

## Recurrent Pediatric Headache: A Comprehensive Review

Mark Connelly

*Behavioral Pediatrics*  
*University of Kansas Medical Center*

Recurrent pediatric headache is an increasingly common chronic pain syndrome in children and adolescents that is associated with impairments in functioning and quality of life; the condition thus warrants continued clinical and research attention. This article provides a review of the major areas of and developments in pediatric headache research so as to equip pediatric psychologists with a comprehensive knowledge base for this condition. The epidemiology of pediatric headache is reviewed, followed by an overview of clinical features, diagnosis and assessment, etiology, and pharmacological and psychological treatments. Finally, the article concludes with an identification of current trends and areas for further research.

Headaches are a universal feature of the human experience (Annequin, Tourniaire, & Massiou, 2000). It is estimated that general and family physicians are visited 9 million times per year by patients complaining of headache (Solomon, 1997), and recurrent headache is second only to seizure as the most common reason for referral to a pediatric neurologist (Jay & Tomasi, 1981). The societal effect of recurrent headache in terms of the cost of therapy and lost productivity parallels that of many chronic diseases (Breslau & Rasmussen, 2001; Edmeads et al., 1993; Goadsby, 1997; Holmes, MacGregor, & Dodick, 2001; Lipton, Stewart, & von Korff, 1997; Solomon, Skobieranda, & Gragg, 1994). Moreover, individuals suffering from recurrent headaches demonstrate marked impairments in mental health, interpersonal functioning, and general quality of life both during and between headache attacks (Dahlöf & Dimenas, 1995; Solomon, 1997; Stewart & Lipton, 1997).

Most of the extant literature on recurrent headache has focused on adults. However, scientific and clinical attention increasingly has focused on the unique pain

problems of children and adolescents during the past 20 years (McGrath, 2001a). Similar to the condition in adults, recurrent pediatric headache is a pain syndrome that occurs frequently, results in significant episodic functional disability and suffering, produces chronic debilitation, and represents a substantial cost to the patient, the patient's family, and the healthcare system (Holden, Deichmann, & Levy, 1999; McGrath, 2001b; Labbé, 1999; Stang & Osterhaus, 1993). Yet, recurrent pediatric headache is also unique from the condition in adults with respect to features and sequelae and thus warrants study in its own right.

Although the empirical literature on recurrent pediatric headache has increased over the past two decades, attention to this condition lags behind that devoted to other chronic physical illnesses in children and adolescents. This is particularly problematic given substantial evidence indicating that recurrent headaches during childhood are a precursor to potentially severe headache syndromes later in life (Bille, 1981; Hockaday, 1978; Holden, Levy, Deichmann, & Gladstein, 1998; McGrath, 2001b; Metsähonkala, 1998; Sillanpää, 1983) and indications that the overall prevalence of pediatric headache has increased over the past 30 years (Sillanpää & Anttila, 1996). Thus, recurrent pediatric headache remains an important area for research and clinical efforts by pediatric psychologists.

Several reviews in the area of pediatric headache now exist (e.g., Holden et al., 1998, 1999; Jensen, 1999; Karwautz et al., 1999; Lipton, 1997; Martin & Smith, 1995; McGrath & Reid, 1995; Rothner, 1999). However, these reviews tend to focus on a particular area of pediatric headache (e.g., medical treatment, psychosocial etiological factors, or headache assessment and diagnosis). Given the multidisciplinary environment associated with current pediatric practice and the fact that many pediatric practitioners will see children with recurrent headaches as a primary or secondary issue, a comprehensive overview of the status of the literature in this area is needed. The purpose of the present article is to provide a review of the major developments in pediatric headache research so as to equip pediatric practitioners with a comprehensive knowledge base for this condition. This article begins with a review of the epidemiology of pediatric headache, followed by an overview of clinical features, diagnosis and assessment, etiology, and pharmacological and psychological treatments. Finally, the article concludes with an identification of current trends and areas for further research.

## EPIDEMIOLOGY

The most frequently cited study on the epidemiology of pediatric headache was conducted 40 years ago. Bille (1962) conducted interviews with parents of all children between the ages of 7 and 15 residing in Uppsala, Sweden, and found that by the age of 7, 40% of children had already experienced at least one headache episode. In a more recent study, Lipton (1997) reviewed the epidemiology of pediatric

headache and found that headache prevalence by age 7 is 35–51%; this increases to 57–82% by age 15. Thus, the majority of children have experienced headache within the first decade of life.

With respect to recurrent pediatric headache, prevalence rates are estimated to be 25.3 per 1000 (Newacheck & Taylor, 1992). Of these, as many as 40% of children and adolescents report that recurrent headache is a major cause of suffering and disability (McGrath, 2001b). The most common type of recurrent headache in children is migraine (Bush, 1987; Masek & Hoag, 1990). Approximately 1 million children and adolescents have headaches of this type, although prevalence rates vary from 1.4–27% depending on the characteristics of the study population and the diagnostic criteria employed (McGrath, 2001b; Stang & Osterhaus, 1993). Children as young as 2 years old may experience migraine (Barlow, 1994), but the typical onset is 7–12 years of age with increasing prevalence into adulthood (Stewart, Lipton, Celentano, & Reed, 1992). Migraines are equally common in males and females prior to puberty (Masek & Hoag, 1990). Subsequently, more females report migraines by a ratio of about 3 to 2 (Williamson, Baker, & Cubic, 1993), although some epidemiological studies have not supported this finding (see McGrath, 2001b).

The epidemiology of other types of recurrent pediatric headache has been less thoroughly explored. Prevalence estimates for nonmigrainous recurrent headache vary from 6.3% to 49% (McGrath, 2001b). The frequency of pediatric mixed headache or tension-type headache is lowest in children under age 7, and increases to a peak level between the ages of 12 and 18 (Bille, 1962; Labbé, 1998). A recent Finnish study (Anttila et al., 2002) of 1409 schoolchildren found a prevalence rate of 12.2% for episodic tension-type headache, with 15.2% of these children having a history of weekly headache episodes for at least 6 months. Although newer diagnostic categories have recently evolved, such as chronic daily headache (Holden, Bachanas, Kullgren, & Gladstein, 2001; Holden, Gladstein, Trulsen, & Wall, 1994; McGrath, 2001c; Solomon, Lipton, & Newman, 1992), little is yet known about their epidemiology.

## CLINICAL FEATURES

It is thought that the description of headache traces back to Hippocrates about 25 centuries ago, although it was not until 600 years later that Galen coined the term “hemicrania” to describe the type of headache now generically referred to as migraine (Rothner, 2001). Headaches in children were distinguished from those of adults as early as the 16th century (e.g., Tissot, 1780). It was not until recently, however, that headaches in childhood have received independent study.

Children generally describe headache pain as throbbing or aching, which typically corresponds to a diagnosis of migraine or tension-type headache, respec-

tively (Labbé, 1998). In pediatric migraine, children typically experience a throbbing or pulsating pain accompanied by nausea, vomiting, or abdominal pain (Masek & Hoag, 1990). The pain is typically localized in the frontal and temporal regions and may be unilateral, but is usually bilateral (McGrath, 2001b). Children generally describe pain from a migraine headache as moderate to severe and report that the pain can be relieved to some extent by sleep and rest. Migraine headaches in children can last a few hours to most of the day, typically occur two to eight times per month, and demonstrate a seasonal pattern in which fewer headaches are reported during the summer (Labbé, 1998). This latter point is important in that this finding is a potential history confound for treatment studies.

A subgroup of children experience recurrent abdominal pain with vascular changes (e.g., flushing and loss of appetite) similar to symptoms of migraine. In this subgroup, however, there is an absence of head pain. This condition has been referred to in the literature as “abdominal migraine” (Symon & Russell, 1986). These children tend to develop recurrent migraine headaches later in life, and thus being alert for symptoms indicative of abdominal migraine is important for pediatric practitioners.

Another form of recurrent headache in children has been labeled *muscle-contraction headache* or *tension-type headache* (both terms are generally used interchangeably). The clinical features of this type of headache include a frequent tightness around the head and pain localized around the back of the head and shoulders as well as the frontal region. The pain tends to increase during the day and is often relieved through massage and stretching (Labbé, 1998). Pain associated with this type of headache is generally described as less severe than that of migraine (Rothner, 2001), although the two types of headache can co-occur in a “mixed” headache syndrome.

A relatively new category of headache, chronic daily headache, has been identified in the past decade and has been thought to create more functional disability than migraine (Gladstein & Holden, 1996; Holden et al., 1994, 2001; Matthew, Reuveni, & Perez, 1987; McGrath, 2001c; Silberstein, Lipton, Solomon, & Matthew, 1994; Solomon et al., 1992). Chronic daily headache has as its defining feature the presence of daily or near-daily headaches that vary in duration, intensity, and accompanying symptoms and occur in the absence of organic pathology. This condition in children has been found to differ from its counterpart in adults (Gladstein & Holden, 1996; McGrath, 2001c). Specifically, chronic daily headache usually evolves from migraine or medication overuse in adults, whereas in children the condition appears to be characterized by a pattern in which tension headaches occur daily and migraine headaches emerge concomitantly.

Information on childhood presentations of headache typically seen in routine pediatric practice has not been reported empirically to my knowledge. In general, pediatric practitioners are most likely to encounter children presenting with migraine headache given that the associated symptoms may cause concern in the par-

ents. A diagnosis of migraine is typically indicated by the child reporting a throbbing head pain above the eyes or around the ears. The child often also reports nausea or vomiting, a desire to rest or sleep, and hypersensitivity to lights and sounds. Tension-type headache may be less commonly seen in clinical practice because parents may not believe the symptoms warrant medical attention. Nevertheless, tension-type headache presentations are still encountered frequently in pediatric practice and may come up as a secondary issue in therapy for certain conditions (e.g., anxiety, depression, or attention-deficit hyperactivity disorder). A diagnosis of tension-type headache is often indicated by the child reporting a squeezing pain toward the back of the head in the absence of vomiting or sensitivity to lights or sounds. Parents may attempt to treat tension-type headache with over-the-counter analgesics. If used frequently, analgesics can have the unfortunate consequence of actually inducing more headaches (i.e., “rebound headache”). Thus, it is important for practitioners to inquire about headaches in children to avert a potentially vicious cycle.

Other types of headache that may be encountered by pediatric practitioners include headache associated with temporomandibular joint dysfunction (TMJ), headache secondary to an underlying condition causing internal pressure (e.g., benign arachnoid cyst), or cluster headache (i.e., attacks of unilateral orbital or temporal pain occurring as many as eight times per day for a series of weeks or months followed by a remission period; Maytal, Lipton, Salomon, & Shinnar, 1992; Rothner, 2001). However, types of pediatric headache other than migraine and tension-type headache are encountered far less frequently in pediatric practice. As such, pediatric migraine and tension-type headache will be the primary focus of the remainder of this article.

## DIAGNOSIS AND ASSESSMENT

Historically, headaches have been dichotomized into migraine or tension-type (muscle-contraction) headaches based on the presence of autonomic nervous system symptoms (e.g., nausea, vomiting, photophobia, etc.) and the quality of the pain (e.g., pulsatile vs. squeezing or band-like). Much of the early research supporting this distinction was done on the adult population, however, and failed to consider pediatric presentations. More recently, researchers have proposed a continuum model of headache based on severity (or some other dimension) rather than trying to differentially categorize migraine and muscle-contraction headaches (see Holden et al., 1998; Rapoport & Sheftell, 1996). The severity model is supported by data indicating that as headache increases in severity, so do the number of vascular symptoms (e.g., weakness, flushing, or transient visual disturbance; Anttila et al., 2002; Joffe, Bakal, & Kaganov, 1983). However, others still argue for the utility of the differentiation between migraine and muscle-contraction headache (e.g.,

Blanchard, 1992), and many assert that treatment is most effective when matched to a specific headache type.

One of the first classification systems for pediatric migraine was established by Vahlquist (1955) and is still used by many researchers. Within this system, migraine episodes must have a paroxysmal onset, include at least two of the symptoms of nausea, visual scotomata (e.g., absence of vision within the visual field), or one-sided pain, and must be separated by headache-free intervals. The Ad Hoc Committee on the Classification of Headaches (1962a, 1962b) developed a more formalized classification of pediatric migraine that was essentially identical to the Vahlquist criteria: paroxysmal headaches separated by symptom-free intervals and accompanied by at least two of unilateral pain, nausea or vomiting, visual aura in connection with headache, and family history of migraine. Subsequent clinical observations indicated that childhood migraine is often bilateral, may not be accompanied by nausea or vomiting, and is rarely preceded by visual aura (Holden et al., 1998, 1999). Further modifications have thus been proposed (see Prensky, 1976; Prensky & Sommer, 1979), and new diagnostic criteria have been outlined for pediatric headache (see Table 1).

