence of both anxiety and mood disorders is high in CTTH: Over 40% of CTTH sufferers in primary care settings and even higher percentages of CTTH sufferers seen in specialty settings receive either an anxiety or mood disorder diagnosis by standardized diagnostic assessments (Goncalves & Monteiro, 1993; Guidetti et al., 1998; Holroyd et al., 2000; Puca, Genco, & Prudeniano, 1999). Moreover, a comorbid anxiety or mood disorder appears to increase the disability associated with CTTHs, suggesting that the identification and effective management of comorbid psychiatric disorders may play an important role in the management of CTTHs (Holroyd et al., 2000).

Clinical wisdom suggests that a comorbid mood or anxiety disorder reduces the effectiveness of standard behavioral and drug therapies, but no hard evidence is available to support this impression. *Studies that examine the effectiveness of behavioral treatments for migraine or CTTH in individuals with comorbid anxiety or mood disorders are needed. The possibility that treatment outcomes can be improved in these patients if cognitive-behavioral interventions that have proven effective in the treatment of anxiety and mood disorders (Gould, Otto, Pollack, & Yap, 1997; Robinson, Berman, & Neimeyer, 1990) are incorporated into behavioral treatments for headache deserves attention.*

Psychological Management of Headache

At the time of the 1992 review, three forms of psychological treatment were judged to have received empirical support for the management of tension-type headache: (a) relaxation training (RLX), typically in the form of progressive muscle-relaxation

¹ Berkson's paradox refers to a spurious association that is observed because persons with two or more disorders are more likely to enter treatment than an individual with one disorder (Berkson, 1946).

training; (b) electromyographic biofeedback training (EMG-BF), typically to reduce muscle activity in forehead and often neck and shoulder muscles; and (c) specific forms of cognitive-behavioral therapy (CBT). The addition of CBT to RLX also was judged to increase the effectiveness of RLX.

Two forms of psychological treatment also were judged to have received empirical support for the management of migraine: (a) RLX, typically combined progressive muscle relaxation and autogenic training, and (b) thermal ("hand-warming") biofeedback training (TBF), typically combined with RLX. However, the utility of CBT for migraine was questioned because CBT did not appear to enhance the effectiveness of simpler RLX or TBF therapies. Finally, cephalic vasomotor (temporal artery) biofeedback was judged to be promising largely on the basis of ideas about the vascular basis of migraine that are less convincing now than at that time. This technically complicated biofeedback procedure is rarely used clinically, so it is not considered further, although it continues to receive research attention, particularly in Europe (e.g., Kropp, Gerber, Keinath-Specht, Kopal, & Niederbereger, 1997; Lisspers & Ost, 1990).

Migraine

The movement for empirically based clinical guidelines in medicine (Clinton, McCormick, & Besteman, 1994) and the attention this has brought to psychological treatments for headache may have been the most striking development in the past decade. The first formal guidelines in medicine to recommend the use of specific psychological treatments for migraine—if only as adjunctive therapies for "motivated" patients, or for patients who preferred nondrug therapy—were published by the Canadian Headache Society (CHS; Pryse-Phillips et al., 1997). The CHS guidelines used expert panels to grade the quality of evidence (A = good evidence for use to E = good evidence against use) for each medical and psychological therapy. Evidence supporting the use of RLX, TBF, and CBT were each graded as "fair" (a grade of B), with the primary limitation of the evidence base judged to be the lack of placebo-controlled studies.²

In the United States, the Agency for Health Care Policy and Research (AHCPR)³ commissioned a comprehensive evaluation of evidence for medical, behavioral, and physical treatments for migraine (Goslin, Gray, & McCrory, 1999). Figure 1 presents both the effect size and the percentage reduction in migraine for the four psychological treatments that were judged efficacious in the AHCPR evidence report. Unfortunately, only about half of the available controlled trials (39 of 70) were included in the AHCPR meta-analysis, because behavioral treatment studies often fail to report data necessary for effect size calculations. The small number of studies that were included in the AHCPR meta-analysis may explain the large confidence intervals evident in Figure 1.

The AHCPR evidence report essentially repeats the conclusions of reviews published in the psychological literature for more than a decade.⁴ The significance of this evidence report is thus likely to be more political than scientific: The report provides the type of "official" independent evaluation necessary for psychological therapies to be considered for inclusion in clinical guidelines in medicine. It is thus not surprising that the AHCPR evidence reports provided the stimulus for the formation of the U.S. Headache Consortium, an affiliation of influential medical organizations⁵

created to develop clinical guidelines for the management of migraine. The U.S. Headache Consortium guidelines, published in brief in the journal *Neurology* (Silberstein & Rosenberg, 2000) and in more detail on the American Academy of Neurology Web site (<http://www.aan.com>; Campbell, Penzien, & Wall, 2000) conclude that "relaxation training, thermal biofeedback combined with relaxation training, EMG biofeedback, and cognitive-behavioral therapy may be considered as treatment options for the prevention of migraine." (pp. 16–17)⁶

In spite of the legitimacy that guidelines provide for behavioral interventions, the evidence base for psychological treatment of migraine continues to suffer from two salient limitations. The small scale of most treatment studies (less than 20 patients per treatment group) and the absence of controlled studies conducted in the primary care or specialty neurology settings where recurrent headache disorders are typically treated limit the generalizability and, thus, the acceptance of positive findings by the larger medical community. In addition, it difficult to convincingly rule out the placebo effect as an explanation for positive findings in the absence of placebo-controlled studies.

