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Psychiatric Symptomatology in Migraine Sufferers and the Relationship with Headache-Related Self-Efficacy

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Psychiatric Symptomatology in Migraine Sufferers and the Relationship with Headache-Related Self-Efficacy

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Abstract

Introduction: Self-efficacy (SE) refers to one's belief in their ability to successfully complete a task. Increases in SE are associated with improved pain outcomes as well as improvements in anxiety and depression (Turner, 2005). Headache-related self-efficacy (HSE) refers to a person's belief in his/her ability to manage migraine attacks. Previous studies illustrate that HSE and psychiatric comorbidities are independently related to migraine disability, severity, and treatment outcomes. The purpose of the present study was to assess the relationship between HSE and psychiatric comorbidities among individuals with migraine.

Methods: 852 young adult migraineurs (M age = 19.22; 79.9% female; 77.0% Caucasian) completed measures about headache and related variables, including the Headache Management Self-Efficacy Scale (HMSE; French, 2000), the Depression Anxiety Stress Scale (DASS-21; Lovibond, 1995), and the Post-traumatic Stress Disorder Checklist (PCL; Wilkins, 2011). Of the 852 migraine participants, 56.8% met criteria for migraine without aura, 25% met for migraine with aura, and 18.2% met for chronic migraine. Linear regressions were run to identify associations between psychiatric symptoms (depression, anxiety, and PTSD) and HSE.

Results: Men reported higher HSE than women (M = 108.67 [18.91] vs. 104.66 [21.28], $p = .025$). Comorbid psychiatric symptoms explained a significant proportion of variance in HSE scores ($R^2 = .047$, $p < .001$). When controlling for sex, headache frequency, and disability, the percentage of unique variance accounted for by psychiatric comorbidities was 1.5% ($p = .005$) of the total.

Conclusion: Consistent with our hypothesis, a significant relationship exists between comorbid psychiatric symptoms and HSE scores, but psychiatric symptoms only accounted for a small amount of variance in HSE. This small effect size likely indicates that numerous variables influence self-efficacy, and that psychiatric symptoms represent a relatively weak influence; perhaps also stronger relations would be evident in a clinical sample. Future research into variables that affect headache-related self-efficacy would be valuable for piecing together a better understanding of this powerful predictor of headache prognosis.

Table of Contents

3. Strengths, Limitations, and Future Directions

References

Tables

Appendix

Psychiatric Symptomatology in Migraine Sufferers and the Relationship with Headache-Related Self-Efficacy

By: Lindsay W. Wencel

Migraine

Diagnosis

Migraine sufferers experience headache attacks lasting between 4 and 72 hours if untreated (International Headache Society [IHS], 2013). The pain is characterized by two or more of the following; unilateral location, pulsating/throbbing pain, moderate to severe intensity, or aggravation by normal activity. These attacks must also be accompanied by nausea or vomiting or both photophobia (sensitivity to light) and phonophobia (sensitivity to sound). The headache must also not be attributable to any other condition such as substance use, head/neck trauma, or another disorder. Migraine is divided into two subtypes, episodic and chronic, based upon frequency of headache attacks. Episodic migraine occurs on less than 15 days per month, while chronic migraine exhibits the above characteristics but has a frequency of at least 15 headache days per month, with migraine symptoms on at least eight of those days. For the condition to be considered chronic, this frequency must be maintained for a period of time greater than three months.

Migraine with aura is less common than migraine without aura, affecting 25% of all migraine sufferers. Aura presents as temporary symptoms that are precursors of the actual headache attack, and the patient must have experienced at least two headaches with aura (IHS, 2013). The aura symptoms are usually visual in nature (e.g., blurred vision, zigzag lines), though sensory, speech/language, motor, brain stem or retinal disturbances occur in a minority of those

with aura. Aura symptoms typically develop gradually over 5-20 minutes, last less than an hour, and then resolve coinciding with onset of head pain.

Prevalence and Burden

Migraine is very common, with data from the World Health Organization indicating that migraine is the third most prevalent medical condition worldwide (Vos, Flaxman, Naghavi, Lozano, Michaud, and Ezzati, 2012). In the US, migraine has a lifetime prevalence of 43% for women and 18% for men (Stewart, Wood, Reed, Roy, & Lipton, 2008). Episodic migraine is the most common form of migraine and was found in a 2012 survey study of more than 160,000 American adults to have a yearly prevalence of 12% (Buse et al., 2012). Chronic migraine is far less common than episodic, affecting approximately 1% of the general population.

Both nationally and globally, migraine is a significant source of disruption in sufferers' lives, leading to a decrease in overall work, school, home, and personal functioning (Vos et al., 2012). Financially, migraine costs the US between $13 and $17.2 billion each year when considering direct and indirect expenses related to the disorder (Pesa & Lage, 2004). Headache is the fifth most common cause of emergency room visits nationally and accounts for a large proportion of medical expenses each year (Pesa & Lage, 2004; Smitherman, Burch, Sheikh, & Loder, 2013). Individually, migraineurs spend more than twice their non-migraine counterparts on medical care each year (Pesa & Lage, 2004). They also have more sick days and an overall decrease in job performance compared to those without migraine (Pesa & Lage, 2004; Singh, 2014).

Migraine causes reductions in school or work performance, household responsibilities, and social and leisure activity participation (Leonardi, 2010). A World Health Organization

study found migraine to be the 8th most disabling condition worldwide (Vos et al., 2012), and 66-93% of migraine sufferers experience moderate to severe disability (Bera, Khandelwal, Sood, & Goyal, 2014; Lipton et al., 2007). Migraine is also associated with decreased quality of life (Leonardi, Raggi, Bussone, & D'Amico, 2010; Paschoal et al., 2013), and this burden increases with migraine frequency. People with chronic migraine experience higher disability than those with episodic migraine (Buse et al., 2012). Other factors associated with increased migraine disability associated include female gender, high stress, symptom severity, and presence of psychiatric comorbidity (Buse, Silberstein, Manack, Papapetropoulos, & Lipton, 2013).

Pathophysiology

Several hypotheses for the causes of migraine have been presented, but currently there is not a consensus for its pathophysiology. One of the earliest hypotheses for the cause of migraine was cerebral vasoconstriction followed by vasodilation (Horváth, 2014). This vascular theory seemed to be supported by the efficacy of triptans for migraine, which effect vascular changes in the brain. Further research has discredited the vascular theory by showing that vascular changes are not a primary cause of headache but instead secondary to other initiating events. The current accepted theory of migraine is the neurovascular theory, which asserts that migraine is attributable to a hypersensitive central nervous system and influenced both by brain stem and cortical structures.

Genetics also play a role in migraine, which has a heritability of 30–60%, with a higher probability for inheritance for migraine with aura (Chasman et al., 2014). Chasman et al. (2014) identified 12 single nucleotide polymorphisms (SNPs) that show evidence of association with migraine in women. Their research also discovered SNPs associated with migraine

nonspecifically, migraine with aura, specific migraine characteristics, and development of other body systems involved in migraine, such as neurotransmitter systems and vascular development. This information has the potential to open new avenues for migraine diagnosis and treatment, but a comprehensive account of migraine pathophysiology remains elusive.