The Headache Classification Committee of the International Headache Society (IHS; 1988) later proposed new operationalized criteria for migraine with and without aura and episodic tension-type headache (see Table 1). However, the new criteria were established primarily for adults and have been found to produce poor sensitivity for diagnosing headaches in children (Maytal, Young, Schechter, & Lipton, 1997). Several researchers have proposed changes to the IHS criteria, arguing that headache duration and the number of autonomic symptoms required to confirm diagnoses need to be altered (Gladstein, Holden, Peralta, & Raven, 1993; Maytal et al., 1997). Abu-Arefeh and Russell (1994) concluded that the IHS criteria were adequate for 5–15-year-olds and adolescents with the exception of requiring the adjustment of the minimum acceptable duration of headache from 2 hours to 1 hour. Winner and colleagues (Winner, Wasiewski, Gladstein, & Linder, 1997) further improved the sensitivity of the IHS migraine criteria from 66% to 93% by making duration, location, and symptom requirement changes (see Table 1). Still, some (e.g., McGrath, 2001b) have argued that proposed modifications to date are insufficient for infants and very young children, and that criteria should include other symptoms such as irritability, head banging, sleep disturbance, behavioral disturbance, abdominal pain, and pallor.

With respect to diagnostic criteria for what has historically been labeled tension-type headache (or muscle-contraction headache), the IHS criteria (Headache Classification Committee, 1988) are often used without modification for pediatric cases (see Table 1). Tension-type headache in children has not received as extensive investigation, nor has there been as much debate regarding diagnostic criteria modifications, as compared to pediatric migraine. However, there is still some indication that the IHS criteria for tension-type headache are not sensitive or specific

TABLE 1  
Diagnostic Criteria for Recurrent Pediatric Headache

<i>Headache Type</i>	<i>Prensky &amp; Sommer (1979)</i>	<i>IHS Criteria (1988)</i>	<i>Winner et al. (1997)</i>
Pediatric migraine without aura (formerly “common migraine”)	Throbbing and pulsating pain	At least five attacks fulfilling the criteria below	At least five attacks fulfilling the criteria below
	Unilateral or bilateral pain in frontal or temporal region Relief after rest	Headache attack lasting 2 to 48 hr  Headache has at least two of the following: unilateral location, pulsating quality, moderate to severe intensity, and aggravation by routine physical activity	Headache attack lasting 1 to 48 hr  Headache has at least two of the following: bilateral (frontal/temporal) or unilateral location, pulsating quality, moderate to severe intensity, aggravation by routine physical activity
	Accompanied by photophobia (acute sensitivity to lights), nausea, or vomiting	During headache, at least one of the following: nausea or vomiting, photophobia and phonophobia (acute sensitivity to lights and sounds, respectively)	During headache, at least one of the following: nausea or vomiting, photophobia or phonophobia
Pediatric migraine with aura (formerly “classic migraine”)	Throbbing and pulsating pain	At least two attacks fulfilling the following	At least two attacks fulfilling the following
	Unilateral or bilateral pain in frontal or temporal region	At least three of the following: one or more fully reversible aura symptoms indicating focal cortical or brain stem dysfunction; at least one aura developing gradually over time and lasting for more than 4 mins, or two or more symptoms occurring in succession; no auras lasting more than 60 min; headache follows in less than 60 min	At least three of the following: one or more fully reversible aura symptoms indicating focal cortical or brain stem dysfunction; at least one aura developing gradually over time and lasting for more than 4 min, or two or more symptoms occurring in succession; no auras lasting more than 60 min; headache follows in less than 60 min

(continued)

TABLE 1 (Continued)

<i>Headache Type</i>	<i>Prensky &amp; Sommer (1979)</i>	<i>IHS Criteria (1988)</i>	<i>Winner et al. (1997)</i>
	Relief after rest Accompanied by photophobia, nausea, or vomiting Also accompanied by prodromes (dizziness, scotomata, or flashing lights)		
Muscle-contraction headache (tension-type headache)	Band-like and constant dull aching pain  Bilateral or back of head or neck  No relief after rest Responds to massage	At least 10 previous headache episodes fulfilling criteria below  The number of days with such headache is less than 15 per month Headache lasting from 30 min to 7 days At least two of the following: pressing or tightening pain quality, mild or moderate pain intensity, bilateral location, no aggravation of pain by walking stairs Both of the following: no nausea or vomiting, absence of photophobia and phonophobia (or one but not the other is present) History and examination does not suggest organic disorder, or one is present but headache does not occur for the first time in close temporal relation to the disorder	na (only updated migraine criteria)

to pediatric presentations of this condition (Anttila et al., 2002; McGrath, 2001b). New IHS diagnostic criteria are currently under development and review.

Although no formalized diagnostic scheme yet exists for the new headache category of chronic daily headache, Silberstein and colleagues (1994) specified diagnostic criteria for four subtypes of chronic daily headache: transformed migraine, chronic tension-type headache, new daily persistent headache, and hemicrania continua. This typology is generally the one used in studies of chronic daily headache. All types of chronic daily headache share the presence of daily or near-daily headaches (5 or more days per week) that vary in duration, intensity, and accompanying symptoms (Solomon et al., 1992). Gladstein and Holden (1996) later added the category of “comorbid migraine” as a special case of chronic daily headache in children and adolescents. The symptom criteria for each of these subtypes are given in Table 2.

Other classification systems in addition to those reviewed exist, although these are less commonly used in the literature. For example, Rothner (1978, 1999, 2001) classifies headache by plotting the course of a patient’s headache condition over time against its severity. This system results in five types of headache, including acute, acute recurrent, chronic progressive, chronic nonprogressive, and mixed. Chronic progressive headache, defined as worsening in frequency and severity over time with symptoms of increased intracranial pressure, is an important differential diagnosis given that concerned parents of child or adolescent headache sufferers often attribute the cause of recurrent headaches to an organic source. The presence of serious organic pathology may

TABLE 2  
Definitions of Chronic Daily Headache Categories

<i>Transformed Migraine</i>	<i>Chronic Tension-Type Headache</i>	<i>New Daily Persistent Headache</i>	<i>Hemicrania Continua</i>	<i>Comorbid Migraine</i>
Duration of more than 4 hr per day	More than 180 headache episodes per year	Abrupt onset of head pain on a daily basis	Headache on a daily basis for at least 1 month	Daily tension-type headache
Increasing severity	Absence of autonomic symptoms	More than 4 hr per day	Unilateral pain of moderate severity	Intermittent and less frequent episodes of migraine
	Pressing or squeezing and bilateral pain	No history of migraine or tension-type headache	No apparent precipitation	
	Transformed from episodic tension-type headache			

*Note.* The information in this table is based on an article by Gladstein and Holden (1996).

be implicated by the following signs and symptoms associated with headache episodes: explosive onset, marked severity, confusion, lethargy, fever, seizure, and neck stiffness (Hämäläinen, 1998a; Masek & Hoag, 1990; Rapoff, Walsh, & Engel, 1988). Other differential diagnoses for migraine and muscle-contraction headaches also need to be considered, including headache caused by fever, convulsive states, or hypoxia, headache related to a sinus condition or a visual refractive error, and headache as a manifestation of primary psychopathology. Headache secondary to depression and other psychological conditions (e.g., generalized anxiety disorder or attention deficit hyperactivity disorder) is often reported clinically, but little empirical information is available on the connection between "primary" psychological conditions and headache in children (Egger, Angold, & Costello, 1998). Reports in the adult literature (e.g., Breslau et al., 2000) suggest three possible relationships: headaches lead to the development of a psychological condition (e.g., depression), a psychological condition leads to the development of headache through some mechanism, or a common factor leads to the development of both headache and a psychological condition. More work in articulating the links between psychological conditions and headache is indicated.

Despite the voluminous literature on diagnostic issues in adult headache, most individuals with migraine headache make a self-diagnosis and never seek medical advice (Goadsby, 1997; MacGregor, 1997). This is probably different in pediatric cases given that, as mentioned, many parents are concerned that recurrent presentations of headache in their child are indicative of organic pathology. Nevertheless, it is possible that some parents will never take their child for a diagnostic work-up, especially in cases of milder recurrent headache or family history of headache. Another important factor in diagnosis is that lack of verbal ability in younger children complicates assessment. Moreover, developmental changes in conceptualizing pain (see Marcon & Labbé, 1990) may contribute to the poor reliability of diagnosis documented in the pediatric literature.

Headache diagnosis is most accurately rendered after a thorough assessment of features of headache episodes themselves as well as events surrounding the episodes. History of headache activity, a complete description of the pain, and an exploration of the antecedents and consequences of the pain behavior are the minimum requirements for an assessment and are usually derived from an interview with the child and parent. Psychological, behavioral, and emotional functioning of the child and family are often evaluated as well. Given the flaws of retrospective reporting, an often-used measure of assessment is the headache diary (e.g., Blanchard & Andrasik, 1985). This generally requires children to rate headache activity on a daily basis, often several times per day, using a scale from 0 (*no headache*) to 5 (*intense and incapacitating headache*). From this diary, one can derive frequency, duration, peak intensity, and headache-free days to aid in diagnosis and treatment conceptualization.

## ETIOLOGY

The pathophysiology of headache remains poorly understood. Most childhood headaches are not caused by an underlying disease or disorder but rather are typically related to cognitive, behavioral, and emotional factors (Andrasik et al., 1988; McGrath, 2001b). This was recognized even a century ago: "Headaches in the young are for the most part due to bad arrangements in their lives" (Day, 1873, p. 156). A comprehensive model of etiology, however, needs to account for factors on several levels (e.g., genetic, biological, cognitive, and behavioral) and needs to differentiate etiology of the initiation of a headache episode from the development of a recurrent headache syndrome.

The etiology of migraine has received far more attention than has that of any other form of recurrent pediatric headache. Yet, no definitive pathophysiology of migraine has been identified (Labbé, 1998). On a genetic level, there is a large body of evidence supporting a substantial genetic contribution (around 40–50% concordance in twin studies), with the contribution being stronger for females and the phenotypic expression being moderated by environmental factors (Barlow, 1984; Honkasalo et al., 1995; Larsson, Bille, & Pederson, 1995; Prensky, 1976; Scheller, 1995; Ziegler, Hur, Bouchard, Hassanein, & Barter, 1998). Migraine has been traced to three different gene loci on chromosomes 19p, 1q21–23, and 1q31. Cases linked to 19p are associated with mutations of a brain-expressed calcium channel subunit, which can lead to a disorder of ion channel regulation (Levin, 2001). Findings such as these can be used to generate explanations of migraine etiology both in terms of the initiation of a headache episode as well as the development of a recurrent syndrome.

The genetic level for tension-type headache has been investigated far less. However, studies that have included a tension-type headache sample have generally arrived at the same conclusion as in studies of migraine. Specifically, tension-type headache also appears to have a strong genetic basis, with the phenotypic expression being modified by environmental factors (Honkasalo et al., 1995; Larsson et al., 1995). No data are yet published on associations of tension-type headache with specific gene loci.

On a biochemical level, there are several potential mechanisms for producing headache. Traction on vascular structures, dilatation or inflammation of cranial vascular structures, displacement of intracranial contents by tumor, increased intracranial pressure, direct pressure on cranial nerves, sustained contraction of head and neck muscles, and pathologic processes outside of the head all may result in pain referred to the head (Annequin et al., 2000; Rothner, 2001). Pain from extracranial and intracranial structures from the front half of the skull are mediated via the fifth cranial nerve (the trigeminal nerve), and thus much of the focus in headache has been on the trigeminal system.

There are two main biochemical theories of migraine pathophysiology. The vascular theory posits that migraine is a disorder of cerebral and extracranial

blood vessel regulation, in which circulating vasoactive amines cause cerebral arteries to constrict, and this in turn causes some vessels of the scalp to dilate. The result is pressure in surrounding tissue and inflammation, leading to the symptoms of migraine headache (Hämäläinen, 1998a; Labbé, 1998; Markowitz, Saito, & Moskowitz, 1988; Masek & Hoag, 1990; Moskowitz, 1984; Saeed, Pumariega, & Cinciripini, 1992). This theory predominated the early understanding of migraine but is currently regarded as an incomplete explanation for the full range of migraine symptoms.

The other main biochemical theory of migraine pathophysiology is the neurogenic model. Proponents of this model posit that migraine results from instability of monoaminergic transmission that renders patients highly vulnerable to sudden changes in internal or external states (Hämäläinen, 1998a; Saeed et al., 1992). Certain factors (e.g., alterations in circadian rhythm, stressors, etc.) are thought to impact on brain stem nuclei that project throughout the cerebral cortex. This induces a phase of sympathetic discharge followed by monoamine depletion. The depletion of monoamines in turn leads to the constriction of intracranial blood flow and dilation of extracranial vasculature, ultimately resulting in focal neurological symptoms and head pain (Saeed et al., 1992). Based on the discovery that serotonin agonists could alleviate an acute attack of migraine (Goadsby, 1994), the neurogenic model has increasingly focused on the depletion of a specific monoamine neurotransmitter, namely serotonin (5-HT). It is now believed that a depletion of serotonin in specific locations throughout the cerebrum and the consequent cascade of prostaglandin, substance P, histamine, bradykinin, and  $\gamma$ -aminobutyric acid leads to the symptoms of migraine headache (Holden et al., 1998). Given that the actions of serotonin are mediated by its receptors, further specificity for the role of serotonin in migraine has been achieved through studying the functions of serotonin receptor groups and subgroups. Most researchers (e.g., Goadsby, 1994; Levin, 2001; Rapaport & Sheftell, 1996; Rothner, 2001; Saeed et al., 1992) believe that depletion of serotonin at the 5-HT<sub>1D</sub> receptor, and perhaps at the 5-HT<sub>1A</sub> receptor, is critical in migraine pathophysiology. Current biochemical models incorporate aspects of both the vascular and neurogenic theories (e.g., Moskowitz 1991, 1992). Thus, migraine attacks are thought to start as a neuronal disorder in the brainstem, which leads to the release of neurotransmitters and other vasoactive substances that cause cerebral vasoconstriction and extracranial vasodilation.