There are, of course, exceptions to both generalizations. In a *randomized trial* conducted in a pain management clinic, Reich (1989) reported positive results with TBF in 392 migraine sufferers treated with either TBF or one of three multimodal comparison treatments. Also, Blanchard and colleagues (Blanchard, Appelbaum, Radnitz, Morrill, et al., 1990; Blanchard et al., 1997) have used a number of innovative pseudotherapy procedures in controlled studies of TBF. *Nonetheless, larger scale controlled studies (50 subjects or more per treatment group) conducted in medical settings where headache disorders are typically treated would provide valuable information about the effectiveness and transportability of psychological treatments. Ideally, such studies would compare the effectiveness of psychological treatment with an accepted pharmacological treatment and include pill placebo or pseudotherapy controls.*

Psychological treatment will inevitably be influenced by the increasingly important role the *triptans* (drugs such as sumatriptan that abort migraine attacks; Saxena & Tfelt-Hansen, 2000) play in the management of migraine. The high cost of the triptans (e.g., \$14 a tablet or an average of \$21 per migraine attack for sumatriptan; Adelman, Brod, Von Seggem, Mannix, & Rappoport,

² Evidence supporting the use of the best validated preventive drug therapy (beta-blockers) was rated A, and the use of tricyclic antidepressants was rated B. Psychotherapy and hypnosis were unrated because of a lack of supportive evidence.

³ This agency is now the Agency for Healthcare Research and Quality.

⁴ The AHCPR evidence report does draw attention to three small studies ($N_s = 6-12$ per treatment group; Lake, Rainey, & Papsdorf, 1979; McGrady et al., 1994; Reading, 1984) that are interpreted as supporting the effectiveness of EMG-BF for migraine.

⁵ These organizations include the American Academy of Family Physicians, American Academy of Neurology, American Headache Society, American College of Emergency Physicians, American College of Physicians, American Osteopathic Association, and National Headache Foundation.

⁶ In support of this conclusion, multiple well-designed randomized clinical trials, directly relevant to the recommendation, yielded a consistent pattern of findings.

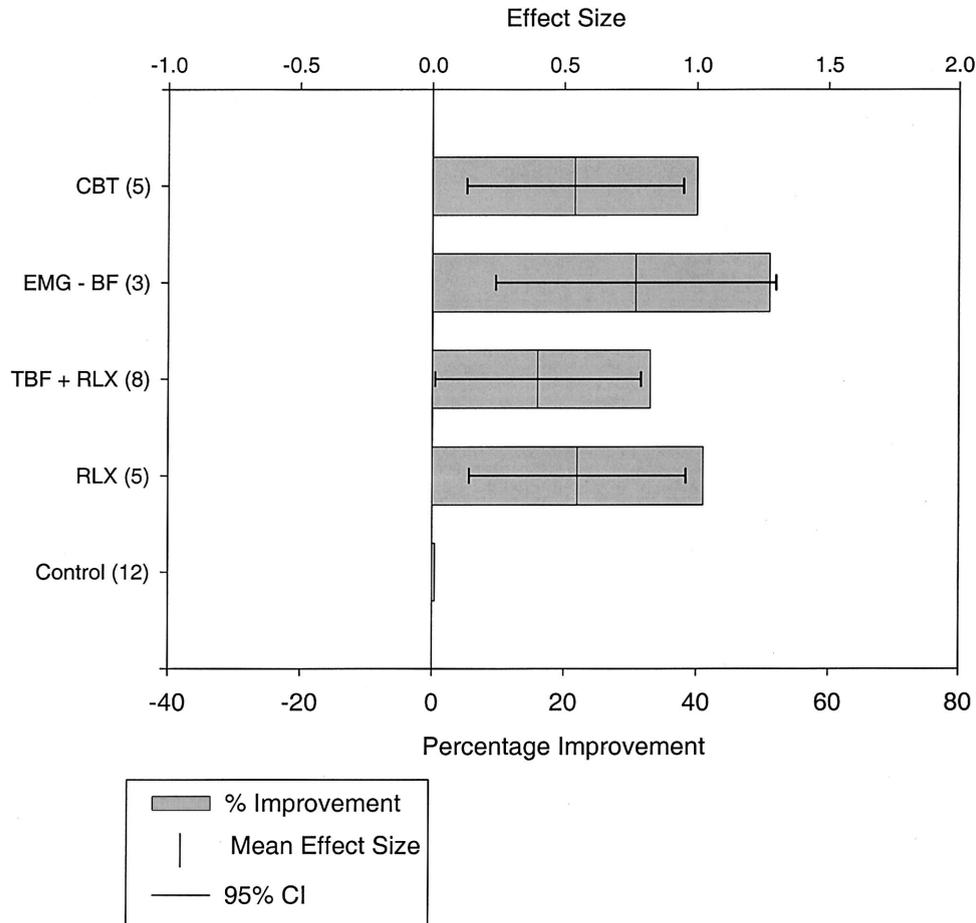


Figure 1. Efficacy of behavioral treatments for migraines: effect size with 95% confidence interval (top axis) and percentage reduction in migraine (bottom axis). CBT = cognitive-behavioral therapy; EMG-BF = electromyographic biofeedback training; TBF + RLX = thermal biofeedback training plus relaxation training; RLX = relaxation training; Control = headache monitoring control. Figure is based on data from Goslin et al. (1999).

1998) has already created a need for interventions to effectively promote the cost-effective use of this medication. In addition, the availability of the triptans has promoted the development of distinct “stratified care” treatment strategies for high-severity migraine (characterized by frequent disabling migraine episodes) and moderate severity migraine (characterized by less frequent and disabling migraine episodes) (Lipton, 1998; Lipton, Stewart, Cady, et al., 2000; Lipton, Stewart, Stone, Lainez, & Sawyer, 2000). *Psychological treatment strategies for high- and for moderate severity migraine need to be similarly differentiated* (Holroyd, Lipchik, & Penzien, 1998). *Psychological treatment of high-severity migraine will need to not only address the management of frequent migraines and associated disability but also help patients use the new triptan therapies cost effectively.*

Tension-Type Headache

Similar effect size and percentage improvement data from the evidence report on the management of tension-type and cervicogenic headache prepared by the Agency for Health Care Research

and Quality are presented in Figure 2 (McCrorry, Penzien, Hasselblad, & Gray, 2001). One can see that the four behavioral treatments have each yielded a 40% to 50% reduction in tension-type headache activity when results are averaged across trials. This evidence report concludes, “Behavioral treatments for tension-type headache have a consistent body of research indicating efficacy” (McCrorry et al., 2001, p.7). As in the evidence report on the treatment of migraine only a portion of the identified trials (35 of 107) could be included in the formal meta-analysis. Nonetheless, additional analyses suggested that overall findings would not change dramatically in this larger pool of trials. Meta-analyses conducted for the evidence report are also consistent with results from earlier, more inclusive meta-analyses (Blanchard, Andrasik, Ahles, Teders, & O’Keefe, 1980; Bogaards & ter Kuile, 1994; Holroyd & Penzien, 1986) that used different statistical techniques.