Treatment

Management of attacks is crucial to the well-being of individuals who experience migraine and is divided into preventive and acute treatment. Preventive treatment is used to decrease the frequency of attacks. When patients experience frequent migraine attacks (at least 4-6 attacks per month) or require acute treatment more than twice in a week period, then a preventive medication is indicated (Singh, 2014). The most effective of these medications are beta-blockers, tricyclic antidepressants, and anti-convulsants, taken on a daily basis. Along with pharmacologic interventions, behavioral modifications such as improving sleeping habits, exercising, stopping smoking, managing weight, and avoiding triggers are also recommended (Lipton et al., 2007; Singh, 2014). Psychological treatments, such as relaxation training, various forms of biofeedback, and cognitive behavioral therapy, have also shown efficacy for migraine similar to that observed with the best preventive medications (Wells & Loder, 2012).

Acute treatments are used in the event of a migraine attack and usually shorten the duration of the attack and improve symptoms. Triptans are the most commonly used agents and are effective in treating attacks if used early enough in the episode (Singh, 2014). They have cardiovascular contraindications and side effects, which prohibit some sufferers from utilizing them. In cases of severe migraine, anti-emetics and ergot derivatives are also used.

When comorbid disorders are present, treating the comorbid disorder can also lead to a reduction in migraine attacks as well as severity of attacks. A study by Calhoun et al. (2007) found that treating sleep disturbance in individuals with chronic migraine and insomnia led to a reduction in headache frequency. Similar results have also been illustrated in cases of comorbid obesity (Bond, Roth, Nash, & Wing, 2011).

Psychological Factors in Migraine

Several studies have shown that psychiatric symptoms commonly occur in migraine sufferers, with 25- 62.5% of all sufferers having a lifetime diagnosis of at least one psychiatric comorbidity (Bera et al., 2014; Buse et al., 2013; Semiz, Sentürk, Balaban, Yagiz, & Kavakçi, 2013). Psychiatric disorders are two to ten times as prevalent in those with migraine as in the general population (Buse et al., 2013). Comorbidities are significantly more common in female migraineurs than their male counterparts, likely due to hormonal and serotonin influences on both conditions (Baskin & Smitherman, 2009). Studies have demonstrated that psychiatric comorbidities have an adverse impact on headache-related disability, headache severity, quality of life, and both treatment adherence and outcomes (Baskin & Smitherman, 2009; Semiz et al., 2013). Comorbid psychiatric symptoms also increase the likelihood of headache chronification, or the transition from episodic to chronic frequency of migraine (Buse et al., 2013). Treatment of migraine with psychiatric comorbidities is further complicated due to the fact that several common medications for migraine exacerbate psychiatric conditions (Finocchi, Villani, & Casucci, 2010).

Increased rates of psychiatric comorbidities occur not only among older treatment-seeking patients but also among young adult or college student migraineurs. In a study by Semiz

et al. (2013), of 169 college students who experience migraine, at least one diagnosable psychiatric disorder was found in 23.1% of the participants. Further, 43.2% of migraineurs surveyed had a psychiatric diagnosis at some point in their lives. Those individuals with comorbidities were also found to have more frequent, severe and disabling headaches.

Anxiety

Several studies have illustrated that, of all the affective disorders, anxiety has the strongest association with migraine (Baskin & Smitherman, 2009; Hamelsky & Lipton, 2006). Comorbid anxiety is associated with increased headache intensity and disability as well as significantly reduced quality of life (Baskin & Smitherman, 2009; Oh, Soo-Jin Cho, Yun, Jae-Moon Kim, & Min, 2014; Paschoal et al., 2013). Anxiety is a strong contributing factor to migraine persistence, chronification and worsened overall prognosis (Baskin et al., 2009; Buse et al., 2013). Comorbid anxiety is also associated with doubled total medical costs compared to having migraine without anxiety (Pesa & Lage, 2004).

The prevalence of generalized anxiety disorder (GAD) and subclinical anxiety symptoms among those with migraine differs by population, with between 5-34% of sufferers presenting diagnosable GAD (Bera et al., 2014; Bhatia & Gupta, 2012; Semiz et al., 2013. In general, migraine sufferers are between 4 and 5 times as likely to have GAD as those without migraine (Semiz et al., 2013). Similarly, panic disorder, another anxiety disorder, is 3.76 times as likely in migraineurs than in the general population (Smitherman, Kolivas, & Bailey, 2013). This increased in prevalence of anxiety disorders could be due to an increase in stress from migraine attacks, anxiety operating as a migraine trigger, or maladaptive coping behaviors that develop in response to headaches (Radat et al., 2008). The relationship between migraine and anxiety has

been shown to be bidirectional, in that people with anxiety are also more likely to develop migraine (Baskin & Smitherman, 2009; Hamelsky et al., 2006).

Depression

Between 14-37.5% of migraineurs experience depression or depressive symptoms (Baskin et al., 2009; Bera et al., 2014; Bhatia et al., 2012; Semiz et al., 2013). Several studies have shown that people with migraine are between 2-4 times as likely to develop depression when compared with the general population (Baskin & Smitherman, 2009, Buse et al., 2013; Hamelsky et al., 2006), and rates are highest among migraineurs with aura and those with chronic migraine.

As with anxiety, there is a bidirectional relationship between migraine and depression, but it is slightly more common to develop depression after migraine than vice versa (Hamelsky et al., 2006). The high frequency of depression with migraine may be due to the unpredictability of migraine attacks and the perceived inability to control attacks, as well as the effects of reduced serotonergic transmission seen in both conditions. (Baskin & Smitherman, 2009; Hamelsky et al., 2006). As with anxiety, depression is linked with higher medical costs. (Pesa & Lage, 2004).

Comorbid depression is related to an increase in frequency, symptomatology and severity of migraine, as well as, an increased risk for chronification (Buse et al., 2013; Ching-I Hung, Chia-Yih Liu, Yeong-Yuh Juang, & Shuu-Jiun Wang, 2006; Finocchi et al. , 2010; Hamelsky et al., 2006). Migraineurs with depression also have a marked decrease in functioning and overall quality of life and an increase in headache-related disability (Hamelsky et al., 2006) (Paschoal et al., 2013). Furthermore, migraine sufferers have more severe depression and higher risk of suicide attempts than those with depression but without migraine (Baskin & Smitherman, 2009)

(Buse et al., 2013). Treatment of migraine has been shown to have positive effects on depressive symptoms (Buse et al., 2013), but most studies of antidepressant migraine agents have excluded depressed patients.