The majority of biochemical models of migraine have been formulated to explain adult presentations. Although it is probable that findings from the adult population generalize to some extent to pediatric and adolescent cases, there may be different mechanisms operating across the lifespan. Indeed, the consistent finding of differences between the symptom presentations of pediatric and adult migraine seems to argue for potentially different mechanisms, including mechanisms operative at the biochemical level. It is possible that a “kindling” mechanism is opera-

tive in migraine, in which repeated episodes of migraine headache initially engendered by external events (e.g., stressors) may sensitize the system on a biochemical or even genetic level such that subsequent presentations of migraine are qualitatively and quantitatively distinct from initial episodes (Connelly, 2003; Post & Silberstein, 1994). The notion of kindling is derived from the epilepsy literature (e.g., Adamec, 1990; Post, 1992; Teskey & Cain, 1989; Weiss & Post, 1998) but may also apply to headache given that there is evidence to suggest a strong association between migraine and epilepsy (Lipton, 1997; Ottman & Lipton, 1996). Thus, biochemical explanations of migraine derived from adult presentations may not generalize to children due to potential changes in migraine pathophysiology across the lifespan.

With respect to the biochemical level of explanation for tension-type headache, knowledge remains limited. One potential reason for this is that no precise definition of tension-type headache existed prior to the IHS criteria in 1988. Also, investigators have often assumed that because mental stress and tension are frequently reported precipitants of this type of headache, these factors must be the cause. It is true that research to date has failed to demonstrate associations with substance P, neuropeptide Y, vasoactive intestinal peptide, serotonin, plasma lactate, and pyruvate levels (Levin, 2001). What has been generally (albeit not unequivocally) supported is the finding of increased pericranial muscle tenderness and altered texture (Jensen, 1999; Sakai, Ebihara, Akiyama, & Horikawa, 1995); this implicates a peripheral mechanism (i.e., activation of peripheral nociceptors). More recently, focus has turned toward the central nervous system due to evidence suggesting a sensitization effect (i.e., lower pain thresholds) in pain relay systems in the brain and spinal cord (Bendsten, Jensen, & Olesen, 1996; Holroyd, 2002; Jensen, 1999). In an excellent recent review of studies on the pathophysiology of tension-type headache, Jensen and Olesen (2000) concluded

The underlying pain mechanisms in tension-type headache are highly dynamic, as tension-type headache represents a wide variety of frequency and intensity, not only between individuals, but also within the individual subject over time. The initiating stimulus may be either a condition of mental stress, nonphysiological motor stress, a local myofascial release of irritants or a combination of these. Secondary to the peripheral stimuli, the supraspinal pain perception structures may be activated, and because of the central modulation of the incoming stimuli, a self-limiting process will be the result in most individuals. (p. 286)

Again, it is important to note that biochemical explanations of tension-type headache are generally directed toward the adult population and may not generalize to tension-type headache etiology in children and adolescents.

Early life factors have also been examined for potential etiological roles in the development of headache in general, although only one study has evaluated this hypothesis systematically. Aromaa, Rautava, Helenius, & Sillanpää (1998) conducted a longitudinal case-control study in Finland and followed 1,443 young fam-

ilies expecting their first baby until the child reached the age of 6. These researchers found that frequent headache in the mother before pregnancy, proteinuria during pregnancy, maternal assessment of poor health and feeding problems in her child at the age of 9 months, and depression and sleeping difficulties at the age of 3 years predicted later headache. Other significant predictors measured when the child was 5 years old included nocturnal enuresis, travel sickness, concentration difficulties, behavioral problems, unusual tiredness, and high sociability. Although this study is useful with respect to elucidating factors associated with headache, these factors should not be interpreted as necessarily being causal.

The psychological level of headache etiology has received a great deal of empirical attention both in the adult and pediatric literature. In general, models of psychological etiology focus on the development and maintenance of recurrent headache rather than the initiation of individual episodes. Differential psychological etiology across headache type (e.g., migraine and muscle-contraction headache) is rarely delineated, although for some time the concept of a "migraine personality" was accepted as the cause of migraine. Specifically, children and adolescents who were "over-achievers," conscientious, and perfectionist were thought to be more prone to developing migraine than children who were in the normative range on these attributes. However, this notion generally lacks empirical support (Masek & Hoag, 1990; McGrath, 2001b), with the exception that children with migraine headache may work longer on homework and may have an increased fear of academic failure relative to those who do not develop recurrent migraine headache (Martin & Smith, 1995).

Several studies have examined differential psychological profiles as a function of headache status, with mixed conclusions. Anxiety, depression, or psychological conflicts have historically been viewed as potential causes of chronic headache (Andrasik et al., 1988; Cunningham et al., 1986; Lanzi, Balottin, Borgatti, Guderzo, & Scarabello, 1988). More recently, this perspective has had to be modified in light of new findings. For example, autonomic arousal, finger temperature, vasomotor responses, and subjective stress sensitivity were not found to discriminate between children with migraine and healthy controls (Hermann & Blanchard, 1998). Also, frequency of negative life events has not been found to differentiate children and adolescents with recurrent headache from those without this condition (Martin & Smith, 1995). Labbé (1998) drew attention to the important point that all of the studies on psychological differences between those with and without recurrent pediatric headache have been on those children whose parents were seeking treatment for them; thus, the sample is not entirely representative of children and adolescents with recurrent headache. Finally, headache group scores on psychological assessment instruments have almost always been found to be within normal range (Labbé, 1998; McGrath, 2001b), and slight elevations observed may be secondary to living with the pain condition rather than being of etiological significance.

The conclusion based on these findings is that what appears to be an important factor in the development of recurrent pediatric headache on a psychological level is the failure to resolve normal childhood stressors associated with academic, social, and physical activities (Holden et al., 1994; McGrath, 2001b; McGrath & Hillier, 2001a). Ineffective coping skills may be learned through modeling of parental coping approaches (Mikail & von Baeyer, 1990), and secondary gain for reporting pain plays a role in maintaining the headache activity (White, Alday, & Spirito, 2001). McGrath and Hillier (2001a) proposed the following model: A stressful situation occurs that does not get resolved and leads to increased anxiety, which in turn leads to the development of a headache attack. The child is thereby removed from the situation and provided with temporary stress reduction until another stressful situation occurs; the cycle then repeats itself. Factors that interact in this model include child factors (e.g., age, gender, cognitive level, pain experience, family learning, etc.), cognitive factors (e.g., beliefs about headache etiology, beliefs about pain control, and beliefs about the role of stress), behavioral factors (e.g., child and parent behavior during the attack and parental behavior in response to repeated attacks), and emotional factors (e.g., situation-specific stress, anxiety, fear about an undiagnosed condition, frustration regarding disruption to activities, etc.). A similar model was formerly introduced into the adult literature by Martin (1993) and thought to be adaptable to pediatric presentations (Holden et al., 1998).

Certain environmental, physical, emotional, and psychological stimuli have generally been thought to provoke specific headache attacks, especially in migraine presentations. Frequently reported “triggers” include foods (e.g., soda, chocolate, eggs, nuts, cheese, wheat products), environmental conditions (e.g., heat, high humidity), certain sensory stimuli (e.g., bright lights, noise), and certain behaviors (e.g., physical activity, doing schoolwork). Although such triggers are anecdotally reported and are clinically appealing as a starting point for treatment, almost no scientific evidence exists supporting the notion that specific stimuli trigger headache attacks in children. This has led to the interesting speculation that neutral stimuli eventually *can* provoke a headache attack, but only because children are anxious about them (McGrath & Hillier, 2001a). Specifically, parents may search a child’s environment for causes of headache, become convinced that certain things trigger the child’s headache and try to have the child avoid them. If children cannot avoid them, they approach with apprehension and anxiety. It is then this apprehension that results in headache and convinces the parent or child, or both, that the stimuli are a cause of headache. Thus, triggers in pediatric headache may be largely learned.

In short, the etiology of recurrent pediatric headache remains to be fully understood. It is clear that a model of headache etiology needs to account for genetic, biochemical, emotional, cognitive, and behavioral factors. A comprehensive model must also postulate how these various factors interact with one another to cause individual headache episodes and the development of recurrent pediatric

headache. Figure 1 represents the current understanding of the etiology of pediatric migraine and tension-type headache. The top left part of the model represents the biochemical pathways that produce migraine headache, and the bottom left part of the model represents the biochemical pathways that produce tension-type headache. The right part of the figure represents the common person and environment factors that interact with the biological pathways to initiate and maintain headache syndromes. An inherited biological predisposition (e.g., problems with ion channel regulation in the case of migraine, or sensitive pericranial muscles in the case of tension-type headache) renders certain individuals vulnerable to the influence of environmental stressors that, in the absence of adequate coping mechanisms, can induce the physiological changes necessary to engender a headache episode. Headaches may then become recurrent via neurobiological pathways, in which headache episodes beget future episodes through a kindling mechanism

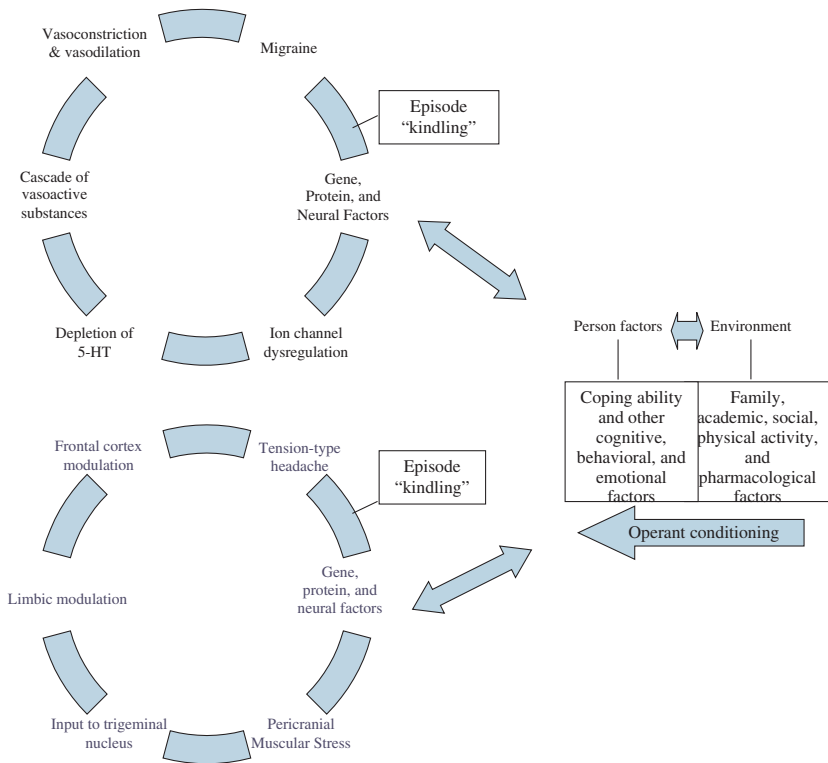


FIGURE 1 Model of migraine and tension-type headache etiology.

(e.g., sensitization of headache pain pathways due to repeated headache episodes or due to repeated administration of short-acting analgesic medication). Headaches may also become recurrent via behavioral pathways, in which headache pain behaviors may be reinforced through operant conditioning. Recurrence can be represented in the figures by envisioning the “cycling” of the left side of the figures. Other factors that moderate headache recurrence include person variables (e.g., coping style, emotional state) and environmental factors (e.g., family supports, academic stressors). A comprehensive understanding of recurrent pediatric headache is of critical importance in clinical applications, in that children and families are best served when they have at least a rudimentary understanding of the headache condition.

### PHARMACOLOGICAL TREATMENT OF PEDIATRIC HEADACHE

The literature on pharmacotherapy for headache in the adult population is quite extensive, although the counterpart in the pediatric literature is less so. Few randomized controlled trials have evaluated drug therapy for recurrent pediatric headache, and thus much of the knowledge in this area is based on clinical opinion. It appears that medications rarely rid a child of headache entirely, but rather reduce headache activity to a more manageable level (Labbé, 1998). Patient self-management of medically unconfirmed headache conditions using nonprescription agents is frequently encountered in the adult population (MacGregor, 1997) and may have an analog of parents’ medical management of their child in pediatric cases. Thus, it is possible that some mild cases of headache syndromes are adequately treated in the absence of medical consultation.