CBT tailored for treatment of tension-type headache (Holroyd, Andrasik, & Westbrook, 1977; Tobin, Holroyd, Baker, Reynolds, & Holm, 1988) continues to receive research attention. Mosley,

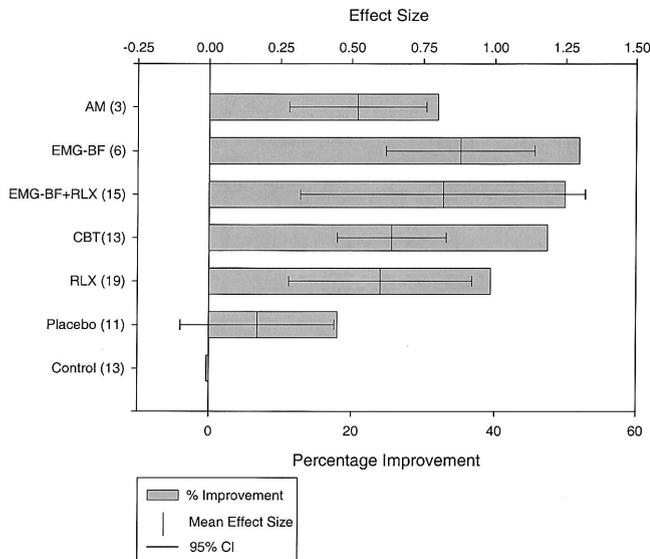


Figure 2. Efficacy of treatments for tension-type headache: effect size with 95% confidence interval (top axis) and percentage reduction in tension-type headache activity (bottom axis). AM = amitriptyline HCl; EMG-BF = electromyographic biofeedback training; EMG-BF + RLX = electromyographic biofeedback training plus relaxation training; CBT = cognitive-behavioral therapy; RLX = relaxation training; Placebo = pseudotherapy or false biofeedback training; Control = headache monitoring control. Figure is based on data from McCrory et al. (2001).

Grotheus, and Meeks (1995) extended to an older (over 65 years of age) patient population the earlier finding (Blanchard, Applebaum, Radnitz, Michultka, et al., 1990; Murphy, Lehrer, & Jurish, 1990; Tobin et al., 1988) that CBT is more effective than RLX alone. Also, in a large study ($N = 203$) focused specifically on CTTH (mean of 26 headache days per month), CBT was more effective than pill placebo and comparable in effectiveness to tricyclic antidepressant medication in reducing headache activity, analgesic medication use, and headache-related disability (Holroyd et al., 2001). *The effectiveness of CBT is relatively well established for both the episodic and chronic forms of tension-type headache. Studies are now needed to evaluate the feasibility of integrating CBT into primary practice and specialty medical settings.*

EMG-BF and RLX received less attention in the past decade than in previous decades, though positive results continue to be reported, particularly with EMG-BF (e.g., Grazi, D'Amico, & Bussone, 1992; Reich, 1989; Reich & Gottesman, 1993; Rokicki et al., 1997). In one of the few randomized controlled studies conducted in a medical setting, Reich (1989) reported positive results and excellent maintenance of improvements over a 2-year period with EMG-BF in 287 tension-type headache sufferers treated with EMG-BF or one of three multimodal comparison treatments. *The effectiveness of EMG-BF and CBT have yet to be compared in the treatment of CTTH. An evaluation of the separate and combined effects of these two treatment modalities with this difficult-to-treat disorder seems warranted.*

Controlled studies of hypnosis have been rare. However, two recent studies (1995; Zitman, Van Dyck, Spinhoven, & Linssen, 1992) reported that the addition of hypnotic instructions failed to improve outcomes obtained with RLX. Spanos and colleagues

(1993) also reported that hypnotic instruction and a pseudohypnosis control intervention yielded similar reductions in headache activity in college student volunteers. These studies are consistent with earlier findings (e.g., Schlutter, Golden, & Blume, 1980) that cast doubt on specific benefit of hypnosis in the management of tension-type headaches. *In spite of these negative findings, additional research to determine the benefits of adjunctive hypnosis in behavioral treatments for headache seems warranted.*

Pediatric Headache

Longitudinal data from the National Birth Cohort Study in Great Britain not only confirmed the relationship between stress (*psychosocial adversity*) and headaches in children but also found that childhood headache predicts recurrent headache problems and other physical and psychiatric complaints in adulthood (Fearon & Hotopf, 2001). Teaching strategies for managing headaches and the stresses that trigger headaches may thus not only ameliorate headaches in childhood but also confer long-term benefits in adulthood.

Tables 4 and 5 provide a selective overview of recent studies that have evaluated psychological treatments for pediatric migraine or tension-type headache and that included at least 10 subjects per treatment group (see also Hermann, Kim, & Blanchard, 1995).⁷ One can see that empirical support continues to accumulate for the effectiveness of RLX, CBT, and TBF. Moreover, positive results have been reported with home-based—even largely self-administered—behavioral treatment. Also, preliminary findings raise the possibility that simple behaviorally based public health interventions in the schools could be of help in preventing the later development of more severe headache problems in vulnerable adolescents.