Combined Anxiety and Depression

Both anxiety and depression occur jointly with migraine, specifically in those individuals with chronic migraine (Hamelsky et al., 2006; Radat et al., 2008). Mixed anxiety and depression has been found to have a prevalence of 18% in migraineurs (Bhatia et al., 2012). A population study of almost 3000 participants found that two-thirds of all migraine sufferers with depression, also had anxiety, and that one-third of sufferers with anxiety also had depression (Oh et al., 2014). Individuals presenting with both anxiety and depression are at twice the risk of developing migraine than their non-anxious, non-depressed counterparts (Hamelsky et al., 2006).

The combination of anxiety and depression has been related to increased headache frequency and higher risk of migraine chronification (Oh et al., 2014). Individuals with both anxiety and depression also take significantly more acute medications and experience decreased effectiveness of those medications (Radat et al., 2008). This leads to an even more significant increase in stress and decrease in quality of life than either comorbid anxiety or depression alone (Paschoal et al., 2013; Radat et al., 2008). In the case of this dual-comorbidity, treatment of one of these disorders has the potential to improve all three conditions (Hamelsky et al., 2006).

Post-traumatic Stress Disorder

Post-traumatic stress disorder is a psychiatric disorder characterized by exposure to a traumatic event or events, including death, threatened death, actual or threatened serious injury,

or actual or threatened sexual violence (American Psychiatric Association [APA], 2013). This exposure must be followed by persistently re-experiencing the event via intrusive recollections, dissociative reactions, nightmares, increased physiologic reactivity, or distress at exposure to traumatic reminders and the development of avoidance behavior. Individuals with PTSD also must experience negative changes in thought process and mood as well as arousal and reactivity as a result of the trauma they experienced. These symptoms must persist of a period of time greater than a month and cannot be related to any other physical or psychological disorder. In the general population, PTSD has a lifetime prevalence of 8.3% (Kilpatrick et al., 2013). PTSD is significantly more prevalent in migraine patients than in the general population (Peterlin et al., 2009). Numerous studies have demonstrated a strong association between PTSD and migraine, with prevalence rates between 22-50% of migraine sufferers (Buse et al., 2013; Semiz et al., 2013).

Traumatic events are more common among those with migraine than in the general population. A study by Peterlin et al. (2007), examining headache patients presenting at tertiary care clinics, found that 27.3% of migraine patients had experienced some form of physical or sexual abuse. Peterlin et al. (2008) studied a group of 62 migraine patients and found diagnostic criteria for PTSD were met in 12.5% of episodic migraineurs and in 42.9% chronic migraineurs (Peterlin, Tietjen, Meng, Lidicker, & Bigal, 2008). Another study of 80 migraine and tension-type headache patients found similar results, with 25% of patients exhibiting evidence of current PTSD (De Leeuw, Schmidt, & Carlson, 2005). These studies, as well as others, indicate that PTSD is significantly more common in those with chronic migraine (Buse et al., 2013; Peterlin et al., 2008). Unlike other psychiatric comorbidities, the dual diagnosis of migraine and PTSD is significantly more prevalent in men (Peterlin, Calhoun, & Balzac, 2012). Further study by

Smitherman et al. (2013) determined that PTSD, not merely exposure to trauma, is most associated with migraine diagnosis.

Comorbid PTSD is associated with higher risk for chronification of migraine and intensified headache severity (Peterlin et al., 2009; Peterlin et al., 2008; Smitherman & Kolivas, 2013). Post-traumatic symptoms are positive correlated with headache-related disability, so as PTSD worsens so does migraine disability (Smitherman & Kolivas, 2013). PTSD has also been linked to poorer coping mechanisms and prognosis as well as a heightened risk of developing medication overuse headache. Post-traumatic symptoms have also been associated with new or worsening depression in migraine patients (Peterlin et al., 2009). Considered in conjunction, psychological disorders and symptoms are critical in understanding and treating migraine. Most recently, interest in psychological factors has expanded beyond disorders and into other psychological constructs, such as self-efficacy.

Self-efficacy

Self-efficacy is a person's belief in her ability to complete a given task or achieve a desired outcome (Bandura, 2014; Yancey, 2014). It is the basis for one's interactions with their environment, effecting initiation, persistence, and success in all ventures. Self-efficacy is developed based upon past experiences of success or failure, witnessed success or failure of others, and other's opinions about a person's abilities. Individuals with high self-efficacy believe that they are in control of their circumstances, which leads to enhanced task performance and, in turn, additional increases in self-efficacy. Conversely, those with low self-efficacy believe that success is out of their hands and expect failure when attempting new tasks. This defeatist mindset often leads to failure and worsening of self-efficacy, or an unwillingness to even attempt

a certain action. Stress and anxiety can lead to marked decrease in task performance and self-efficacy, which can precipitate or worsen anxiety and depression and lead to a continuous cycle of anxiety, failure, and low self-efficacy.

Improving self-efficacy has been shown to have positive effects on medical outcomes. In a review article by Magklara et al. (2014), post-operative self-efficacy in joint replacement patients was a significant determinant of both short and long term recovery outcomes. A study by Turner et al (2005), consisting of 140 elderly adults with chronic pain, found that increased self-efficacy was modestly, but significantly, associated with decreases in disability and depressive symptoms. Furthermore, a study by Hadjistavropoulos et al. (2007) of recurrent pain in a college population found that heightened self-efficacy was highly correlated with improved coping methods and functioning. High self-efficacy was also negatively correlated with pain intensity and impairment, as well as negative affect. Self-efficacy, and the positive outcomes related to it, has similar value in migraine sufferers as in other pain patients.

Headache-Related Self-Efficacy

Headache-related self-efficacy is defined as one's confidence in her ability to properly manage and prevent headaches (Seng & Holroyd, 2010). Research into headache-related self-efficacy is fairly new area of study, but early studies have provided noteworthy results. Headache severity and disability are inversely correlated with headache-related self-efficacy (Paschoal et al., 2013), and high self-efficacy is also correlated with a decrease in anxiety (French et al., 2000). Conversely, low self-efficacy has been related to decreased pain tolerance (French et al., 2000).

Significant evidence exists showing psychological treatments, such as behavioral migraine management strategies; improve headache-related self-efficacy in episodic migraineurs (Nicholson, 2005; Seng & Holroyd, 2010; Sorbi, Kleiboer, Van Silfhout, Vink, & Passchier, 2014). A study of almost 200 migrainuers by Sorbi et al. (2014) found that online behavioral training in headache management significantly improved both headache and self-efficacy over a 10-month period. Psychological treatments for improving headache-related self-efficacy have also shown efficacy for individuals with chronic migraine (Thorne et al., 2007). Further, Voerman et al. (2014) found that gains in headache outcomes and headache-related self-efficacy were maintained 2-4 years later. Improved headache-related self-efficacy has also been associated with utilization of positive coping strategies, along with better treatment adherence and improved prognosis (French et al, 2000; Seng & Holroyd, 2010). Although there has been significant research in to headache-related self-efficacy's relationship to headache outcomes, few studies have explored variables that contribute to headache-related self-efficacy itself.