Pharmacological treatment of migraine in both adults and children has received far more attention than pharmacotherapy for tension-type headache. There are two types of migraine pharmacotherapy: symptomatic (also called abortive) and prophylactic. Symptomatic medications include mild pain relievers (e.g., simple analgesics, and non-steroidal anti-inflammatory drugs or NSAIDs), triptans, and ergot derivatives in order of potency and medical preference. The mild pain relievers are thought to inhibit prostaglandin synthesis, which is thought to thereby counteract the painful dilation of blood vessels caused by prostaglandins (Hämäläinen, 1998b). Of the mild pain relievers used for headache, acetaminophen (a simple analgesic) has become the mainstay for all age groups with a dose of 15–20 mg/kg up to every 4 hours (Welborn, 1997). Ibuprofen (an NSAID) is an alternative at 5–10 mg/kg every 8 hours for children ages 2–12. Although ibuprofen is generally more effective than acetaminophen, there are possible risks associated with ibuprofen use such as gastrointestinal bleeding, renal failure, and anaphylaxis (Levin, 2001); acetaminophen is thus the treatment of choice. Both acetaminophen and ibuprofen

have been found to be effective in a placebo-controlled crossover trial with pediatric migraine cases (Hämäläinen, Hoppu, & Valkeila, 1997). Naproxen (another NSAID) has been found to be useful for “menstrual migraine” (Levin, 2001), a form of migraine that develops at the onset of menses.

Aspirin appears to be about equal to acetaminophen in efficacy and can be delivered rectally if the oral route is unavailable due to vomiting during migraine attacks (Hämäläinen, 1998b). However, aspirin is associated with the development of Reye’s syndrome in children less than 12 years old, a potentially fatal condition characterized by severe edema of the brain, increased intracranial pressure, and liver dysfunction (Hämäläinen, 1998b; Lanzi et al., 1996). Thus, aspirin is generally avoided as a treatment for pediatric headache. If NSAIDs are found to be unsuccessful, pain relievers in the opioid class may be considered. Codeine given orally or morphine given intravenously or intramuscularly are often used. It is also recommended that anti-nausea agents (antiemetics) such as metoclopramide (Reglan) or dimenhydrinate (available without prescription) be given along with these pain relievers (Levin, 2001).

The adult literature suggests that some patients become habituated to over-the-counter abortive migraine medication and develop what has been referred to as “analgesic rebound” headache. In this condition, headaches gradually increase and headache characteristics are modified as a result of the consumption of analgesics (Symon, 1998). Consequently, an individual takes additional analgesics and the result is a vicious cycle. Analgesic rebound headache is now also being observed and diagnosed in the pediatric and adolescent population (Lanzi et al., 1996; Vasconcellos, Piña-Garza, Millan, & Warner, 1998). Thus, non-prescription drug intake without consultation in pediatric migraine cases can be potentially problematic.

The next level of abortive medications for migraine, the triptans, has received extensive attention in the literature since the development of the first drug in this class. The triptans mimic the action of serotonin by binding to serotonin receptors (5-HT<sub>1B</sub>) and causing extracerebral arteries to constrict (Ferrari, 1997). They also act through inhibiting the transmission of pain signals in the peripheral trigeminal sensory nerves and in the brainstem by binding to 5-HT<sub>1D</sub> receptors (Hargreaves & Shephard, 1999). The efficacy of such serotonin agonists was in fact the impetus for the serotonin model of migraine pathophysiology. Sumatriptan (Imitrex), available in oral and nasal formats, has been in existence the longest. This drug represented a major advance over traditional migraine therapies. Sumatriptan was initially only used in adults and only approved in this population, but it began to be increasingly used with children and adolescents (Linder, 1996; Solomon, 1995). However, high headache recurrence rates, poor response rates, adverse side effects (e.g., chest or neck dyesthesia, dizziness), and high cost (\$14 per tablet) have rendered the use of sumatriptan untenable in some cases (Adelman, Brod, von Seggem, Mannix, & Rappoport, 1998; Levin, 2001; Plosker & McTavish, 1994;

Tfelt-Hansen, 1993; Vasconcellos et al., 1998). Also, studies on sumatriptan for children and adolescents have yielded conflicting results (Welborn, 1997). Newer triptans are now available that have reduced side effects and increased efficacy, such as zolmitriptan (Zomig), naratriptan (Amerge), and rizatriptan (Maxalt; Rothner, 1999, 2001; Solomon, Cady, & Klapper, 1997; Vasconcellos et al., 1998). The latter, rizatriptan, is available in a wafer that dissolves instantly on the tongue without liquid (Pakalnis, 2001), making it a potentially desirable alternative in pediatric cases.

For the more severe migraine attacks, ergotamine (Bellergal-S) and its derivatives (e.g., dihydroergotamine or Migranal) are often used. These drugs have complex actions but are thought to help relieve pain by constricting swollen blood vessels in the outer area of the brain and scalp. In general, ergotamine and dihydroergotamine are only administered intravenously in children in the emergency room, although adolescents can be taught to administer dihydroergotamine subcutaneously for severe migraine attacks (Levin, 2001). There is a dependency issue associated with the use of this class of medications, and most children experience intense fear and may become agitated shortly after intravenous infusion (Holden et al., 1998). Efficacy studies on the use of ergotamine for pediatric migraine have yielded mixed results, with some finding it efficacious (Linder, 1994) and others finding the contrary (Welborn, 1997). Thus, the potential adverse events, method of administration, and the questionable efficacy of this class of medications have restricted the use of ergotamine and its derivatives to the most intractable presentations of migraine.

Prophylactic migraine therapy is reserved for cases characterized by significant functional disability (e.g., more than 3 school days missed per month, social isolation, family discord due to headaches, and severe migraine episodes). Prophylaxis is generally continued for approximately 2 months and then children are weaned, although longer courses for up to 1 year may be indicated (Levin, 2001). Data for prophylactic medications in the child and adolescent migraine literature are scarce. Cardiovascular drugs (beta-blockers and calcium channel blockers) have shown some efficacy in reducing migraine attacks despite an unknown mechanism (Becker, 1999; Welborn, 1997). For example, the calcium channel blocker flunarizine at a dose of 5 mg/day has been found to reduce the frequency and severity of migraine in children (Levin, 2001). In an early double-blind crossover study of the beta-blocker propranolol, Ludvigsson (1974) found this agent to produce a significantly better remission rate over placebo in pediatric migraine cases. However, other studies indicate that propranolol, though effective in adults, is ineffective or even counter-therapeutic in adolescents and children (Hermann, Kim, & Blanchard, 1995; Forsythe, Gillies, & Sills, 1984). Also, propranolol is contraindicated in children with asthma, congestive heart failure, and diabetes (Muszkat & Vergani, 1991; Welborn, 1997). Beta-blockers other than propranolol (e.g., metoprolol) may therefore be considered.

Psychotropic medications (i.e., medications that act on pathways in the central nervous system) have been used to manage chronic pain conditions and may work in part by influencing the psychological factors that modify the pain experience. Using psychotropic medications for pain conditions in children requires caution due to the potential toxicities associated with such medications (e.g., blood pressure changes, blurred vision, weight gain) and potential lethal consequences of an overdose. In childhood headache, tricyclic antidepressants have been tried and have shown preliminary efficacy in migraine prophylaxis despite largely unknown mechanisms (Wasiewski, 2001). Specifically, both amitriptyline (Elavil) and trazadone (Desyrel) have been found in open trials to be effective prophylactic agents in children and adolescents (Levin, 2001). These agents may prevent headache by regulating ion channels in pain pathways of the brain and by influencing the emotional factors having an etiological role in the recurrent headache syndrome. An open trial of pediatric migraine cases also found the serotonin antagonist pizotifen (Sandomigran) to be effective (Muszkat & Vergani, 1991), and this drug has also been recommended in the treatment of abdominal migraine (Levin, 2001). A new and promising approach to migraine prophylaxis in adults includes the use of anticonvulsant drugs such as valproic acid (Depakote) and gabapentin (Neurontin; Levin, 2001; Rothner, 1999, 2001), but there are as of yet no studies of these agents in the pediatric population. It is possible that the anticonvulsants prevent the kindling of a headache syndrome such that these agents have prophylactic utility (Connelly, 2003; Post & Silberstein, 1994). Other prophylactic agents include riboflavin, magnesium infusions, antihistamines, and certain NSAIDs, but there are scant data on the use of these agents in children and adolescents. Also, the Food and Drug Administration has not approved these agents for the prevention of migraine in children and adolescents. Sufficient data are not yet available to definitively justify particular prophylactic medication regimens for pediatric headache.

Tension-type headache is rarely treated with prescription medication, and, as such, little data exist on pharmacotherapy for this condition. Acute episodes are typically treated with simple analgesics or NSAIDs, although the latter are poorly substantiated. Tricyclic antidepressants have also been used for prophylaxis of tension-type headache, but the evidence is somewhat contradictory. Botulinum toxin, however, has a confirmed prophylactic effect in tension-type headache presentations in adults (Jensen & Olesen, 2000), with the mechanism thought to be an interruption of the interaction between peripheral nociception and central pain processing. However, much remains to be learned before a specific pharmacotherapy can be recommended for recurrent pediatric tension-type headache.

Given the relative novelty of the headache category "chronic daily headache," little is known about efficacious pharmacological treatments for this set of conditions in adults. Data on pharmacotherapy for chronic daily headache in children are virtually nonexistent. Typically, chronic daily headache is treated with similar

medications to those used to treat migraine and tension-type headache depending on the particular presentation (Redillas & Solomon, 2000).

An important caveat regarding pharmacotherapy approaches to recurrent headache is adherence. This has rarely been addressed in recurrent pediatric headache, which is unfortunate given that “patients’ failure to comply with therapeutic regimens is an all too common reason for lack of effective migraine control” (MacGregor, 1997). Research indicates that as many as half of recurrent headache sufferers fail to adhere properly to drug treatment regimens, and as many as two-thirds fail to make optimal use of abortive medications (Holroyd et al., 1988). In one study in the adult population (Steiner et al., 1994), migraine patients were prescribed standard prophylactic therapies with differing regimens taken for 8 weeks. Adherence was monitored using a microelectromechanical system (MEMS) device. Adherence rates were found to be 66% for qd (once daily) dosing and 30% for bid (twice daily) and tid (thrice daily) dosing. A similar study by Packard and O’Connell (1986) found 52% of adult headache patients to be noncompliant with the medication regimen. Adherence is often found to be quite low in chronic conditions in adolescents and children (Rapoff, 1999), and thus can pose a problem for pharmacological interventions in recurrent headache. Efforts to improve adherence to headache regimens is therefore an important area for clinical efforts by pediatric psychologists.

## PSYCHOLOGICAL AND BEHAVIORAL TREATMENT OF PEDIATRIC HEADACHE

Due to the potential adverse side-effects of certain pharmacological treatment, low medication adherence rates, the ubiquitous nature of headaches, and the role of psychological factors in headaches, there has been a large interest in behavioral strategies as an alternative or supplemental headache management modality (Allen & McKeen, 1991; Engel & Rapoff, 1990a, 1990b; McGrath, 2001a). In addition, children seem more adept than adults at using nondrug therapies (McGrath, Stewart, & Koster, 2001), perhaps due to being less biased than adults about their potential efficacy. Early studies on psychological and behavioral treatments for pediatric migraine and tension-type headache showed evidence of their efficacy (Duckro & Cantwell-Simmons, 1989; Hoelscher & Lichstein, 1984; Rapoff et al., 1988), although variations in headache types, floor effects, and treatment integrity issues continue to preclude definitive conclusions (Gutkin, Holborn, Walker, & Anderson, 1992; Hillenberg & Collins, 1982). Psychological and behavioral interventions for headache have traditionally included relaxation approaches, biofeedback, contingency management, and cognitive techniques. Similar to pharmacological approaches, these interventions might be conceived of as abortive versus prophylactic, although most psychological and behavioral approaches contain elements of both.

Treatments falling under the heading of relaxation include progressive relaxation training, self-hypnosis, autogenic training, and guided imagery. Although these approaches are modified across studies, progressive relaxation generally includes cycles of tensing and relaxing various muscle groups with the goal of attaining the ability to induce a “relaxation response” at will (Jacobsen, 1938). Hypnosis involves directing forces within the body to produce changes, beginning with an induction phase (e.g., having the child imagine his or her favorite cartoon or movie) and followed by gradual suggestions for relaxation, reduced anxiety, increased control, and reduced pain (McGrath et al., 2001). Self-hypnosis involves an identical procedure without assistance from a therapist. Autogenic training for recurrent pediatric headache generally refers to a process of guiding children to imagine and then experience specific sensations of warmth or heaviness (e.g., “Imagine your hands becoming warm and relaxing”). Finally, guided imagery involves invoking a child’s ability to imagine specific scenes or events that induce feelings of relaxation.