On the other hand, the lack of pseudotherapy or placebo-controlled studies is a significant limitation of the pediatric literature, because it is likely that children are more responsive than adults to placebo. Data also are too limited to determine whether the addition of a parent intervention to patient-focused behavioral treatment improves treatment outcomes, though this may vary with the age of the patient and the nature of the parent-child relationship. Also, few studies have compared the effectiveness of behavioral and drug therapies (Olness, MacDonald, & Uden, 1987; Sartory, Muller, Metsch, & Pothmann, 1998)—in fact, information about the efficacy of the drug therapies themselves remains surprisingly limited (e.g., Forsythe, Gillies, & McCarran, 1984; Olness et al., 1987).

Larger studies conducted in primary care or pediatric settings are needed, possibly evaluating nurse- or paraprofessional-administered home-based or self-administered treatments that could be practically integrated into busy medical practice settings. Ideally such studies would include a pseudotherapy or placebo control, usual care, or drug therapy comparison group and would provide information about the relative effectiveness of treatments in pre- and postpubertal females. Further pilot evaluations of school-based behavioral interventions to explore their value in both the treatment and secondary prevention of chronic headache disorders also seem warranted.

⁷ By combining these tables with the equivalent table in Blanchard (1992), one can obtain an overview of the pediatric treatment literature.

Table 4
Pediatric Treatment Studies: Clinic- and Home-Based Studies

| Variable | Helm-Hylkema, Orlebeke, Enting, Thussen, and VanRee (1990) | Allen and McKeen (1991) | McGrath et al. (1992) | Labbe (1995) | Hermann, Blanchard, and Flor (1997) | Allen and Shriver (1998) | Mehta (1992) |
|--|--|---|--|--|--------------------------------------|--|-------------------------------|
| Sample size | 20 | 15 | 87 | 30 | 32 | 27 | 18 |
| Age range | 10–19 | 7–12 | 11–18 | 7–18 | 8–16 | 7–18 | — |
| Mean age | — | — | — | 12 | 11.8 ± 2.1 | — | 10.7 |
| Sex ratio (M/F) | 4/16 | — | 24/63 | 17/13 | 19/13 | 11/16 | 11/7 |
| HA type | Migraine | Migraine | Migraine | Migraine | Migraine | Migraine | Recurrent HA |
| Type of study | Comparative group outcome | Multiple baseline | Controlled group outcome | Controlled group outcome | Single group outcome | Comparative group outcome | Single group outcome |
| Treatment condition | 1. CBT/RLX + TBF—winter (8 sessions) 2. CBT/RLX + TBF—summer (8 sessions) | TBF (home-based) + PI (3-, 6-, 9-week baseline) | 1. CBT/RLX—self-administered (8 weeks) 2. CBT/RLX—clinic (8 weeks) 3. UC | 1. TBF + RLX (1 session, 7 weeks) 2. RLX (10 sessions, 7 weeks) 3. WLC | TBF (home-based 4 sessions, 8 weeks) | 1. TBF 2. TBF + pain behavior management | RLX, CBT, PI (10–14 sessions) |
| Outcome measure | HA diary | HA diary | HA diary | HA diary | HA diary | HA diary | HA diary |
| Result (% improvement at end of treatment) | 1. 56.4% 2. 13.7% HA frequency (<i>ns</i> ; <i>p</i> < .08) | 47% HA index | 1. 50.3% 2. 47.8% 3. 5.8% HA index (1 = 2 > 3) | 1. 83.3% 2. 81.1% 3. 24.4% HA index (1 = 2) | 59.6% HA index | 1. 21.4% 2. 63.4% HA frequency (1 < 2) | 86.9% HA frequency |
| % of sample improved | — | 87% (13/15) | 1. 66.7% (16/24) 2. 43.5% (10/23) 3. 24% (6/25) | 1. 100% (10/10) 2. 90% (9/10) 3. 60% (6/10) | 68.8% (22/32) | 1. 46.2% (6/13) 2. 71.4% (10/14) | 33% (6/18) |
| % improved | — | ≥70% | ≥50% | ≥50% | ≥50% | ≥50% | 50–80% |
| Follow-up length | 9–12 months | 8 months | 12 months | 6 months | — | 12 months | 3 months |
| % change (end of treatment to follow-up) | — | Maintained improvement | 1. 36.1% 2. 19.3% 3. — | 1. 90.9% 2. 71.4% 3. 9.7% | — | 1. 51.5% 2. 40% | 38% |

| Variable | Bussone et al. (1998) | Grazzi et al. (1990) | Womack et al. (1988) | Barry and Baeyer (1997) | Griffiths and Martin (1996) | Kroner-Herwig et al. (1998) | Sartory et al. (1998) |
|--|--|-------------------------------|---|--|--|--|--|
| Sample size | 30 | 10 | 119 | 29 | 42 | 50 | 30 |
| Age range | 11–15 | 12–15 | 4–20 | 7–12 | 10–12 | 8–14 | 8–16 |
| Mean age | — | — | 12.5 ± 3.3 | 9 | 11 | 11 | 11.3 |
| Sex ratio (M/F) | 15/15 | 5/5 | 60/59 | 10/19 | 21/21 | 20/30 | 17/13 |
| HA type | Tension | Tension | Tension or migraine | Tension or migraine | Tension or migraine | Tension or tension + migraine | Migraine or tension + migraine |
| Type of study | Comparative group outcome | Single group outcome | Single group outcome | Controlled group outcome | Controlled group outcome | Controlled group outcome | Comparative group outcome |
| Treatment condition | 1. EMG-BF/RLX (10 sessions, 5 weeks) 2. Self RLX (10 sessions, 5 weeks) | EMG-BF (12 sessions, 6 weeks) | EMG-BF and/or TBF + RLX (8 weekly sessions) | 1. CBT/RLX + PI (2 sessions, 2 weeks) 2. UC | 1. CBT/RLX-clinic (8 sessions) 2. CBT/RLX-home-based (3 sessions) 3. WLC | 1. RLX + PI 2. RLX 3. EMG-BF + PI 4. EMG-BF 5. UC | 1. RLX + CBT (10 sessions) 2. Cephalic vasomotor feedback + CBT (10 sessions) |
| Outcome measure | HA diary | HA diary | HA diary | HA diary | HA diary | HA diary | HA diary |
| Result (% improvement at end of treatment) | 1. 54.2% 2. 56.5% HA index (1 = 2) | 88.6% HA index | — | 1. 6.8% 2. 41.8% HA frequency (1 < 2) | 1. 87% 2. 65.4% 3. 13.3% HA index (1 = 2 > 3) | 1. 46.5% 2. 16.6% 3. 45.5% 4. 71.8% 5. 24.1% HA frequency (1 + 2 + 3 + 4)/4 > 5 | 1. 33% 2. 42% HA frequency (1 = 2) |