Goal of this Study

Previous studies have demonstrated that both psychiatric comorbidities and headache-related self-efficacy have significant relationships with migraine disability, severity, and treatment outcomes. Improvements in general self-efficacy are associated with improved outcomes and reductions in anxiety and depression in pain patients (Hadjistavropoulos et al, 2007; Turner et al., 2005). Similar results have been found in migraine sufferers (French et al., 2000; Peck, 2013; Seng & Holroyd, 2010), but studies are lacking exploring how headache-related self-efficacy is associated with psychiatric comorbidities in migraineurs. This study's

purpose is to examine the possible association between psychiatric comorbidities and headache self-efficacy among individuals with migraine.

Hypothesis

Study Goal 1: to investigate the association between headache self-efficacy and psychiatric comorbidities among individuals with migraine.

Hypothesis 1a: Migraineurs with psychiatric comorbidity will have lower headache-related self-efficacy than those without psychiatric comorbidity.

Hypothesis 1b: Specific psychiatric comorbidities, such as anxiety, depression and PTSD, will have significant relationships with headache-related self-efficacy.

Methods

Participants

Participants were undergraduates recruited from introductory psychology courses at the University of Mississippi, using an online research management program, from 2011-2014. Those who chose to participate completed an online battery including questionnaires about psychiatric symptoms, specifically anxiety, depression, and PTSD, as well as a computerized headache diagnostic interview. Participants with primary episodic and chronic migraine (ICHD-II codes 1.1, 1.2, 1.5.1, 1.6) diagnoses after exclusion of possible secondary causes and adherence to ICHD-II diagnostic criteria were retained for further study. Migraine criterion B, which dictates the duration of headache required to qualify as migraine, was decreased from 4 to 2 hours due to previous studies indicating that young adults commonly experience migraine attacks with all other criteria but that are shorter than 4 hours (Rains, Penzien, Lipchik, &

Ramadan, 2001; Rasmussen, Jensen, & Olesen 1991). Any participant with incomplete batteries in target areas was excluded.

Materials

The Depression Anxiety Stress Scales (DASS-21) The DASS was originally a 42-question scale, with anxiety (DASS-A), depression (DASS-D), and stress (DASS-S) subscales created to quantify negative emotions over a week period (Antony, Bieling, Cox, Enns, & Swinson, 1998). A shorter version, the DASS-21 was later released by Lovibond and Lovibond to decrease the time required for administration (Lovibond & Lovibond, 1995). The DASS-21 is a likert-type scale with high reliability and validity across populations (Oei, Sawang, Goh, & Mukhtar, 2013; Weiss, Aderka, Lee, Beard, & Björgvinsson, 2014). The DASS-21 has also shown validity across cultures (Mellor et al., 2014). The DASS-D is sensitive to alterations in depressive symptomatology (Weiss et al., 2014) and shows efficacy for screening PTSD in certain populations (Kok, De Haan, Van, Najavits, & De Jong, 2015). This measure is located in Appendix B.

Headache Impact Test (HIT-6) The HIT-6 is a 6-item self-report survey for determining disability attributable to headache, in terms of changes in functioning and quality of life due to headache (Rains et al., 2001). The HIT-6 evaluates interference in cognitive, role, and social functioning as well as pain and psychological distress. This scale has strong validity and reliability in both episodic and chronic migraine sufferers (Rains, et al. 2001; Yang, Rendas-Baum, Varon, & Kosinski, 2011). This measure is located in Appendix C.

Headache Management Self-Efficacy Scale (HMSE) The HMSE is a headache specific measure of self-efficacy, consisting of 25 questions, rated on a 1-7 likert-type scale (French et al., 2000). These questions quantify the confidence of those with headache in their own ability to prevent and manage headache attacks. The HMSE has been found to be both reliable and valid (Hansen, Bendtsen, & Jensen, 2009). This measure is located in Appendix D.

The Post-traumatic Stress Disorder Checklist (PCL) The PCL is a 17 Likert-type item questionnaire that evaluates PTSD symptoms over the last month (Wilkins, Lang, & Norman, 2011) resulting from experienced traumas. Three different forms of the PCL exist, one for military personnel (PCL-M), one for civilians (PCL-C), and one for specific traumatic events (PCL-S). This study utilized the PCL-C, which has been found to be highly reliable and have high validity (Conybeare, Behar, Solomon, Newman, & Borkovec, 2012; Wilkins et al., 2011). This measure is located in Appendix E.

Structured Diagnostic Interview for Headache-Revised (SDIH-R) The SDIH is a computer-administered diagnostic interview for all major forms of (Andrew, Penzien, Rains, Knowlton, & McAnulty, 1992). The initial SDIH was a significant improvement in reliability and validity from previous diagnostic techniques. The measures utilized for the study was the SDIH-II, a more recent version revised to fit new International Headache Society parameters for headache diagnosis. This measures is located in Appendix A.

Procedure

Participants were undergraduate students enrolled in psychology courses at The University of Mississippi. They provided informed consent and completed the online measures as part of a larger battery and in exchange for modest course credit.

Statistical Analyses

The study sample was summarized by descriptive statistics and distributions were examined. To understand how headache-related self-efficacy as well as other pertinent variables (anxiety, depression, headache disability, and PTSD) differed between migraine diagnoses, one-way analysis of variance (ANOVA) tests were performed. Additionally, post-hoc tests of significant ANOVA results were conducted to determine which groups differed from each other. Finally, after collapsing all migraine subtypes, linear regressions were performed to examine the amount of variance in HSE scores attributable to the psychiatric symptoms.

Results

Participant Exclusions, Demographics, and Headache Diagnosis

Five thousand five hundred and eighty-three undergraduate students, ranging in age from 18 to 46, initially participated in this study. Of those participants, 286 did not complete the appropriate measures to properly determine a headache diagnosis and were excluded. Additionally, all participants without headache and with other headache subtypes other than migraine and its variants were excluded [nonheadache (n = 1533), probable migraine (n = 1017), chronic TTH (n = 64), episodic TTH (n = 884), probable TTH (n = 791), cluster headache (n = 82), MOH (n = 11)]. Of the remaining 1043 participants, 191 had incomplete self-efficacy data

and were excluded. The remaining 852 with ICHD-II congruent migraine made up the final sample.

The sample had a mean age of 19.22 years (*SD* = 2.43) and was comprised of 79.9% females. The sample was predominantly Caucasian (77%) followed by African American (17.4%), Hispanic/Latino (2%), Multiracial (1.6%), Asian (1.4%), Native American/ Alaskan (0.5%), and Pacific Islander/Native Hawaiian (0.1%). Headache diagnosis for retained participants was primarily episodic migraine without aura (n= 484, 56.8%), followed by episodic migraine with aura (n= 213, 25%), and chronic migraine (n= 155, 18.2%).