The mechanism of effect for relaxation approaches appears to be not simply a distraction from pain but the learning of a skill that has the capacity to induce physiological changes (e.g., dilate the vasculature, increase blood pulse volume, attenuate sympathetic outflow, reduce plasma  $\beta$ -endorphin) that are incompatible with the physiological changes responsible for headache attacks (Helm-Hylkema, Orlebeke, Enting, Thussen, & van Ree, 1990; Holden et al., 1998; Masek & Hoag, 1990; McGrath et al., 2001). A great deal of evidence exists supporting relaxation therapy as a clinically significant treatment strategy for the management of recurrent pediatric headache. The criteria for clinical significance is generally that defined by Blanchard and Schwarz (1988) as one producing a 50% improvement rate without a corresponding rise in the use of headache medication. Supporting studies include randomized placebo-controlled trials (Larsson & Melin, 1986; McGrath et al., 1992), randomized wait-list control trials (Engel, Rapoff, & Pressman, 1992; Wisniewski, Genshaft, Mulick, Coury, & Hammer, 1988), and multiple baseline designs (Engel, 1992). Gains appear to be maintained at least in terms of reductions in headache frequency (Larsson & Melin, 1986). There are, however, a few studies that do not find support for efficacy of relaxation procedures beyond placebo effects (Emmen & Passchier, 1988; McGrath et al., 1988). Such findings may be due to floor effects, variations in relaxation procedures, the high rate of placebo responding observed in headache studies (Battistella et al., 1992), or a true lack of efficacy in the treatment protocol. In addition, relaxation treatment may only have positive effects on headache frequency and not on headache duration and severity (Duckro & Cantwell-Simmons, 1989; Engel & Rapoff, 1990b). The overwhelming preponderance of evidence, however, indicates that relaxation procedures are an empirically validated treatment for recurrent pediatric headache (Holden et al., 1999).

Biofeedback training consists of transforming the electrical activity of the body into observable signals. The most common forms of biofeedback employed in the

treatment of pediatric headache are electromyographic (EMG) biofeedback (monitoring signals from electrical impulses generated from the forehead muscle) and thermal biofeedback (monitoring signals from a detector placed on the fingers). The mechanism of effect of this form of treatment appears to be the ability to modify physiological parameters that play an important role in headache pathogenesis (e.g., volitional control of body temperature has a direct effect on vasoconstriction and vasodilation; McGrath et al., 2001). However, an indirect mechanism might be that of the child's strengthened expectations of personal effectiveness for managing his or her headache condition (Burke & Andrasik, 1989) given that evidence of one's ability to alter physiological parameters is available in an immediate format.

Biofeedback is often used as part of a multi-component treatment package, and it is therefore often difficult to determine its independent contribution to treatment efficacy. However, studies of biofeedback that have been done on children with migraine, tension-type headache, or mixed headache indicate support for this method of treatment and maintenance of gains (Allen & McKeen, 1991; Burke & Andrasik, 1989; Engel & Rapoff, 1990a; Grazi, Leone, & Bussone, 1990; Labbé & Williamson, 1983, 1984; Packard & O'Connell, 1986; Powers et al., 2001). Comparisons between biofeedback and relaxation approaches generally indicate equal efficacy across these approaches (Blanchard & Andrasik, 1985; Fentress, Masek, Mehegan, & Benson, 1986), although one randomized control group study indicated greater effectiveness for autogenic relaxation than thermal biofeedback (Labbé, 1995). Thus, biofeedback appears to be a probably efficacious intervention for the treatment of recurrent pediatric headache (Holden et al., 1999).

In cognitive therapy, the focus is on modifying negative arousal-inducing thoughts associated with headache symptoms. Individual beliefs and expectations assumed to initiate or sustain headache episodes, or both, are changed through education and the substitution of problem-solving reassuring self-statements. Fewer studies have been conducted on cognitive approaches to the treatment of recurrent pediatric headache than on other psychological interventions, perhaps due to some of the cognitive techniques being beyond the grasp of younger children. Studies that do exist generally support the effectiveness of cognitive strategies. For example, in one of the first studies in this area, Richter et al. (1986) found that a cognitive intervention, consisting of cognitive restructuring, problem-solving, and education about coping with stress, was superior to an attention-placebo control group and equal to a relaxation therapy group in reducing migraine attacks. Other randomized trials also indicate support for cognitive techniques in terms of reduced headache frequency and intensity (Müller, Metsch, Pothmann, & Sartory, 1994). Moreover, McGrath et al. (2001) conducted a comprehensive review of pediatric headache treatments and concluded that strong and consistent evidence supports cognitive therapies for treating children's headaches. Thus, cognitive approaches also appear to be a viable alternative for at least prophylactically treating recurrent pediatric headache, although

certain of the cognitive techniques may not be developmentally appropriate for younger children.

Another behavioral approach that has been used to intervene in recurrent pediatric headache cases is referred to as "contingency management." This approach is based on the operant conditioning model and focuses on identifying and altering the precipitating and maintaining variables that are presumed to play a functional role in headache occurrence (Bijttebier & Vertommen, 1999). More specifically, contingency management is based on Fordyce's (1976) proposal that pain behaviors are learned responses maintained by social attention or avoidance of unpleasant circumstances. The goal of this approach is to lessen the behaviors that trigger attacks, exacerbate pain, or prolong disability by applying extinction (such as ignoring the behavior) while concomitantly increasing healthy behaviors by using positive reinforcement (Holden et al., 1998; McGrath et al., 2001). Although no compelling evidence supports the use of contingency management in isolation, early studies (e.g., Lake, 1981; Ramsden, Friedman, & Williamson, 1983) indicated that ignoring child's pain reports and reinforcing headache-free days resulted in increased school attendance and decreases in child's headache reports, with gains maintained 1 year afterward. However, it is possible that children only reported an improvement and actually continued to have headaches.

In clinical practice, the above psychological and behavioral approaches to recurrent pediatric headache are generally combined into a single treatment package. Studies examining such treatment packages have also been published (Holden et al., 1999) and generally support the efficacy of combining various interventions. However, it is then difficult to determine if all of the treatment components are necessary to achieve a positive effect. Other treatment modalities include thermal stimulation (i.e., applying compresses to the painful area), visual modulation (e.g., wearing red-tinted glasses to filter out short-wave flicker), transcutaneous electrical stimulation (TENS), massage, chiropractic adjustment, osteopathy, acupuncture, and herbal remedies (e.g., Feverfew). However, no empirical studies in the pediatric population exist on any of these modalities with the exception of acupuncture. In the one study examining this approach to recurrent pediatric headache (Pintov, Lahat, Alstein, Vogel, & Barg, 1997), acupuncture was found to be effective in reducing the frequency and intensity of pediatric migraine headaches compared to "sham acupuncture" (in which needles were inserted into random points rather than being inserted into points thought to be capable of influencing headache).

With respect to comparisons between psychological and pharmacological interventions, few outcome data exist that directly compare the two. In a widely cited meta-analysis of treatments for recurrent pediatric headache conducted by Hermann et al. (1995), effect sizes for thermal biofeedback and progressive muscle relaxation were found to be comparable to the effect sizes for pharmacological approaches (i.e., propranolol, calcium channel blockers, serotonergic drugs, dopaminergic drugs, clonidine, and papaverine). All major active pharmacologi-

cal and nonpharmacological treatment modalities included in the meta-analysis were shown to be superior to placebo or wait-list control groups. The use of psychological interventions as an adjunct to medications for headache has the potential of being a very fruitful approach but remains largely unexplored. One study examined the effect of combining propranolol treatment with a behavioral (relaxation and thermal biofeedback) intervention and found at 1-month follow-up that headache improvement increased from 50% to 70% for the combined treatment compared to the behavioral treatment by itself (Holroyd et al., 1995). Moreover, almost 90% of the participants receiving the combined treatment were clinically improved. More studies focusing on the combination of behavioral and pharmacotherapy interventions are warranted.

Interestingly, although behavioral approaches to treating headache might be justified by averting (or addressing) medical adherence problems, adherence nevertheless remains an issue in these treatment approaches. Although traditionally the term *adherence* or *compliance* has been used in the medical literature, it is also an important factor in behavioral and psychological treatments. Few studies have reported validating individuals' reports of compliance with home practice of behavioral treatments, and those that have examined compliance with home practice have generally found levels below 50% (Labbé, 1999; Turk & Rudy, 1991) unless a package to increase adherence is included (Fitterling, Martin, Gramling, Cole, & Milan, 1988). For example, Wisniewski et al. (1988) assigned 10 chronic headache sufferers age 12–17 to relaxation therapy or wait-list control. These researchers used a recorder equipped with a watch that would activate whenever the "play" button was depressed and would record the time elapsed from "play" to "stop." Information about adherence to treatment indicated that subjects over-reported actual practice time on average by 70%. Gutkin et al. (1992) measured adherence in 3 tension headache sufferers by using a microcomputer in a pressure mat and a hand-control device, both programmed to receive and record the amount of time spent practicing tense-release cycles presented on audio tapes. Adherence was found to vary from 8.1% to 73.0% of the prescribed practice time spent practicing appropriately. More importantly, the degree of improvement in headache status was related to the degree of adherence. Engel (1993) examined compliance to progressive relaxation training in 10 children by randomly placing a password on a cassette relaxation tape. This study found a fairly high mean compliance rate (84%) and no consistent relationship between compliance and headache relief. Differences in results may be attributed to differential participant characteristics: Child's age, perception of treatment rationale, and frequency of headache have all been found to be related to adherence (Evans & Blanchard, 1988; Guibert, Firestone, McGrath, Goodman, & Cunningham, 1990; Solbach, Sargent, & Coyne, 1989). Given the scant data in this area, it is difficult to draw conclusions. Adherence to nonpharmacological treatment should remain a concern, however, due to the investment of time and money.

## SUMMARY AND CONCLUSION: ISSUES FOR PEDIATRIC PRACTITIONERS

This article has reviewed the epidemiology, clinical features, diagnosis, assessment, etiology, and treatment of recurrent pediatric headache. Although the knowledge base in this area has increased over the past decade, much still remains to be learned in order to fully understand and effectively treat the condition. The following are some issues for further consideration.

### Assessment and Diagnostic Issues

The lack of verbal ability in younger children and developmental changes in conceptualizing pain have continued to pose problems for reliably and validly assessing and diagnosing pediatric headache. Given the possibility that repeated episodes of headache sensitize systems in the body such that future headaches occur more often and are more severe, it follows that attending to headache syndromes early on is critical. A challenge to pediatric practitioners is to develop specific and sensitive assessment and diagnostic tools for pediatric pain conditions such that syndromes such as recurrent pediatric headache are identified and treated early. Moreover, revisions in the International Headache Society's diagnostic criteria need to integrate research findings from the pediatric literature such that an acceptable standard is set for the diagnosis of recurrent pediatric headache. The current recommended interview assessment tool designed for pediatric headache is the Children's Headache Interview (McGrath & Hillier, 2001b), which helps elucidate several issues important for the diagnosis and treatment of pediatric headache (e.g., environmental factors, headache type, pain level and location, typical duration, extent of disability, emotional functioning, coping factors, and pain behaviors). Although the Children's Headache Interview provides valuable information, its length may preclude its use in some settings. A recommended self-report scales is the Children's Headache Assessment Scale (Budd, Workman, Lemsky, & Quick, 1994), which is specific to pediatric headache and is useful for determining the antecedents and consequences of headache. A recently developed assessment instrument, the Pediatric Migraine Disability Assessment Scale (PedsMIDAS; Hershey et al., 2001), evaluates the level of headache-related disability and may be useful for determining the extent of psychological or pharmacological treatment needed by the child. More instruments designed specifically to aid in distinguishing between different headache syndromes in children and capable of providing information useful for treatment matching would be helpful to pediatric practitioners. Newer methods for scale development involving Item Response Theory (Embretson & Reise, 2000) may prove useful for constructing brief assessment instruments that provide important conceptual and treatment information for pediatric headache.

The finding that the use of over-the-counter analgesics to treat headache can qualitatively and quantitatively alter subsequent headache activity presents an important assessment issue as well. Headaches are a common experience in children, and many of these headaches may be treated with over-the-counter analgesics by well-meaning parents who want to rapidly reduce their child's pain. In order to reduce the potential for analgesic-rebound headache syndromes, pediatric practitioners should routinely inquire about headaches in children seen clinically. If there is an indication of recurrent headaches, a more formal assessment should be done to ensure that effective interventions are implemented early. Routinely inquiring about headache may prevent the development of treatment-resistant headache syndromes.

### Adherence

Adherence to medical or psychosocial treatment regimens for recurrent pediatric headache is an area that remains to be fully explored. The minimal literature in this area to date has converged on the notion that lack of adherence is a common reason for lack of effective headache control (Holroyd et al., 1989; MacGregor, 1997). If knowledge is to be gained about the relative efficacy of various treatment strategies for recurrent pediatric headache, attention to adherence is critical. Moreover, adherence to treatment regimens is an important area for clinical intervention by pediatric psychologists. Efforts to predict nonadherence and to increase adherence rates for those individuals likely to be nonadherent have met with success in other areas of pediatric psychology (Rapoff, 1999). Thus, research and clinical efforts in this area have the capacity to boost treatment efficacy rates in pediatric headache.