Table 4 (continued)

| Variable | Bussone et al. (1998) | Grazzi et al. (1990) | Womack et al. (1988) | Barry and Baeyer (1997) | Griffiths and Martin (1996) | Kroner-Herwig et al. (1998) | Sartory et al. (1998) |
|--|-----------------------|----------------------|----------------------|------------------------------------|--|---|-----------------------------------|
| % of sample improved | — | 90% (9/10) | 44.5% (53/119) | 1. 16.7% (2/12) 2. 11.8% (2/17) | 1. 80% (12/15) 2. 62% (9/15) 3. 25% (3/12) | 1. 70% (7/10) 2. 40% (4/10) 3. 60% (6/10) 4. 80% (8/10) 5. 40% (4/10) | 1. 80% (12/15) 2. 53.3% (8/15) |
| % improved | — | ≥50% | ≥50% | ≥50% | ≥50% | ≥50% | ≥50% |
| Follow-up length | 12 months | 12 months | ≥6 months | — | 9 weeks | 6 months | 8 months |
| % change (end of treatment to follow-up) | 1. 86% 2. 60% | 61.8% | — | — | 1. 66.7% 2. 44.4% 3. N/A | 1. 0% 2. 42.9% 3. 41.7% 4. 81.8% 5. N/A | 1. 39% 2. 31% |

Note. HA = headache; CBT = cognitive-behavioral therapy; RLX = relaxation therapy; TBF = thermal biofeedback; PI = parent involvement; UC = unrelated control group; WLC = wait-listed control group; HA index = pain total index scores. Dashes indicate that data are not applicable or were not reported.

Therapeutic Mechanisms

Tension-type headache. The belief that EMG-BF training reduced tension headache activity by enabling individuals to control sustained contractions in pericranial muscles had been challenged at the time of the 1992 review (Andrasik & Holroyd, 1980; Holroyd, Penzien, Holm, & Hursey, 1984). A competing

cognitive-attributional model that emphasized cognitive change as the key therapeutic mechanism in EMG-BF also had received initial support (Holroyd & Penzien, 1983; Holroyd et al., 1984).

Two new studies provide additional support for this cognitive-attributional model. In the first study, false feedback was used to manipulate patients' perceptions of their performance during RLX (Blanchard, Kim, Hermann, & Steffek, 1993). High-success feed-

Table 5

Pediatric Treatment Studies: School-Based Treatment Studies

| Variable | Osterhaus et al. (1993) | Larsson and Carlsson (1996) | Larsson, Melin, and Döberl (1990) | Larsson and Melin (1986) |
|--|---|--|---|---|
| Sample size | 41 | 26 | 48 | 31 |
| Age range | 12–19 | 10–15 | 16–18 | 16–18 |
| Mean age | 14.8 | — | — | — |
| Sex ratio (M/F) | 10/31 | 1/25 | 5/43 | 1/30 |
| HA type | Migraine | Tension | Tension or tension + migraine | Tension or tension + migraine |
| Type of study | Controlled group outcome | Controlled group outcome | Controlled group outcome | Controlled group outcome |
| Treatment condition | 1. CBT/RLX + TBF (8 sessions) 2. WLC | 1. RLX (10 sessions, 5 weeks) 2. UC | 1. Self-RLX (5 weeks) 2. WLC | 1. RLX (9 sessions, 5 weeks) 2. INF (9 sessions, 5 weeks) 3. UC |
| Outcome measure | HA diary | HA diary | HA diary | HA diary |
| Result (% improvement at end of treatment) | 1. 44.1% 2. 3.4% HA index (1 > 2) | 1. 34% 2. 15% HA frequency (1 > 2) | 1. 16.3% 2. 2.0% HA index (<i>ns</i> ; <i>p</i> = .08) | 1. 40% 2. 10% 3. 6% HA frequency (1 > 3) |
| % of sample improved | 1. 45% (14/32) 2. 11% (1/9) | 1. 69% (9/13) 2. 8% (1/13) | 1. 19.4% (6/31) 2. 0% (0/17) | 1. 81.8% (9/11) 2. 7.7% (1/13) 3. 0% (0/7) |
| % improved | ≥50% | ≥50% | ≥50% | ≥50% |
| Follow-up length | 7 months | 6 months | — | 6 months |
| % change (end of treatment to follow-up) | 1. 17.9% 2. — | 1. 6.5% 2. 19.6% | — | 1. 33.3% 2. 6.7% 3. 88% |

Note. CBT = cognitive-behavioral therapy; RLX = relaxation therapy; TBF = thermal biofeedback; UC = unrelated control group; WLC = wait-listed control group; HA = headache; Self-RLX = self-relaxation without instruction or assistance; INF = information contact group; HA index = pain total index scores. Dashes indicate that data are not applicable or were not reported.

back yielded larger improvements (54% reduction) in tension-type headache activity than moderate success feedback (21% reduction), even though behavioral ratings of *actual relaxation behaviors* (Poppen, 1988) during training did not differ in the high-success and moderate success feedback groups. In the second study, combined EMG-BF and RLX produced larger reductions (51% of patients clinically improved) in tension-type headache activity than were observed in untreated controls (15% of patients clinically improved; Rokicki et al., 1997). However, changes in self-efficacy ($r = .43$) but not changes in EMG activity during biofeedback training predicted improvements following treatment.