Covariates

To assess whether the main variables of interest differed as a function of gender, t-tests were conducted comparing males and females on the ASI total, DASS subscales, HIT-6, HMSE, and PCL (see Table 1). Of these, only headache self-efficacy differed as a function of gender, with men reporting higher self-efficacy than women (M = 108.67 [18.91] vs. 104.66 [21.28], p = .025). Gender was thus used as a covariate in later regression analyses. Headache frequency was also used as a covariate, given that those with CM have higher frequency than those with EM and because frequency was significantly correlated with self-efficacy ($r = -.25$, $p < .001$). Finally, disability was used as a covariate because of previously established relationships between disability and self-efficacy (Peck, 2013).

Headache Variables

Headache Disability. As expected, a significant relationship existed between self-efficacy and disability ($R^2 = .061$, $p < .001$). Table 2 presents the group differences on the main variables of

interest. Headache-related disability (HIT-6) scores differed significantly as a function of migraine diagnosis ($p < .001$). Episodic migraineurs without aura reported less disability compared to those with episodic migraine with aura ($p < .001$) as well as those with chronic migraine ($p < .001$). Additionally, chronic migraineurs had higher scores than those with episodic migraine with aura ($p = .022$).

Headache-Related Self-Efficacy. Headache-related self-efficacy also differed significantly by migraine diagnosis ($p < .001$). Fisher's least significant difference (LSD) post-hoc test illustrated that individuals with chronic migraine reported significantly lower self-efficacy than those with episodic migraine without aura ($p < .001$) and with aura ($p < .001$).

Psychiatric Symptomatology Across Headache Groups. Anxiety (DASS anxiety subscale) scores differed significantly by migraine diagnosis ($p < .001$). Fisher's LSD post-hoc test showed that this difference was due to the fact that individuals with episodic migraine without aura had significantly lower scores than those with episodic migraine with aura ($p < .001$) as well as those with chronic migraine ($p < .001$). Table 1 provides the mean scores on the DASS-A and other psychological measures as a function of migraine group.

As with the anxiety scale, depression (DASS depression subscale) scores also differed significantly by migraine diagnosis ($p < .001$). Post-hoc tests indicated that this difference was due to episodic migraineurs without aura scoring reporting significantly fewer symptoms of depression than those with episodic migraine with aura ($F(2,828) = 2.133$, $p = .002$), as well as those with chronic migraine ($F(2,828) = 3.079$, $p < .001$).

Post-traumatic stress symptoms (PCL scores) also differed significantly between the migraine subtypes (p = .001). Post-hoc tests confirmed that individuals with episodic migraine without aura reported fewer symptoms of PTSD than those with episodic migraine with aura ($F(2,793) = 4.138$, $p = .001$) and those with chronic migraine ($F(2,793) = 3.482$, $p = .012$).

Psychiatric symptoms and Self-efficacy

When examining the relationship between psychiatric comorbidities and headache-related self-efficacy, linear regression analysis revealed a modest but significant relationship (see Table 3). Comorbid psychiatric symptoms of anxiety, depression, and PTSD explained a significant proportion of variance in self-efficacy scores, $R^2 = .047$, $F(3, 763) = 12.412$, $p < .001$. To determine whether psychiatric symptoms remained associated with self-efficacy after controlling for the aforementioned covariates (i.e., gender, headache frequency, and disability), a hierarchical linear regression was conducted. Covariates were entered in block 1, and the psychiatric symptomatology scores were entered in step 2. Even after controlling for covariates, psychiatric comorbidities remained significantly associated with headache self-efficacy, though the percentage of unique variance accounted for was small (1.5%; p = .005).

Discussion

Both psychological comorbidities and headache-related self-efficacy have been shown to affect disability, chronification, and treatment outcomes in migraine patients, but rarely has the interaction between these two domains been explored. The present study explored the relationship between psychological comorbidities and headache self-efficacy among individuals with migraine.

Headache Variables and Classification

Analysis of various headache variables found significant differences within migraine subtypes in all variables studied. Specifically, people with chronic migraine experienced significantly increased headache-related disability, self-efficacy, and frequency in comparison with those diagnosed with episodic migraine with and without aura. The episodic migraine with aura group also showed significant increases in the areas of frequency, disability, PTSD, depression, and anxiety when compared those with migraine without aura. These results were comparable with previous studies of the same type (Bigal, Serrano, Reed, & Lipton, 2008; Lipton et al., 2007), and yet are novel in understanding symptomatology of migraine at different levels of frequency and as a function of aura.

Psychiatric Comorbidities and Headache-Related Self-Efficacy

Consistent with our hypothesis, a significant inverse relationship was found between comorbid psychiatric symptoms and headache-related self-efficacy scores. This indicates that as psychiatric symptomatology increased, headache self-efficacy decreased proportionately. This relationship, though significant, was rather modest, with psychiatric symptoms only accounting for a small amount of variance in self-efficacy scores.

Most past studies of self-efficacy have exclusively looked at its relationship with treatment outcomes (Sorbi et al., 2014; Thorne et al., 2007; Voerman et al., 2014), but our results contributes a non-clinical perspective. A better understanding of headache self-efficacy outside of behavioral treatment is necessary for discerning on which headache variables self-efficacy has the greatest influence. By examining how headache-related self-efficacy relates to not only

migraine, but also comorbid disorders, we have gained a better understanding of both conditions. According to a review article by Wang, Chen, & Fuh (2010), there are 11 additional well-established comorbid conditions with migraine. By examining the influence of headache variables on multiple comorbid conditions, the understanding of their relationship with self-efficacy and headache impact can be improved.

Finding only a modest association led us to question what other factors act as contributors of variance in headache-related self-efficacy. Headache frequency would be expected to have a large influence on self-efficacy because of its association with negative outcomes, and indeed its influence was reflected in the modest differential self-efficacy scores as a function of episodic versus chronic migraine frequency.

The finding that two factors, psychiatric symptoms and frequency of headache attacks, previously considered to be major contributors to headache-related self-efficacy had only a modest association with self-efficacy was an unexpected finding. This finding suggests that headache-related self-efficacy is likely influenced by multiple variables, each accounting for small yet significant proportions of variance. In this notion, frequency of attacks and psychiatric comorbidities represent only two factors out of many others that collectively influence a migraineur's headache self-efficacy. Previous studies reinforce this notion. For instance, both fear of pain and acceptance show modestly significant relationships with headache self-efficacy (Carpino, Segal, Logan, Lebel, & Simons, 2014; Kalapurakkel, S., Carpino, E.A., Lebel, A. & Simons, L.E., 2014) but were not assessed in the present study. Other variables, such as anxiety sensitivity, severity of headache pain, and average duration of headache attack, may also be valuable in predicting self-efficacy in migraine.

Strengths and Limitations

This study had the benefit of a large and relatively diverse sample. Unlike past studies of the same type, this study utilized the full diagnostic interview for headache to determine migraine diagnosis, which lends increased validity to the results. Additionally, this study assessed the impact of several psychiatric symptoms at the same time. These variables are rarely examined concurrently and by doing so, we were able to gain a greater understanding of how they impact each other as well as self-efficacy.