### Treatment Matching

Providing psychological and behavioral treatments to children with headaches may prevent the development of a lifelong chronic pain syndrome that can become resistant to treatment over time. A critical question that remains about any treatment for recurrent pediatric headache, however, is which treatments are effective for which types of headaches under which subject and environmental conditions (Hermann, Blanchard, & Flor, 1997; Holden et al., 1999). Thus, an important area for future work is to articulate which combination of psychosocial and medical factors favor one treatment strategy over another. Such a knowledge base would have tremendous utility in maximizing headache treatment efficacy and potentially preventing a lifelong pain syndrome.

### Minimal Contact Treatments

One of the challenges facing pediatric psychology in the effective treatment of recurrent headaches is making psychological treatments that work cost-effective so

as to be accessible to those who need it (McGrath, 1999). Pharmacological treatments for headache generally require one clinic visit to determine a prescription and perhaps a 3-month follow-up appointment to ensure the prescription is tolerated. Conversely, the drawback to typical psychological and behavioral interventions has been the length of treatment (e.g., 4–8 clinic visits) and the resulting direct and indirect costs to the health care system and the family. Families may therefore prefer pharmacological treatments, yet children may benefit more from the combination of pharmacological and psychological interventions. Thus, a relatively recent trend has been to evaluate self-directed or minimal contact psychological treatments: “Given the current emphasis on reducing the cost of health-care in this country, minimal contact interventions will likely become the first line intervention in a behavioral step-care approach to the treatment of chronic benign headaches” (Rowan & Andrasik, 1996, p. 230).

There are numerous advantages for a minimal contact approach for the psychological treatment of recurrent headache. For example, training can take place in a milieu where the problem most frequently occurs. Other advantages include reducing the amount of school missed for clinic appointments, increasing accessibility to those living far away from a clinic, enhancing the generalization of skills to the home environment, and increasing cost-effectiveness (Allen & McKeen, 1991; Burke & Andrasik, 1989; Rowan & Andrasik, 1996). There are potential problems associated with this approach as well, however, such as issues of time commitment, motivation, and adherence. Typically, minimal contact treatments for headache involve relaxation or cognitive treatments presented in manuals and accompanied by audiotapes so that children can learn these headache-management strategies largely at home. Portable biofeedback devices have also been used such that children can practice biofeedback in their homes rather than in clinics. Studies on minimal contact treatments for recurrent pediatric headache to date have indicated that providing psychological and behavioral headache treatment in a format requiring few clinic visits (e.g., via manuals, audiotapes, and portable biofeedback devices) has resulted in comparable treatment efficacy to lengthier clinic-based treatments (Blanchard et al., 1985; Blanchard & Schwarz, 1988; Griffiths & Martin, 1996; Guarnieri & Blanchard, 1990; Larsson, Daleflod, Hakansson, & Melin, 1987; Larsson, Melin, & Doberl, 1990; McGrath et al., 1988; Teders et al., 1984). Moreover, the minimal contact treatments were found to be much more cost-effective (producing two to six times more headache reduction per therapist hour than clinic-based treatments) and to not differ from clinic-based treatments with respect to treatment adherence or dropout rates (Rowan & Andrasik, 1996). However, these studies tended to have small sample sizes and methodological problems (e.g., interventions differing beyond just format as a function of being clinic-based or minimal-contact, no standard for what duration of time constitutes minimal contact, or potential treatment contamination). Nevertheless, minimal contact treatments appear to have tremendous utility for reducing health care costs. A current

challenge is therefore to continue to construct efficacious and cost-effective mediums for headache treatment delivery. For example, an approach to treating recurrent pediatric headache involving a stand-alone CD-ROM program and manual (called “Headstrong”) is currently being developed and tested by the author and colleagues (Connelly & Rapoff, 2002). “Headstrong” is a developmentally-tailored program designed to supplement medical care that allows children to learn several different behavioral and cognitive strategies for managing their headaches on their own.

In conclusion, extensive gains have been made over the past decade in conceptualizing and treating pain syndromes in children, with recurrent pediatric headache being no exception. However, clinical and research efforts still need to focus on diagnosis and assessment, improving adherence, matching specific treatments to children with certain medical or psychosocial indicators thought to be best served by those treatments, and modifying psychological treatments such that they can be delivered in a cost-effective (but still efficacious) format. Such efforts are critical in that the prevalence of recurrent pediatric headache is extensive and increasing, and pediatric headache has the capacity of becoming a chronic lifelong pain syndrome in the absence of effective interventions.

## ACKNOWLEDGMENT

I thank Michael Rapoff for his helpful comments on an earlier draft of this article.

## REFERENCES

- Abu-Arefeh, I., & Russell, G. (1994). Prevalence of headache and migraine in schoolchildren. *British Medical Journal*, *309*, 765–769.
- Adamec, R. E. (1990). Does kindling model anything clinically relevant? *Biological Psychiatry*, *27*(3), 249–279.
- Adelman, J. L., Brod, A., Von Seggen, R. L., Mannix, L. K., & Rappoport, A. M. (1998). Migraine preventive medications: A reappraisal. *Cephalalgia*, *18*, 605–611.
- Ad Hoc Committee on Classification of Headache (1962a). Classification of headache. *JAMA*, *179*(9), 127–128.
- Ad Hoc Committee on the Classification of Headaches, National Institute of Neurological Diseases and Deafness (1962b). Classification of headache. *Neurology*, *12*, 378–380.
- Allen, K. D., & McKeen, L. R. (1991). Home-based multi-component treatment of pediatric migraine. *Headache*, *31*, 467–472.
- Andrasik, F., Kabela, E., Quinn, S., Attanasio, V., Blanchard, E. B., & Rosenblum, E. L. (1988). Psychological functioning of children who have recurrent migraine. *Pain*, *34*, 43–52.
- Annequin, D., Tourniaire, B., & Massiou, H. (2000). Migraine and headache in childhood and adolescence. *Pediatric Clinics of North America*, *47*(3), 617–631.
- Anttila, P., Mestähonkala, L., Aromaa, M., Sourander, A., Salminen, J., Helenius, H., et al. (2002). Determinants of tension-type headache in children. *Cephalalgia*, *22*, 401–408.

- Aromaa, M., Rautava, P., Helenius, H., & Sillanpää, M. L. (1998). Factors of early life as predictors of headache in children at school entry. *Headache*, *38*, 23–30.
- Barlow, C. F. (1984). *Headaches and migraine in childhood*. London: Spastics International Medical Publications.
- Barlow, C. F. (1994). Migraine in the infant and toddler. *Journal of Child Neurology*, *9*, 92–94.
- Battistella, P. A., Ruffilli, R., Cernetti, R., Pettenazzo, A., Baldin, L., Bertoli, S., et al. (1992). A placebo-controlled crossover trial using trazodone in pediatric migraine. *Headache*, *33*, 36–39.
- Becker, W. J. (1999). Evidence based migraine prophylactic drug therapy. *Canadian Journal of Neurological Science*, *26*(Suppl. 3), S27–S32.
- Bendsten, L., Jensen, R., & Olesen, J. (1996). Qualitatively altered nociception in chronic myofascial pain. *Pain*, *65*, 259–264.
- Bijttebier, P., & Vertommen, H. (1999). Antecedents, concomitants, and consequences of pediatric headache: Confirmatory construct validation of two parent-report scales. *Journal of Behavioral Medicine*, *22*(5), 437–456.
- Bille, B. O. (1962). Migraine in school children: A study of the incidence, and short-term prognosis, and a clinical, psychological and encephalographic comparison between children with migraine and matched controls. *Acta Paediatrica Scandinavica*, *51*(Suppl. 136), 1–151.
- Bille, B. O. (1981). Migraine in children and its prognosis. *Cephalalgia*, *1*, 71–75.
- Blanchard, E. B. (1992). Psychological treatment of benign headache disorders. *Journal of Consulting and Clinical Psychology*, *60*, 537–551.
- Blanchard, E. B., & Andrasik, F. (Eds.). (1985). *Management of chronic headaches: A psychological approach*. New York: Pergamon.
- Blanchard, E. B., Andrasik, F., Applebaum, A., Evans, D. D., Jurish, S. E., Teders, S. J., et al. (1985). The efficacy and cost-effectiveness of minimal-therapist contact, non-drug treatment of chronic migraine and tension headaches. *Headache*, *25*, 214–220.
- Blanchard, E. B., & Schwarz, S. P. (1988). Clinically significant changes in behavioral medicine. *Behavioral Assessment*, *10*, 171–188.
- Breslau, N., & Rasmussen, B. K. (2001). The impact of migraine: epidemiology, risk factors, and comorbidities. *Neurology*, *56*(6, Suppl. 1), S4–S12.
- Breslau, N., Schultz, L. R., Stewart, W. F., Lipton, R. B., Lucia, V. C., & Welch, K. M. A. (2000). Headache and major depression: Is the association specific to migraine? *Neurology*, *54*, 308–313.
- Budd, K. S., Workman, D. E., Lemsky, C. M., & Quick, D. M. (1994). The Children's Headache Assessment Scale (CHAS): Factor structure and psychometric properties. *Journal of Behavioral Medicine*, *17*(2), 159–179.
- Burke, E. J., & Andrasik, F. (1989). Home vs. clinical-based biofeedback treatment for pediatric migraine: Results of treatment through 1-year follow-up. *Headache*, *29*, 434–440.
- Bush, J. P. (1987). Pain in children: A review of the literature from a developmental perspective. *Psychology and Health*, *1*, 215–236.
- Connelly, M. (2003). *Sensitization as an integrative model for recurrent headache*. Manuscript submitted for publication.
- Connelly, M., & Rapoff, M. A. (2002). New invention disclosure: "Headstrong." *Technology Transfer and Intellectual Property News*, *1*, 1.
- Cunningham, S. J., McGrath, P. J., Ferguson, H. B., Humphreys, P., D'Astous, J., Latter, J., Goodman, J. T., & Firestone, P. (1986). Personality and behavioral characteristics in pediatric migraine. *Headache*, *27*, 16–20.
- Dahlöf, C. & Dimenas, E. (1995). Migraine patients experience poorer subjective well-being/quality of life even between attacks. *Cephalalgia*, *15*, 31–36.
- Day, W. H. (1873). *Essays on diseases of children*. London: Churchill.
- Duckro, P. N., & Cantwell-Simmons, E. (1989). A review of studies evaluating biofeedback and relaxation training in the management of pediatric headache. *Headache*, *29*, 428–433.

- Edmeads, J., Findlay, H., Tugwell, P., Pryse-Phillips, W., Nelson, R. F., & Murray, T. J. (1993). Impact of migraine and tension-type headache on life-style, consulting behaviour, and medication use: A Canadian population survey. *Canadian Journal of Neurological Science, 20*, 131–137.
- Egger, H. L., Angold, A., & Costello, E. J. (1998). Headaches and psychopathology in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 37*, 951–958.
- Embretson, S. E., & Reise, S. (2000). *Item response theory for psychologists*. Mahwah, NJ: Lawrence Erlbaum Associates, Inc.
- Emmen, H. H., & Passchier, J. (1988). Treatment of headache among children by progressive relaxation. *Cephalalgia, 7*, 387–389.
- Engel, J. M. (1992). Relaxation training: A self-help approach for children with headaches. *The American Journal of Occupational Therapy, 46*(7), 591–596.
- Engel, J. M. (1993). Children's compliance with progressive relaxation, procedures for improving headache control. *The Occupational Therapy Journal of Research, 13*(4), 219–230.
- Engel, J. M., & Rapoff, M. A. (1990a). Biofeedback-assisted relaxation training for adult and pediatric headache disorders. *The Occupational Therapy Journal of Research, 10*(5), 283–299.
- Engel, J. M., & Rapoff, M. A. (1990b). A component analysis of relaxation training for children with vascular, muscle contraction, and mixed-headache disorders. *Advances in Pain Research, 15*, 273–290.
- Engel, J. M., Rapoff, M. A., & Pressman, A. R. (1992). Long-term follow-up of relaxation training for pediatric headache disorders. *Headache, 32*, 152–156.
- Evans, D. D., & Blanchard, E. B. (1988). Prediction of early termination from the self-regulatory treatment of chronic headache. *Biofeedback and Self-Regulation, 13*(3), 245–256.
- Fentress, D. W., Masek, B. J., Mehegan, J. E., & Benson, H. (1986). Biofeedback and relaxation-response training in the treatment of pediatric migraine. *Developmental Medicine and Child Neurology, 28*, 139–146.
- Ferrari, M. D. (1997). 311C90: Increasing the options for therapy with effective acute antimigraine 5HT1B/1D receptor agonists. *Neurology, 48*(Suppl. 3), S21-S24.
- Fitterling, J. M., Martin, J. E., Gramling, S., Cole, P., & Milan, M. (1988). Behavioral management of exercise training in vascular headache patients: An investigation of exercise adherence and headache activity. *Journal of Applied Behavior Analysis, 21*, 9–19.
- Fordyce, W. E. (1976). *Behavioral methods for chronic pain and illness*. St. Louis, MO: Mosby.
- Forsythe, W. I., Gillies, D., & Sills, M. A. (1984). Propranolol in the treatment of childhood migraine. *Developmental Medicine and Child Neurology, 26*, 737–741.
- Gladstein, J., & Holden, E. W. (1996). Chronic daily headache in children and adolescents: A 2-year prospective study. *Headache, 36*, 349–351.
- Gladstein, J., Holden, E. W., Peralta, L., & Raven, M. (1993). Diagnoses and symptom patterns in children presenting to a pediatric headache clinic. *Headache, 33*, 497–500.
- Goadsby, P. J. (1994). New insights into the pathogenesis of migraine. In F. C. Rose (Ed.), *New advances in headache research* (Vol. 4, pp. 5–7). London: Smith-Gordon.
- Goadsby, P. J. (1997). Increasing the options for effective migraine management. *Neurology, 48*(Suppl. 3), S1–S3.
- Grazzi, L., Leone, M., & Bussone, G. (1990). A therapeutic alternative for tension headache in children: Treatment and 1-year follow-up results. *Biofeedback and Self-Regulation, 15*, 1–6.
- Griffiths, J. D., & Martin, P. R. (1996). Clinical- versus home-based treatment formats for children with chronic headache. *British Journal of Health Psychology, 1*, 151–166.
- Guarnieri, P., & Blanchard, E. B. (1990). Evaluation of a home-based thermal biofeedback treatment of pediatric migraine headache. *Biofeedback and Self-Regulation, 15*(2), 179–184.
- Guibert, M. B., Firestone, P., McGrath, P., Goodman, J. T., & Cunningham, J. S. (1990). Compliance factors in the behavioral treatment of headache in children and adolescents. *Canadian Journal of Behavioral Science, 22*(1), 37–44.