Studies that examine the mechanisms underlying drug therapies (e.g., Ashina et al., 1999) have begun to assess psychophysiological variables that are hypothesized to reflect the sensitization of trigeminal nucleus circuits. In particular, pericranial muscle tenderness in response to a standardized pressure stimulus has been hypothesized to reflect the sensitization of trigeminal nucleus pain transmission circuits (Bendtsen et al., 1996) and to index a vulnerability to tension-type headaches (Jensen & Olesen, 1996; Neufeld, Holroyd, & Lipchik, 2000). *A study that examines changes in pericranial muscle tenderness, pain detection thresholds, or other indices of CNS sensitization, as well as changes in self-efficacy during EMG-BF, may shed light on the therapeutic mechanisms operating in EMG-BF.*

Migraine. At the time of the 1992 review, efforts to examine therapeutic mechanisms focused primarily on TBF and were guided by the assumption that volitional hand-warming served to stabilize abnormalities in cerebrovascular blood flow postulated to cause migraine (cf. R. J. Mathew et al., 1980). This line of research has continued (e.g., Blanchard et al., 1997; McGrady, Wauquier, McNeil, & Gerard, 1994; Wauquier, McGrady, Aloe, Klausner, & Collins, 1995). For example, Blanchard and colleagues (Blanchard et al., 1997) compared the outcomes produced by four biofeedback conditions: three thermal biofeedback conditions (warming, cooling, and stabilization of hand temperature) and a pseudobiofeedback treatment (feedback for decreasing "alpha" EEG activity). All four biofeedback conditions produced similar small improvements. Unfortunately, home practice was discouraged (rather than encouraged, as is typical), so these findings may not generalize to clinical practice (Blanchard et al., 1991; Gauthier, Cote, & French, 1994). Nonetheless, this study adds to findings that question whether the hand-warming response is causally related to the improvements in migraine achieved with TBF.

Two studies also extended the cognitive-attributional model originally developed to explain therapeutic mechanisms in EMG-BF (Holroyd et al., 1984) to TBF. In the first study ($N = 28$; Blanchard et al., 1994), high- and moderate success performance feedback conditions failed to differentially affect patients' perceptions of their biofeedback training performance, limiting the ability of this study to examine the effect of performance feedback on treatment outcome. However, post hoc analyses including only patients for whom the feedback manipulation had been successful provided support for the cognitive-attributional model, in that performance feedback, but not actual performance (hand warming), predicted improvements in migraine. In the second study ($N = 27$; French, Gauthier, Roberge, & Nouwen, 1997), high- and moderate success performance feedback conditions had a small but statistically significant impact on patients' perceptions of their training performance. However, neither performance feedback (or

associated changes in self-efficacy) nor actual biofeedback training performance (hand warming) predicted improvements in migraine with TBF.

A recent study by Olness and colleagues ($N = 28$; Olness, Hall, Roznecki, Schmidt, & Theoharidies, 1999) illustrates how new neuronal models of migraine could change the focus of research on therapeutic mechanisms in the future. Olness et al. reported that young migraine sufferers (ages 5 to 12 years) who benefited from three sessions of RLX (≤ 1 migraine month following treatment) showed greater reductions in an index of mast cell activation (urinary tryptase levels) than untreated controls. Mast cell activation is of interest in neuronal models of migraine because the activation of mast cells releases vasoactive and noiceptive substances (e.g., nitric oxide, histamine, serotonin) hypothesized to activate trigeminal innervation of the vasculature producing migraine. Although no statistical analyses were reported, my analysis of the authors' data indicates that urinary triptase levels decreased more frequently in relaxation responders (9 of 10) than in untreated controls (4 of 14; $p < .05$) at the 24-week evaluation, but this difference was not significant at the 12-week evaluation. Although the small number of subjects and the large variability in urinary triptase levels across subjects render this finding preliminary, this hypothesis deserves further attention.

Efforts to understand change mechanisms in the behavioral treatment of migraine have yielded few results in the last decade. No study has yet convincingly demonstrated that hand warming is a necessary component of TBF, though this possibility hasn't been excluded. Efforts to identify cognitive change mechanisms in TBF also have been inconclusive. *It may be that the focus on craniovascular mechanisms has been misguided. Studies reporting that psychophysiological measures of neuronal excitability are normalized with successful (drug) therapy (Diener et al., 1989; Maertens de Noordhout et al., 1987; Schoenen et al., 1986; Siniatchkin et al., 1998) and that the products of neuronal activation are altered by RLX (Olness et al., 1999) may provide a more promising model for future research.*

Alternative Treatment Formats

More than a decade ago it was demonstrated that RLX, TBF, and CBT could be effectively administered in a limited contact or home-based treatment format (Haddock et al., 1997; Holroyd, Nash, Pingel, Cordingley, & Jerome, 1991; Jurish et al., 1983; Rowan & Andrasik, 1996; Teders et al., 1984; Tobin et al., 1988). Limited contact treatment format uses a few (e.g., typically 3–4 monthly) clinic visits to introduce RLX, TBF, or CBT skills and to address problems patients encountered in acquiring these self-regulation skills. However, headache-management skills are acquired primarily at home with guidance from home-study materials (typically audiotapes and workbooks) and periodic (e.g., biweekly) therapist phone contacts. *Information about the outcomes obtained with limited contact treatment in medical settings, particularly with headaches that are often unresponsive to treatment—near daily headaches and headaches in patients with comorbid psychological disorders—is needed.*

Efforts to eliminate therapist contact so as to create a completely self-help treatment format have been rare (e.g., Kohlenberg & Cahn, 1981), possibly because self-help treatments lack mechanisms to provide corrective feedback when users encounter prob-

lems in learning, or to maintain the motivation of the user for the months that may be required to learn to use headache-management skills effectively. However, a novel public health intervention in the Netherlands attempted to remedy these limitations of self-help treatment by providing supplementary television and radio instruction (de Bruin-Kofman, van de Wiel, Groenman, Sorbi, & Klip, 1997). Approximately 15,000 participants purchased home-study materials (a workbook and three audiocassettes) that presented a variety of RLX and CBT skills for managing headaches. Each headache management skill was demonstrated in 1 of 10 television programs, and 10 radio programs provided an opportunity for participants to hear solutions to representative problems encountered. Unfortunately, limitations of the program evaluation component of this project, including the assessment of outcome in only a small subsample ($n = 271$) of participants, compromised the program evaluation. Nonetheless, the 164 participants who completed the program evaluation recorded a 50% average reduction in the frequency of headaches and a reduction of about 4.5 days of lost work time over 4 months.