Despite these strengths, the present study has limitations. Because our data were collected from a college population of young adults, external validity as applied to older adults and treatment-seeking migraineurs is unclear. Possibly a larger effect of these psychiatric symptoms would be obtained among individuals seeking treatment. Additionally, since this is a cross-sectional study, causality cannot be determined in the relationship between headache self-efficacy and the psychiatric symptoms.

Future Directions

Future research into variables that affect headache-related self-efficacy would be valuable for piecing together a better understanding of its role in headache prognosis. Specifically, examining other headache variables such as fear of pain, headache severity, and attack duration could provide additional insight into influences on self-efficacy that were not assessed in this study. Another area of study to consider would be the relationship between self-efficacy and medication adherence. Improved self-efficacy has been tied to better treatment adherence in other headache conditions, specifically chronic tension-type headache, and similar ameliorations may be found in migraine (Holroyd, Labus, & Carlson, 2009). These improved outcomes may

occur because if patients are confident in their ability to actually control their headache pharmacologically they may have greater adherence to their treatment regimen. Additionally, examining the relationship between headache self-efficacy and locus of control could prove valuable in furthering out understanding migraine. As locus of control is a similar construct to self-efficacy, investigating which of these variables has higher predictive utility in headache variables and outcomes could prove useful.

With a better understanding of self-efficacy, non-pharmacological treatments could be improved to enhance migraine outcomes by specifically targeting self-efficacy. Increasing self-efficacy as part of a non-pharmacological treatment could provide additional headache improvements insofar as it would facilitate adherence to treatment and utilization of skills during attacks.

In conclusion and consistent with our hypothesis, a significant relationship exists between comorbid psychiatric symptoms and headache self-efficacy. This relationship, though significant, was not as strong as expected, with psychiatric symptoms only accounting for a small amount of variance in HSE scores. This small effect size likely indicates that numerous variables influence self-efficacy, and that psychiatric symptoms represent only one influential factor of many. Further study of other headache and psychological variables and self-efficacy, as well as the benefits of targeting self-efficacy in treatment, would be beneficial for further understanding self-efficacy and its role in headache.

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Tables

Table 1: Independent T-Tests

Gender Differences in Pertinent Variables

	Group Differences Among Gender	
	Mean	Standard Deviation
DASS-ANX		
Male	7.53	7.72
Female	7.45	7.36
DASS-DEP		
Male	8.12	8.77
Female	7.44	8.37
PCL		
Male	33.58	13.59
Female	35.65	14.65
HMSE		
Male	108.67*	18.91
Female	104.66	21.28

HMSE - Management Self-Efficacy scale, HIT6 – Headache Impact Test, PCL – Post Traumatic Stress Check List, DASS-ANX – Depression Anxiety Stress Anxiety Subscale, DASS-DEP – Depression Anxiety Stress Depression Subscale

* $p < .05$ vs females

Table 2: ANOVA results: Mean differences on variables of interest

	Migraine			***Omnibus p***
	Chronic migraine n = 155	**Episodic migraine n = 484**	**Episodic Migraine w/ aura n = 213**	
HMSE	97.14 (21.4)	108.05 (19.8)	105.66 (21.9)	< 0.001
HIT6	61.49 (6.6)	55.84 (7.8)	59.68 (7.5)	< 0.001
PCL	37.05 (15.5)	33.57 (13.9)	37.71 (14.6)	< 0.001
DASS-ANX	9.07 (8.7)	6.40 (6.4)	8.69 (8.2)	< 0.001
DASS-DEP	9.55 (9.8)	6.47 (7.6)	8.60 (8.9)	< 0.001
DASS-STR	14.39 (9.0)	10.56 (7.7)	13.87 (9.1)	< 0.001
HA-Days Per Month	18.29 (4.1)	5.87 (3.8)	6.43 (4.0)	< 0.001

HMSE - Management Self-Efficacy scale, HIT6 – Headache Impact Test, PCL – Post Traumatic Stress Check List, DASS-ANX – Depression Anxiety Stress Anxiety Subscale, DASS-DEP – Depression Anxiety Stress Depression Subscale, DASS-STR – Depression Anxiety Stress Subscale, HA-Days Per month – Headache days per month (frequency)

Table 3: Regression Analyses

Block (Step) Predictor	B	95% CI for B	P-Value	ΔR^2 of Block	Total R^2
Headache Self-Efficacy (without covariates)					
1. Anxiety	-.30	-.55, -.04	.02		
Depression	-.03	-.26, -.04	.78	**0.05**	--
PTSD	-.20	-.32, -.08	.001		
Headache Self-Efficacy (with covariates)					
1. Sex	.12	-3.44, 3.67	.95		
Frequency	-.60	-.84, -.35	<. 001	**0.14**	
Disability	-.74	-.93, -.55	<. 001		
2. Anxiety	-.19	-.43, .05	.12		
Depression	.07	-.15, .29	.55	**.015**	.15
PTSD	-.14	-.25, -.03	.01		

Bolded ΔR^2 values indicate statistically significant proportion of unique variance accounted for by Block

Appendix A

Structured Diagnostic Interview for Headache-Revised

Structured Diagnostic Interview for Headache – Revised (Brief Version)

Patient Name: Age: Sex: M F
Patient ID: Interviewer: Date: / /

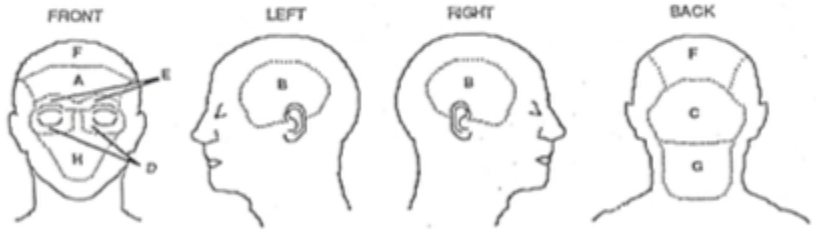
The following items are selected from the long version of the Structured Diagnostic Interview for Headache (SDIH). The SDIH is part of the Headache Evaluation and Diagnostic System (HEDS) which includes software for data entry and diagnostic decision-making. These materials are intended to facilitate diagnosis of selected recurrent, benign headaches according to both IHS (2004) and Ad Hoc Committee (1962) diagnostic criteria. Optimal use of this interview requires expertise with the diagnostic classifications and familiarity with the computer software and manual that accompany the interview.

1. Does the patient get more than one type of headache? Yes No
   (Complete a separate brief interview form for each type of headache) Headache #1 #2 #3

2. Select all pain locations that apply to this type of headache: ***(You must check at least one)***

   frontal (A) temporal (B) occipital (C) orbital (D) supraorbital (E)

3. Select all that apply: top of head (F) base of neck (G) nasal/facial (H)



4. What is the intensity of pain that the patient experiences with a typical headache? ____ ***(Indicate rating from 0-10)***

0 1 2 3 4 5 6 7 8 9 10

No Pain — Slightly Painful — Mildly Painful — Painful — Very Painful — Extremely Painful

5. Which of the following symptoms are a "predominant feature" of this headache type (presume that the headache is untreated)?