- Gotkin, A. J., Holborn, S. W., Walker, J. R., & Anderson, B. A. (1992). Treatment integrity of relaxation training for tension headaches. *Journal of Behavioural Therapy and Experimental Psychiatry*, 23(3), 191–198.
- Hämäläinen, M. L. (1998a). Migraine in children, guidelines for treatment. *CNS Drugs*, 10(2), 105–117.
- Hämäläinen, M. L. (1998b). Attack treatment of migraine in children. *Headache Quarterly, Current Treatment and Research*, 9(3), 241–244.
- Hämäläinen, M. L., Hoppu, K., & Valkeila, E. (1997). Ibuprofen or acetaminophen for the acute treatment of migraine in children: A double-blind, randomized, placebo-controlled, crossover study. *Neurology*, 48(1), 103–107.
- Hargreaves, R. J., & Shephard, S. L. (1999). Pathophysiology of migraine—new insights. *Canadian Journal of Neurological Sciences*, Suppl. 3, S12–S19.
- Headache Classification Committee of the International Headache Society (1988). Classification and diagnostic criteria for headache disorders, cranial neuralgias, and facial pain. *Cephalalgia*, 8(Suppl. 7), 1–96.
- Helm-Hylkema, H. V. D., Orlebeke, J. F., Enting, L. A., Thussen, J. H. H., & van Ree, J. (1990). Compliance factors in the behavioral treatment of headache in children and adolescents. *Canadian Journal of Behavioural Science*, 22, 37–44.
- Hermann, C., & Blanchard, E. B. (1998). Psychophysiological reactivity in pediatric migraine patients and healthy controls. *Journal of Psychosomatic Research*, 44(2), 229–240.
- Hermann, C., Blanchard, E. B., & Flor, H. (1997). Biofeedback treatment for pediatric migraine: Prediction of treatment outcome. *Journal of Consulting and Clinical Psychology*, 65, 611–616.
- Hermann, C., Kim, M., & Blanchard, E. B. (1995). Behavioral and prophylactic pharmacological intervention studies of pediatric migraine: An exploratory meta-analysis. *Pain*, 60, 239–256.
- Hershey, A. D., Powers, S. W., Vockell, A. L., LeCates, S., Kabbouche, M. A., & Maynard, M. K. (2001). PedMIDAS: Development of a questionnaire to assess disability of migraines in children. *Neurology*, 57(11), 2034–9.
- Hillenber, J. B., & Collins, F. L. (1982). A procedural analysis and review of relaxation training research. *Behavior Research and Therapy*, 20, 251–260.
- Hockaday, J. M. (1978). Late outcome of childhood onset migraine and factors affecting outcome, with particular reference to early and late EEG findings. In R. Green (Ed.), *Current concepts in migraine research* (pp. 41–48). New York: Raven.
- Hoelscher, T. J. & Lichstein, K. L. (1984). Behavioral assessment and treatment of child migraine: Implications for clinical research and practice. *Headache*, 24, 94–103.
- Holden, E. W., Bachanas, P., Kullgren, K., & Gladstein, J. (2001). Chronic daily headache in children and adolescents. In P. A. McGrath & L. M. Hillier (Eds.), *The child with headache: Diagnosis and treatment* (Vol. 19, pp. 221–241). Seattle, WA: IASP Press.
- Holden, E. W., Deichmann, M. M., & Levy, J. D. (1999). Empirically supported treatments in pediatric psychology: Recurrent pediatric headache. *Journal of Pediatric Psychology*, 24(2), 91–109.
- Holden, E. W., Gladstein, J., Trulsen, M., & Wall, B. (1994). Chronic daily headache in children and adolescents. *Headache*, 34, 508–514.
- Holden, E. W., Levy, J. D., Deichmann, M. M., & Gladstein, J. (1998). Recurrent pediatric headaches: Assessment and intervention. *Developmental and Behavioral Pediatrics*, 19(2), 109–116.
- Holmes, W. F., MacGregor, E. A., & Dodick, D. (2001). Migraine-related disability: Impact and implications for sufferers' lives and clinical issues. *Neurology*, 56(6, Suppl. 1), S13–S19.
- Holroyd, K. A. (2002). Assessment and psychological management of recurrent headache disorders. *Journal of Consulting and Clinical Psychology*, 70(3), 656–677.
- Holroyd, K. A., Cordingley, G. E., Pingel, J. D., Jerome, A., Theofanous, A. G., Jackson, D. K., et al. (1989). Enhancing the effectiveness of abortive therapy: A controlled evaluation of self-management training. *Headache*, 29, 148–153.

- Holroyd, K. A., France, J. L., Cordingley, G. E., Rokicki, L. A., Kvaal, S. A., Lipchik, G. L., et al. (1995). Enhancing the effectiveness of relaxation-thermal biofeedback training with propranolol hydrochloride. *Journal of Consulting and Clinical Psychology, 63*, 327–330.
- Holroyd, K. A., Holm, J. E., Hursey, K. G., Penzien, D. B., Cordingley, G. E., Theofanous, A. G., et al. (1988). Recurrent vascular headache: Home-based behavioral treatment versus abortive pharmacological treatment. *Journal of Consulting and Clinical Psychology, 56*, 218–223.
- Honkasalo, M. L., Kaprio, J., Winter, T., Phlic, K. H., Sillanpää, M., & Koskenvuo, M. (1995). Migraine and concomitant symptoms among 8167 adult twin pairs. *Headache, 35*, 70–78.
- Jacobson, E. (1938). *Progressive relaxation*. Chicago: University of Chicago Press.
- Jay, G. E., & Tomasi, L. G. (1981). Pediatric headache: A one year retrospective analysis. *Headache, 21*, 5–9.
- Jensen, R. (1999). Pathophysiological mechanisms of tension-type headache: A review of epidemiological and experimental studies. *Cephalalgia, 19*, 602–621.
- Jensen, R., & Olesen, J. (2000). Tension-type headache: An update on mechanisms and treatment. *Current Opinion in Neurology, 13*, 285–289.
- Joffe, R., Bakal, D. A., & Kaganov, J. (1983). A self-observation study of headache symptoms in children. *Headache, 23*, 20–25.
- Karwautz, A., Wöber, C., Lang, T., Böck, A., Wagner-Ennsgraber, C., Vesely, C., et al. (1999). Psychosocial factors in children and adolescents with migraine and tension-type headache: A controlled study and review of the literature. *Cephalalgia, 19*, 32–43.
- Labbé, E. E. (1995). Treatment of childhood migraine with autogenic training and skin temperature biofeedback: A component analysis. *Headache, 35*, 10–13.
- Labbé, E. E. (1998). Pediatric headaches. In T. H. Ollendick & M. Hersen (Eds.), *Handbook of child psychopathology* (3rd ed., pp. 381–394). New York: Plenum.
- Labbé, E. E. (1999). Commentary: Salient aspects of research in pediatric headache and future directions. *Journal of Pediatric Psychology, 24*(2), 113–114.
- Labbé, E. E., & Williamson, D. A. (1983). Temperature biofeedback in the treatment of children with migraine headaches. *Journal of Pediatric Psychology, 8*, 317–326.
- Labbé, E. E., & Williamson, D. A. (1984). Treatment of childhood migraine using autogenic feedback training. *Journal of Consulting and Clinical Psychology, 52*, 968–976.
- Lake, A. E. (1981). Behavioral assessment considerations in the management of headache. *Headache, 21*, 170–178.
- Lanzi, G., Balottin, U., Borgatti, R., Guderzo, M., & Scarabello, E. (1988). Different forms of migraine in childhood and adolescence: notes on personality traits. *Headache, 28*, 618–622.
- Lanzi, G., Balottin, U., Zambrino, C. A., Cernibori, A., Del Bene, E., Gallai, V., et al. (1996). Guidelines and recommendations for the treatment of migraine in paediatric and adolescent patients. Italian Society for the Study of Headache. *Functional Neurology, 11*, 269–275.
- Larsson, B., Bille, B., & Pederson, N. L. (1995). Genetic influence in headaches: A Swedish twin study. *Headache, 35*, 513–519.
- Larsson, B., Daleflod, B., Hakansson, L., & Melin, L. (1987). Therapist-assisted versus self-help relaxation treatment of chronic headaches in adolescents: A school-based intervention. *Journal of Child Psychology and Psychiatry, 28*, 127–136.
- Larsson, B., & Melin, L. (1986). Chronic headaches in adolescents: Treatment in a school setting with relaxation training as compared with information-contact and self-registration. *Pain, 25*, 325–336.
- Larsson, B., Melin, L., & Doberl, A. (1990). Recurrent tension headache in adolescents treated with self-help relaxation training and a muscle relaxant drug. *Headache, 30*, 665–671.
- Levin, S. D. (2001). Drug therapies for childhood headache. In P. A. McGrath & L. M. Hillier (Eds.), *The child with headache: Diagnosis and treatment* (Vol. 19, pp. 109–127). Seattle, WA: IASP Press.
- Linder, S. (1994). Treatment of childhood headache with dihydroergotamine mesylate. *Headache, 34*, 578–580.

- Linder, S. (1996). Subcutaneous sumatriptan in the clinical setting: The first 50 consecutive patients with acute migraine in a pediatric neurology office practice. *Headache*, *36*, 419–422.
- Lipton, R. B. (1997). Diagnosis and epidemiology of pediatric migraine. *Current Opinion in Neurology*, *10*(3), 231–236.
- Lipton, R. B., Stewart, W. F., & von Korff, M. (1997). Burden of migraine: Societal costs and therapeutic opportunities. *Neurology*, *48*(Suppl. 3), S4–S9.
- Ludvigsson, J. (1974). Propranolol used in prophylaxis of migraine in children. *Acta Neurologica Scandinavica*, *50*, 1009–115.
- MacGregor, E. A. (1997). The doctor and the migraine patient: Improving compliance. *Neurology*, *48*(Suppl. 3), S16–S20.
- Marcon, R. A., & Labbé (1990). Assessment and treatment of children's headaches from a developmental perspective. *Headache*, *30*, 586–592.
- Markowitz, S., Saito, K., & Moskowitz, M. A. (1988). Neurogenically mediated plasma extravasation in dura mater: Effect of ergot alkaloids. *Cephalalgia*, *8*, 83–91.
- Martin, P. R. (1993). *Psychological management of chronic headaches*. New York: Guilford.
- Martin, S. E., & Smith, M. S. (1995). Psychosocial factors in recurrent pediatric headache. *Pediatric Annals*, *24*(9), 469–474.
- Masek, B. J., & Hoag, N. L. (1990). Headache. In A. M. Gross & R. S. Drabman (Eds.), *Handbook of clinical behavioral pediatrics: Applied clinical psychology* (pp. 99–109). New York: Plenum.
- Matthew, N. T., Reuveni, U., & Perez, F. (1987). Transformed or evolutive migraine. *Headache*, *27*(2), 102–106.
- Maytal, J., Lipton, R. B., Salomon, S., & Shinnar, S. (1992). Childhood onset cluster headache. *Headache*, *32*, 275–279.
- Maytal, J., Young, M., Schechter, A., & Lipton, R. B. (1997). Pediatric migraine and the International Headache Society (IHS) criteria. *Neurology*, *48*, 602–607.
- McGrath, P. J. (1999). Commentary: Recurrent headaches: Making what works available to those who need it. *Journal of Pediatric Psychology*, *24*(2), 111–112.
- McGrath, P. A. (2001a). Understanding children's headache: Current status and future challenges. In P. A. McGrath & L. M. Hillier (Eds.), *The child with headache: Diagnosis and treatment* (Vol. 19, pp. 242–252). Seattle, WA: IASP Press.
- McGrath, P. A. (2001b). Headache in children: The nature of the problem. In P. A. McGrath & L. M. Hillier (Eds.), *The child with headache: Diagnosis and treatment* (Vol. 19, pp. 1–27). Seattle, WA: IASP Press.
- McGrath, P. A. (2001c). Chronic daily headache in children and adolescents. *Current Pain and Headache Reports*, *5*(6), 557–566.
- McGrath, P. A., & Hillier, L. M. (2001a). Recurrent headache: Triggers, causes, and contributing factors. In P. A. McGrath & L. M. Hillier (Eds.), *The child with headache: Diagnosis and treatment* (Vol. 19, pp. 77–107). Seattle, WA: IASP Press.
- McGrath, P. A., & Hillier, L. M. (Eds.). (2001b). *The child with headache: Diagnosis and treatment* (Vol. 19). Seattle, WA: IASP Press.
- McGrath, P. J., Humphreys, P., Goodman, J. T., Keene, D., Firestone, P., Jacob, P., et al. (1988). Relaxation prophylaxis for childhood migraine: a randomized placebo-controlled trial. *Developmental Medical Child Neurology*, *30*(5), 626–631.
- McGrath, P. J., Humphreys, P., Keene, D., Goodman, J. T., Lascelles, M. A., Cunningham, S. J., et al. (1992). The efficacy and efficiency of a self-administered treatment for adolescent migraine. *Pain*, *49*, 321–324.
- McGrath, P. J., & Reid, G. J. (1995). Behavioral treatment of pediatric headache. *Pediatric Annals*, *24*, 486–491.
- McGrath, P. A., Stewart, D., & Koster, A. L. (2001). Nondrug therapies for childhood headache. In P. A. McGrath & L. M. Hillier (Eds.), *The child with headache: Diagnosis and treatment* (Vol. 19, pp. 129–158). Seattle, WA: IASP Press.