Headache diagnosis, delivery of RLX training materials, and evaluation of treatment outcomes were all conducted over the Internet in an innovative study (Strom, Peterson, & Andersson, 2000). Unfortunately, over half of enrolled subjects dropped out, and the subjects who did complete the RLX treatment showed only modest reductions in headache activity (33% reduction in headache index). These outcomes are what might be expected with self-help workbooks alone, possibly because RLX consisted primarily of a workbook sent via e-mail. *Studies that make greater use of the capacity of the Internet to individualize treatment (e.g., individualizing learning goals and feedback) and to provide social support and assistance (e.g., chat groups) are needed.*

A recent work-site headache management intervention provides one model for such an Internet intervention. Schneider, Furth, Blalock, and Sherrill (1999) used multimedia touch-screen computer kiosks placed at worksites to provide individualized summaries of headache information (e.g., likely triggers, aggravating factors, modified IHS diagnosis based on a computer algorithm) for users and their physicians and, at one site, to provide more generic information on headache management (e.g., biofeedback, coping with headaches, communicating with physicians). Data from participants ($N = 177$) at a 3-month follow-up indicated small but statistically significant reductions in headache days (5.5 to 4 headache days per month) but more impressive reductions in urgent care and emergency department visits for headache (1.74 to 0.42 visits in 6 months). Unfortunately, high attrition (51%) between the baseline and follow-up assessments also compromised this evaluation. Although maintenance of the hardware for the computer kiosks proved a problem, locating the intervention on a Web site could eliminate this problem.

The development of effective, self-help, and worksite interventions to teach behavioral headache management skills is in its infancy, and current evidence for the effectiveness of these interventions is unconvincing. Nonetheless, if future interventions could be shown to do no harm (e.g., by delaying the seeking of needed treatment) and to be even modestly effective, they have the potential to contribute to the development of a public health (population-based) approach to the management of headaches. *New methods of teaching behavioral skills that creatively use the potential of emerging technologies, as in the Schneider et al.*

(1999) study, as well as traditional media, as in the de Bruin-Kofman et al. (1997) study, thus deserve the creative attention of psychologists. In the United States, thorny legal, liability, and professional licensing questions will need to be resolved before headache diagnosis and treatment without clinician contact is likely to be offered over the Internet or mass media. Initially, Internet-based educational and behavioral interventions may therefore need to be evaluated as adjunct therapies in patients already receiving treatment at a "bricks and mortar" clinic.

Behavior Therapy, Hormones, and Headaches

Recurrent headaches, particularly migraines, can be influenced by female reproductive hormones throughout the life cycle—by menstruation, pregnancy, and menopause, as well as by the use of oral contraceptives or hormonal replacement therapy (MacGregor, 1997; Silberstein, 1998). Conventional wisdom holds that such hormonally influenced headaches are less responsive than other headaches to psychological treatment. *However, data on the treatment of menstruation-related migraine are conflicting (Gauthier, Fournier, & Roberge, 1991; Kim & Blanchard, 1992; Solbach, Sargent, & Coyne, 1984; Szekely et al., 1986), and data on the treatment of headaches in postmenopausal women, particularly women on hormone replacement therapy, are lacking (Holroyd & Lipchik, 2000; Silberstein, 1995). Clinicians treating headache could provide a valuable service by providing data on the effectiveness of psychological treatments in these subgroups of patients.*

Behavioral interventions are particularly attractive for the management headaches during pregnancy or breast feeding because, unlike drugs, behavioral interventions should pose no risk to the developing baby. It is thus noteworthy that Marcus and colleagues ($N = 25$; Marcus, Scharff, & Turk, 1995) found that an eight-session multimodal treatment program (RLX, TBF, and physical therapy exercises) produced larger mean reductions in headache activity (81% vs. 33%) in pregnant women than did a four-session pseudotherapy control treatment. Moreover, improvements were maintained throughout the perinatal period and at 3- and 6-month follow-up evaluations (Scharff, Marcus, & Turk, 1996). The small number of subjects and a 4-hr difference in contact time in the treatment and control conditions render conclusions tentative. *Hopefully, these promising findings will encourage other investigators to examine the effectiveness of psychological interventions for the management of headaches during pregnancy.*

Integrating Drug and Psychological Therapies

Two early studies found that TBF combined with RLX compared favorably with the gold standard for preventive drug therapy (propranolol HCl) in controlling migraines (Penzien et al., 1989; Sovak, Kunzel, Sternbach, & Dalessio, 1981); however, contradictory findings favoring preventive drug therapy were reported in one large trial (N. T. Mathew, 1981). CBT for tension-type headache also compared favorably with the gold standard preventive drug therapy (amitriptyline HCl) in one study (Holroyd, Nash, et al., 1991).

Migraine

In the absence of trials that directly compare the effectiveness of drug and psychological therapies, meta-analysis provides the best

method of comparing the effectiveness of these two treatment modalities (Holroyd, 1993). Holroyd and Penzien (1991) compared results reported in 25 preventive drug therapy trials (using propranolol HCl) and in 35 TBF + RLX trials that included over 2,400 patients. Nearly identical outcomes were reported with propranolol HCl and TBF: Each treatment yielded, on average, a 55% reduction in migraine activity, while a pill placebo yielded only a 12% reduction in migraine activity.