   Pain Location ***(Select only one)***: Unilateral Not Unilateral

   Pain Features ***(Select only one)***: Pulsating Pressing/Tightening (non-pulsating) Other

6. How often does the patient experience this type of headache pain? ____ d w m y
   (Indicate frequency in x per day, week, month, or year)

7. How long have these headaches been occurring at this rate? ____ months years
8. What is the total number of this type of headache ever experienced: 1 2-4 5-9 ≥10 ____
   (Indicate total number experienced)

9. How long does this headache last if untreated or unsuccessfully treated? (If patient falls asleep and wakes up without headache, duration of attack is until waking up. Check unremitting if patient reports never experiencing headache less than 7 days in duration). ***(Indicate duration in minutes, hours, or days)***

   Unremitting **OR**

   ____ m h d Typical Average ____ m h d Typical Minimum ____ m h d Typical Maximum

10. Has anything about this headache (except freq.) changed in the last 6 months? Yes No
    If **YES**, explain: _____
    _____

11. Is the patient's typical headache pain aggravated by routine physical activities (i.e., walking, lifting, bending, etc.)?
    Yes No

12. Do any of the following symptoms occur with this headache?

    Loss of appetite/Anorexia
    Headache worsened by conversational noise levels (phonophobia)
    Headache worsened by normal light (photophobia)
    Nausea ***(Indicate intensity)*** Mild Moderate Severe
    Vomiting ***(Indicate intensity)*** Mild Moderate Severe

13. Does the patient ever experience symptoms before this headache pain begins? Yes No
    If **YES**, and if any of the reported symptoms provide evidence of focal cerebral cortical, and/or brainstem dysfunction, complete **Appendix 1**
    If **NO**, skip to #14

14. Does this headache have severe unilateral orbital, supraorbital, and/or temporal pain, and/or does the interviewer suspect a cluster-type headache? Yes No
    If **YES**, complete **Appendix 2**
    If **NO**, skip to #15

15. Does the patient use any medications to relieve headache pain? Yes No
    If **YES**, complete #15a, #15b, #15c
    If **NO**, skip to #16

    15a. How long has the patient been using the medication(s) to relieve headache pain? ____ d w m y
    (Indicate duration in days, weeks, months, or years)

    15b. What is the frequency of medication use? ____days per week ____days per month ____ times per day

    15c. Did this headache develop or markedly worsen during medication overuse? Yes No
    If **YES**, complete **Appendix 3**
    If **NO**, skip to #16

16. Is this headache related to any head injury or trauma? Yes No
    If **YES**, complete **Appendix 4**
    If **NO**, skip to #17

17. Is this headache suspected to be attributed to a physical or other neurological disorder? Yes No

APPENDIX 1 — Migraine Aura Symptoms

1. How many aura attacks has the patient experienced? ____

2. What best describes the aura symptoms? ***(Select all that apply)***
   - At least one aura symptom develops gradually over more than 4 minutes, **AND/OR** 2 or more symptoms occur in succession over 4 minutes
   - Each aura symptom lasts longer than 4 minutes but less than 60 minutes
   - Headache begins during aura **OR** follows aura with a headache-free interval of less than 60 minutes

3. Indicate which of the following aura symptoms are present during this type of headache: ***(Select all that apply)***

X	SYMPTOM	X	SYMPTOM
	Partial loss of sight (scotoma)		Uncoordinated movements (ataxia)
	Scintillation		Dizziness (vertigo)
	Blurred vision		Ringing in ears (tinnitus)
	Fortification spectra (zig-zag lines)		Decreased hearing acuity
	Double vision		Decreased level of consciousness
	Tingling or numbness (paresthesias)		Aphasia or unclassifiable speech
	Weakness (paresis)		Poorly articulated speech (dysarthria)
	Other:		Other:

APPENDIX 2 — Cluster Headache Symptoms

1. Have the headaches occurred in cluster periods? Yes No
   - If **YES**, complete #1a
   - If **NO**, skip to #2

   1a. What is the total number of cluster periods experienced? ____

   1b. What is the duration of cluster periods? ____ d w m y ***(Indicate duration in days, weeks, months, or years)***

2. Are the headaches separated by remission periods? Yes No
   - If **YES**, complete #2a
   - If **NO**, skip to #3

   2a. What is the duration of remission periods? ____ d w m y ***(Indicate duration in days, weeks, months, or years)***

3. Indicate which of the following symptoms are present, as well as side affected, during this type of headache: ***(Select all that apply)***

X	SYMPTOM	SIDE	X	SYMPTOM	SIDE
	Red eyes (conjunctival injection)	R L		Forehead and facial sweating	R L
	Tearing of the eyes (lacrimation)	R L		Pupillary constriction (miosis)	R L
	Nasal congestion	R L		Drooping eyelids (ptosis)	R L
	Runny nose (rhinorrhoea)	R L		Eyelid swelling (oedema)	R L
	Restlessness or agitation			Other:	

APPENDIX 3 — Medication-Overuse Headache Symptoms

1. Has the patient withdrawn from the overused medication? Yes No
   If **YES**, complete #1a and #1b
   If **NO**, skip to #2

   1a. Did headache resolve or revert to its previous pattern within 2 months after discontinuation of overused medication?
   Yes No

   1b. Has medication overuse ceased within the last 2 months, but headache has not resolved or reverted back to its previous pattern? Yes No

2. Has intake of ergotamine, triptan, opioid **OR** combination of ergotamine, triptan, opioid, or analgesic occurred on 2 or more days per week, for 10 or more days per month, for greater than 3 months ***(Must not have combination overuse of any single class alone)***? Yes No
   If **YES**, indicate drug(s): ergotamine triptan opioid analgesic _____

3. Has the patient's intake of analgesic occurred on 2 or more days per week, for 15 or more days per month, for greater than 3 months? Yes No
   If **YES**, indicate drug: _____

4. Has the patient's intake of combination analgesics occurred on 2 or more days per week, for 10 or more days per month, for greater than 3 months? Yes No

   If **YES**, indicate drugs:
   _____

5. Has the patient's intake of medication other than ergotamine, triptan, analgesic, or opioid occurred on a regular basis for greater than 3 months? Yes No
   If **YES**, indicate drug: _____

APPENDIX 4 — Post-Traumatic Headache Symptoms

1. Was there a loss of consciousness associated with head trauma? Yes No
   If **YES**, complete #1a
   If **NO**, skip to #2

   1a. What was the duration of unconsciousness? ____ m h d ***(Indicate duration in minutes, hours, or days)***

2. Is head injury attributed to whiplash? Yes No
   If **YES**, skip #5 through #8
   If **NO**, complete #3 through #8

3. Did headache develop within 7 days after head trauma (or after regaining consciousness)? Yes No

4. How long has the headache continued? ***(Select most representative category)***
   Resolves within 3 months after head trauma
   Persists for greater than 3 months after head trauma
   Persists but 3 months have not passed since head trauma