- Metsähonkala, L. (1998). Headache and school. *Headache Quarterly, Current Research and Development*, 9(3), 233–236.
- Mikail, S. F., & von Baeyer, C. L. (1990). Pain, somatic focus, and emotional adjustment in children of chronic headache sufferers and controls. *Social Science Medicine*, 31, 51–59.
- Moskowitz, M. A. (1984). The neurobiology of vascular head pain. *Annals of Neurology*, 16, 157–161.
- Moskowitz, M. A. (1991). The visceral organ brain: Implications for the pathophysiology of vascular head pain. *Neurology*, 41, 182–186.
- Moskowitz, M. A. (1992). Neurogenic versus vascular mechanisms of sumatriptan and ergot alkaloids in migraine. *Trends in Pharmacological Science*, 13, 307–311.
- Müller, B., Metsch, J., Pothmann, R., & Sartory, G. (1994). A comparison of psychological and pharmacological prophylaxis of childhood migraine. In F. C. Rose (Ed.), *New advances in headache research* (Vol. 4, p. 328). London: Smith-Gordon.
- Muszkat, M., & Vergani, M. (1991). Headaches in childhood—prospective study of 155 cases. *Headache Quarterly, Current Treatment and Research*, 2(3), 222–224.
- Newacheck, P. W., & Taylor, W. R. (1992). Childhood chronic illness: Prevalence, severity, and impact. *American Journal of Public Health*, 82, 364–371.
- Ottman, R., & Lipton, R. (1996). Is the comorbidity of epilepsy and migraine due to a shared genetic susceptibility? *Neurology*, 47, 918–924.
- Packard, R. C., & O'Connell, P. (1986). Medication compliance among headache patients. *Headache*, 26, 416–419.
- Pakalnis, A. (2001). New avenues in treatment of pediatric migraine: A review of the literature. *Family Practice*, 18(1), 101–106.
- Pintov, S., Lahat, E., Alstein, M., Vogel, Z., & Barg, J. (1997). Acupuncture and the opioid system: Implications in management of migraine. *Pediatric Neurology*, 17, 129–133.
- Plosker, G. L., & McTavish, D. (1994). Sumatriptan. A reappraisal of its pharmacology and therapeutic efficacy in acute treatment of migraine and cluster headache. *Drugs*, 47, 622–651.
- Post, R. M. (1992). Transduction of psychosocial stress into the neurobiology of recurrent affective disorder. *American Journal of Psychiatry*, 149(8), 999–1010.
- Post, R. M., & Silberstein, S. D. (1994). Shared mechanisms in affective illness, epilepsy, and migraine. *Neurology*, 44(10 Suppl. 7), S37–S47.
- Powers, S. W., Mitchell, M. J., Byars, K. C., Benti, A. L., LeCates, S. L., & Hershey, A. D. (2001). A pilot study of one-session biofeedback training in pediatric headache. *Neurology*, 56(1), 133.
- Prensky, A. I. (1976). Migraine and migrainous variables in pediatric patients. *Pediatric Clinics of North America*, 23, 461–471.
- Prensky, A. L., & Sommer, D. (1979). Diagnosis and treatment of migraine in children. *Neurology*, 29, 506–510.
- Ramsden, R., Friedman, B., & Williamson, D. (1983). Treatment of childhood headache reports with contingency management procedures. *Journal of Clinical Child Psychology*, 12, 202–206.
- Rapoff, M. A. (1999). *Adherence to pediatric medical regimens*. New York: Kluwer Academic.
- Rapoff, M., Walsh, D., & Engel, J. M. (1988). Assessment and management of chronic pediatric headache. *Issues in Comprehensive Nursing*, 11, 159–178.
- Rapoport, A. M., & Sheftell, F. D. (1996). *Headache disorders: A management guide for practitioners*. Philadelphia: Saunders.
- Redillas, C., & Solomon, S. (2000). Prophylactic pharmacological treatment of chronic daily headache. *Headache*, 40(2), 83–102.
- Richter, J. L., McGrath, P., Humphreys, P. J., Goodman, J. T., Firestone, P., & Keene, D. (1986). Cognitive and relaxation treatment of pediatric migraine. *Pain*, 25, 195–203.
- Rothner, A. D. (1978). Headache in children: A review. *Headache*, 18, 169.

- Rothner, A. D. (1999). Headache in children and adolescents. *Child and Adolescent Psychiatric Clinics of North America*, 8(4), 727–745.
- Rothner, A. D. (2001). Differential diagnosis of headaches in children and adolescents. In P. A. McGrath & L. M. Hillier (Eds.), *The child with headache: Diagnosis and treatment* (Vol. 19, pp. 57–76). Seattle, WA: IASP Press.
- Rowan, A. B., & Andrasik, F. (1996). Efficacy and cost-effectiveness of minimal therapist contact treatments of chronic headaches: A review. *Behavior Therapy*, 27, 207–234.
- Saeed, M. A., Pumariega, A. J., & Cinciripini, P. M. (1992). Psychopharmacological management of migraine in children and adolescents. *Journal of Child and Adolescent Psychopharmacology*, 2(3), 199–211.
- Sakai, F., Ebihara, S., Akiyama, M., & Horikawa, M. (1995). Pericranial muscle hardness in tension-type headache: A non-invasive measurement method and its clinical application. *Brain*, 118, 523–531.
- Scheller, J. M. (1995). The history, epidemiology, and classification of headaches in childhood. *Seminars in Pediatric Neurology*, 2, 102–108.
- Silberstein, S. D., Lipton, R. B., Solomon, S., & Matthew, N. (1994). Classification of daily and near-daily headaches: Proposed revision to the IHS criteria. *Headache*, 34, 1–7.
- Sillanpää, M. (1983). Changes in the prevalence of migraine and other headaches during the first seven school years. *Headache*, 23, 15–19.
- Sillanpää, M., & Anttila, P. (1996). Increasing prevalence of headache in 7-year-old schoolchildren. *Headache*, 36, 466–470.
- Solbach, P., Sargent, J., & Coyne, L. (1989). An analysis of home practice patterns for non-drug headache treatments. *Headache*, 29, 528–531.
- Solomon, G. D. (1995). The pharmacology of medications used in treating migraine. *Seminars in Pediatric Neurology*, 2, 165–177.
- Solomon, G. D. (1997). Evolution of the measurement of quality of life in migraine. *Neurology*, 48(Suppl. 3), S10–S15.
- Solomon, G. D., Cady, R. K., & Klapper, J. A. (1997). Clinical efficacy and tolerability of 2.5mg zolmitriptan for the acute treatment of migraine. The 042 Clinical Trial Study Group. *Neurology*, 49(5), 1219–1225.
- Solomon, S., Lipton, R. B., & Newman, L. C. (1992). Evaluation of chronic daily headache: Comparison to criteria for chronic tension-type headache. *Cephalalgia*, 12, 365–368.
- Solomon, G. D., Skobieranda, F. G., & Gragg, L. A. (1994). Does quality of life differ among headache diagnoses? Analysis using the Medical Outcomes Study instrument. *Headache*, 34, 143–147.
- Stang, P. E., & Osterhaus, J. T. (1993). Impact of migraine in the United States: Data from the National Health Interview Survey. *Headache*, 33, 29–35.
- Steiner, T. J., Mülleners, W. M., Whitmarsch, T. E., Couturier, E. G. M., Catarci, T., & Hering, R. (1994). Do we know what our patients are taking? In L. A. H. Hogenhuis & T. J. Steiner (Eds.), *Headache and migraine* (Vol. 3, pp. 63–68). Utrecht, Netherlands: Wetenschap-pelijke uitgeverij Bunge.
- Stewart, W. F., & Lipton, R. B. (1997). Work-related disability: Results from the American Migraine Study. *Cephalalgia*, 16, 231–238.
- Stewart, W. F., Lipton, R. B., Celentano, D. D., & Reed, M. L. (1992). Prevalence of migraine headache in the United States: Relation to age, income, race, and other sociodemographic factors. *JAMA*, 267(1), 64–69.
- Symon, D. N. (1998). Twelve cases of analgesic headache. *Archives of Disabilities in Children*, 78, 555–556.
- Symon, D. N. K., & Russell, G. (1986). Abdominal migraine: A childhood syndrome defined. *Cephalalgia*, 6, 223–228.
- Teders, S. J., Blanchard, E. B., Andrasik, F., Jurish, S. E., Neff, D. F., & Arena, J. G. (1984). Relaxation training for tension headache: Comparative efficacy and cost-effectiveness of a minimal therapist contact versus a therapist-delivered procedure. *Behavior Therapy*, 15, 59–70.

- Teskey, G. C., & Cain, D. P. (Eds.). (1989). Recent developments in kindling: proceedings of a satellite symposium to the 18th annual meeting of the Society for Neuroscience. *Neuroscience and Biobehavioral Reviews*, 13(4), 247–322.
- Tfelt-Hansen, P. (1993). Sumatriptan for the treatment of migraine attacks: A review of controlled clinical trials. *Cephalalgia*, 13, 238–244.
- Tissot, S. (1780). *Traité des nerfs et de leurs maladies*. Paris: P. F. Didot jeune.
- Turk, D. C., & Rudy, T. E. (1991). Neglected topics in the treatment of chronic pain patients—relapse, noncompliance, and adherence enhancement. *Pain*, 44, 5–28.
- Vahlquist, B. (1955). Migraine in children. *International Archives of Allergy*, 7, 348–355.
- Vasconcellos, E., Piña-Garza, J. E., Millan, E. J., & Warner, J. S. (1998). Analgesic rebound headache in children and adolescents. *Journal of Child Neurology*, 13, 443–447.
- Wasiewski, W. (2001). Preventive therapy in pediatric migraine. *Journal of Child Neurology*, 16(2), 71–78.
- Weiss, S. R. B., & Post, R. M. (1998). Kindling: Separate versus shared mechanisms in affective disorders and epilepsy. *Neuropsychobiology*, 38(3), 167–180.
- Welborn, C. A. (1997). Pediatric migraine. *Emergency Medical Clinics of North America*, 15, 625–636.
- White, K. S., Alday, C. S., & Spirito, A. (2001). Characteristics of children presenting to a behavioral treatment program for pediatric headache. *Journal of Clinical Psychology in Medical Settings*, 8(2), 109–117.
- Williamson, D. A., Baker, J. P., & Cubic, B. A. (1993). Assessment in pediatric headache research. In T. H. Ollendick & P. J. Prinz (Eds.), *Advances in clinical child psychology* (Vol. 15, pp. 275–304). New York: Plenum.
- Winner, P., Wasiewski, W., Gladstein, J., & Linder, S. (1997). Multicenter prospective evaluation of proposed pediatric migraine revisions to the IHS criteria. *Headache*, 37, 545–548.
- Wisniewski, J. J., Genshaft, J. L., Mulick, J. A., Coury, D. L., & Hammer, D. (1988). Relaxation therapy and compliance in the treatment of adolescent headache. *Headache*, 28, 612–617.
- Ziegler, D. K., Hur, Y. M., Bouchard, T. J., Jr., Hassanein, R. S., & Barter, R. (1998). Migraine in twins raised together and apart. *Headache*, 38, 417–422.