Two trials also examined the benefits of combining propranolol HCl with TBF. Holroyd et al. (1995) compared the effectiveness of limited contact TBF alone and when combined with propranolol HCl (60 to 180 mg/day). Propranolol significantly enhanced the effectiveness of TBF on measures of migraine activity, analgesic medication use, and quality of life. Earlier, N. T. Mathew (1981) also found that propranolol HCl (60 to 120 mg/day) increased the effectiveness of a 10-session multimodal biofeedback (EMG-BF and TBF) training package; however, propranolol HCl was more effective than biofeedback training and about as effective as the combined treatment. The high dropout rate (38% of patients) from biofeedback training alone raises the possibility that outcomes were compromised by poor patient compliance.

Clinical trials are needed to provide information about the distinct benefits of psychological and preventive drug therapies for moderate and high-severity migraines. For example, for frequent disabling—high severity—migraines, a trial might ask whether preventive drug therapy, psychological treatment, or combined therapy best adds to benefits achieved with migraine-specific (triptan) medication. For less frequent and disabling—moderate severity—migraine, a trial might ask whether migraine-specific medication or brief home-based psychological treatment is more cost-effective in the long-term management of migraines.

Tension-Type Headache

Two new studies provide information about the benefits of combined psychological and drug therapy for tension-type headache. Reich & Gottesman (1993) examined the benefits of adding amitriptyline (up to 75 mg/day) to an intensive (30-session) multiple-site EMG-BF protocol. The combination of amitriptyline HCl and EMG-BF yielded more rapid improvement in tension-type headache activity than EMG-BF alone; however, beginning at Month 8 and continuing through the 24-month evaluation period, the combined treatment showed no advantage over EMG-BF alone. In fact at the 20- and 24-month observation periods—after withdrawal from amitriptyline HCl—patients who had received EMG-BF alone recorded significantly fewer hours of headache activity than patients who had received the combined treatment.

Holroyd and colleagues ($N = 203$; Holroyd et al., 2001) examined the separate and combined effects of CBT and tricyclic antidepressant medication for CTTHs. Patients received one of four treatments: tricyclic antidepressant medication (amitriptyline HCl to 100 mg/day or nortriptyline HCl to 75 mg/day), medication placebo, limited-contact CBT (three clinic sessions) plus antidepressant medication, or CBT plus placebo. Antidepressant medication and CBT yielded similar reductions in CTTHs, analgesic medication use, and headache-related disability at a 6-month evaluation, but improvements tended to be more rapid in the two antidepressant-medication conditions than with CBT alone (see Figure 3). However, the combined treatment was more likely (64%

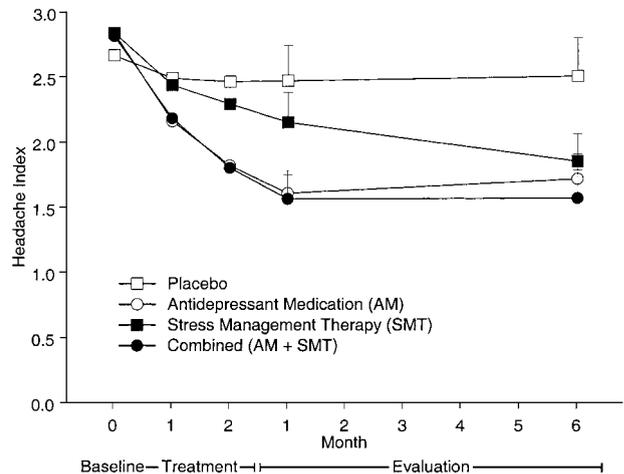


Figure 3. Mean headache index scores at baseline, during treatment (dose adjustment), and (with standard error) at 1-month and 6-month evaluations. Headache index is calculated as a mean of daily headache ratings (range 0–10) over 1-month periods. From “Management of Chronic Tension-Type Headache with Tricyclic Antidepressant Medication, Stress-Management Therapy, and Their Combination: A Randomized Controlled Trial,” by K. A. Holroyd, F. J. O’Donnell, M. Stensland, G. L. Lipchik, G. E. Cordingley, & B. Carlson, 2001, *Journal of the American Medical Association*, 285, p. 2212. Copyright 2001 by the American Medical Association. Adapted with permission.

of patients) to produce clinically significant ($\geq 50\%$) reductions in CTTHs than either antidepressant medication alone (38% of patients) or CBT medication (35% of patients).

The combination of antidepressant medication and CBT appears to be a promising treatment for chronic tension-type headache, particularly for patients who do not respond to one of the individual treatments. Nonetheless, methods of enhancing the effectiveness of this treatment are needed, because many patients continue to experience frequent headaches even following the combined treatment. Information about the long-term treatment outcomes with CTTH are also needed, because CTTH may be more prone to relapse than ETTH, particularly following withdrawal of antidepressant medication. The possibility that CBT or other psychological interventions can help CTTH sufferers successfully withdraw from antidepressant medication also deserves evaluation.

Conclusion

In spite of recent advances in medicine, most individuals with a recurrent headache disorder—even a disorder that impairs functioning—do not have the means to effectively manage their headaches (Adelman, 2000; Dowson & Jagger, 1999; R. B. Lipton et al., 1998); in addition, a third of patients who receive medical treatment for headache problems discontinue treatment because they are dissatisfied with the care they receive (Edmeads et al., 1993). Advances in drug therapy alone are unlikely to remedy these problems. Rather, effective headache management may require that individuals be empowered to manage their own headache problems. Psychological treatment strategies can play a central role in empowering patients, but to do this effectively,

psychological treatment strategies must adapt to developments in the medical and public health sciences, use new information technologies to improve the delivery and the reach of psychological interventions, and rigorously assess the benefits and limitations of psychological interventions in the clinical settings where headache problems are treated. In addition, research on the psychological aspects of headache management will need to capitalize on advances in the biological and medical sciences. Throughout this review I have attempted to highlight promising opportunities for the integration of biological, medical, and psychological science. I encourage readers—both scientists and clinicians—to join in what promises to be an exciting decade of clinical research with recurrent pain disorders.

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