5. Did coma develop? Yes No
   If **YES**, indicate severity on Glasgow Coma Scale: GCS <13 *[moderate/severe]* GCS ≥13 *[mild]*

6. Did post-traumatic amnesia develop and continue for longer than 48 hours? Yes No

7. Did symptoms/signs develop diagnostic of a concussion? Yes No

8. Were abnormal neuroimaging results attained suggestive of a traumatic brain lesion? Yes No

Appendix B

Depression Anxiety Stress Scale-21

DASS21

Name: *Date:*

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1	I found it hard to wind down	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I found it difficult to work up the initiative to do things	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I experienced trembling (eg, in the hands)	0	1	2	3
8	I felt that I was using a lot of nervous energy	0	1	2	3
9	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting agitated	0	1	2	3
12	I found it difficult to relax	0	1	2	3
13	I felt down-hearted and blue	0	1	2	3
14	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
15	I felt I was close to panic	0	1	2	3
16	I was unable to become enthusiastic about anything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life was meaningless	0	1	2	3

Appendix C

Headache Impact Test

HIT-6™
(VERSION 1.1)

HEADACHE IMPACT TEST™

This questionnaire was designed to help you describe and communicate the way you feel and what you cannot do because of headaches.

To complete, please circle one answer for each question.

		Never	Rarely	Sometimes	Very Often	Always
1	When you have headaches, how often is the pain severe?					
2	How often do headaches limit your ability to do usual daily activities including household work, work, school, or social activities?					
3	When you have a headache, how often do you wish you could lie down?					
4	In the past 4 weeks, how often have you felt too tired to do work or daily activities because of your headaches?					
5	In the past 4 weeks, how often have you felt fed up or irritated because of your headaches?					
6	In the past 4 weeks, how often did headaches limit your ability to concentrate on work or daily activities?					

COLUMN 1 (6 points each) + COLUMN 2 (8 points each) + COLUMN 3 (10 points each) + COLUMN 4 (11 points each) + COLUMN 5 (13 points each)

To score, add points for answers in each column.

Please share your HIT-6 results with your doctor.

Total Score

Higher scores indicate greater impact on your life.

Score range is 36-78.

Appendix D

Headache Management Self-Efficacy Scale

HMSE-25

Strongly Disagree	Moderately Disagree	Slightly Disagree	Neither Agree or Disagree	Slightly Agree	Moderately Agree	Strongly Agree
1	2	3	4	5	6	7

1)	I can keep even a *bad* headache from disrupting my day by changing the way I respond to the pain.	1	2	3	4	5	6	7
2)	When I'm in some situations, nothing I do will prevent headaches.*	1	2	3	4	5	6	7
3)	I can reduce the intensity of a headache by relaxing.	1	2	3	4	5	6	7
4)	There are things I can do to reduce headache pain.	1	2	3	4	5	6	7
5)	I can prevent headaches by recognizing headache triggers.	1	2	3	4	5	6	7
6)	Once I have a headache there is nothing I can do to control it.*	1	2	3	4	5	6	7
7)	When I'm tense, I can prevent headaches by controlling the tension.	1	2	3	4	5	6	7
8)	Nothing I do reduces the pain of a headache.*	1	2	3	4	5	6	7
9)	If I do certain things every day, I can reduce the number of headaches I will have.	1	2	3	4	5	6	7
10)	If I can catch a headache before it begins I often can stop it.	1	2	3	4	5	6	7
11)	Nothing I do will keep a mild headache from turning into a bad headache.*	1	2	3	4	5	6	7
12)	I can prevent headaches by changing how I respond to stress.	1	2	3	4	5	6	7
13)	I can do things to control how much my headaches interfere with my life.	1	2	3	4	5	6	7
14)	I cannot control the tension that causes my headaches.*	1	2	3	4	5	6	7
15)	I can do things that will control how long a headache lasts.	1	2	3	4	5	6	7
16)	Nothing I do will keep a bad headache from disrupting my day.*	1	2	3	4	5	6	7
17)	When I'm not under a lot of stress I can prevent many headaches.	1	2	3	4	5	6	7
18)	When I sense a headache is coming, there is nothing I can do to stop it. *	1	2	3	4	5	6	7
19)	I can keep a *mild* headache from disrupting my day by changing the way I respond to the pain.	1	2	3	4	5	6	7
20)	If I am under a lot of stress there is nothing I can do to prevent headaches.*	1	2	3	4	5	6	7
21)	I can do things that make a headache seem not so bad.	1	2	3	4	5	6	7
22)	There are things I can do to prevent headaches.	1	2	3	4	5	6	7
23)	If I am upset there is nothing I can do to control the pain of a headache.*	1	2	3	4	5	6	7
24)	I can control the intensity of headache pain.	1	2	3	4	5	6	7
25)	I can do things to cope with my headaches.	1	2	3	4	5	6	7

Appendix E

Post Traumatic Tests Disorder Checklist

PTSD Checklist (PCL)

Patient Name: _____ **Date:** _____

If an event listed on the Life Events Checklist **happened to you** or you **witnessed it,** please complete the items below. If more than one event happened, please choose the one that is **most troublesome to you now.**

The event you experienced was _____ (EVENT) on _____ (DATE).

Instructions: Below is a list of problems and complaints that people sometimes have in response to stressful life experiences. Please read each one carefully, then **circle** one of the numbers to the right to indicate how much you have been **bothered** by the problem **in the past month.**

BOTHERED BY	NOT AT ALL	A LITTLE BIT	MODERATELY	QUITE A BIT	EXTREMELY
1. Repeated disturbing memories, thoughts, or images of the stressful experience?	1	2	3	4	5
2. Repeated, disturbing dreams of the stressful experience?	1	2	3	4	5
3. Suddenly acting or feeling as if the stressful experience were happening again (as if you were reliving it)?	1	2	3	4	5
4. Feeling very upset when something reminded you of the stressful experience?	1	2	3	4	5
5. Having physical reactions (e.g., heart pounding, trouble breathing, or sweating) when something reminded you of the stressful experience?	1	2	3	4	5
6. Avoiding thinking about or talking about the stressful experience or avoiding having feelings related to it?	1	2	3	4	5
7. Avoiding activities or situations because they remind you of the stressful experience?	1	2	3	4	5
8. Trouble remembering important parts of the stressful experience?	1	2	3	4	5
9. Loss of interest in activities that you used to enjoy?	1	2	3	4	5
10. Feeling distant or cut off from other people?	1	2	3	4	5
11. Feeling emotionally numb or being unable to have loving feelings for those close to you?	1	2	3	4	5
12. Feeling as if your future will somehow be cut short?	1	2	3	4	5
13. Trouble falling or staying asleep?	1	2	3	4	5
14. Feeling irritable or having angry outbursts?	1	2	3	4	5
15. Having difficulty concentrating?	1	2	3	4	5
16. Being "super alert" or watchful or on guard?	1	2	3	4	5
17. Feeling jumpy or easily startled?	1	2	3	4	5