Psychological mislabeling of chronic pain: lessons from migraine in the 20th century (unabridged preprint)

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Abstract

Migraine and many other medical disorders were viewed as psychological problems in disguise for much of the 20th century. Starting with Freud, psychoanalytically oriented practitioners described a complex of unconscious conflicts, desires, and personality traits they believed to be the primary cause of a wide range of medical conditions. With the advance of basic research and pharmacotherapy, the psychogenic model was gradually replaced by a biopsychobehavioral model. This model treats chronic pain as a biologically-based disorder that can be influenced by psychological factors and lifestyle. The present paper argues that for many chronic pain patients, psychological factors may not play a significant role.

keywords: chronic pain; headache; migraine; psychology; psychological; psychosomatic; psychoanalysis; cognitive-behavioral

Introduction

For the last 30 years, a biopsychobehavioral model has guided our understanding of chronic pain. This model can be understood theoretically as a unitary entity made up of three inseparable components. In practice, this model describes a biologically-based disorder that can be influenced by psychological and behavioral factors. A variant of the biopsychosocial model, the biopsychobehavioral model has the advantage of expressing the three systems most frequently targeted in the treatment of chronic pain. The present paper will focus on this model.

A very different perspective governed the theory and treatment of chronic pain prior to the 1970s. Rooted in psychoanalysis, that perspective conceptualized chronic pain as a psychological problem masquerading as physical illness. For proponents of this view, the cause and treatment of migraine, chronic pain and many other medical disorders lay in disturbed emotions rather than disturbed biology.

Chronic pain is hardly the first example of psychological mislabeling in medicine. The history of medicine is rife with organic disorders that were initially thought to have a psychological basis. The standard psychosomatic text of the 1940s presented chapters on no fewer than two dozen largely "psychogenic" conditions – including migraine, asthma, peptic ulcer, arthritis, hypertension, colitis, thyroid disorders, diabetes, menstrual problems, tuberculosis, and skin, eye and ear ailments. To be sure, anxiety, depression, stress, and the like can influence migraine and chronic pain as they can every medical condition. And psychological treatment may be indicated in some cases. On the whole, however, frontline workers no longer consider migraine to be

largely, basically, or underlyingly "psychological." This makes migraine an apt example for the study of psychological mislabeling. The concept of psychogenic migraine has faded from the research literature. Yet psychological misconceptions linger. The migraine personality, for example, has forfeited the interest of researchers (along with the arthritic personality, the respiratory personality, and the like) but the idea that perfectionism plays more of a role in migraine than in other medical disorders still makes an occasional appearance. The overemphasis on psychological causation may be attributable, in part, to the effectiveness of psychological treatment for migraine. CBT (variously considered a psychological, behavioral, psychobehavioral, or psychoeducational treatment) has proved effective in numerous migraine trials. CBT, however, does not posit a psychological etiology for migraine or other medical disorders. From a cognitive-behavioral perspective, everyday anxiety, depression or stress may play a role in a given case of migraine. Or they may not. Emotional issues may even rise to the level of a comorbid psychiatric disorder. For many migraine patients, however, behavior – medication overuse, avoidance of headache triggers – rather than psychology is the focus of cognitive-behavioral treatment.

In this paper I track the origin and demise of the psychogenic model and its replacement by a biopsychobehavioral approach. I argue that the poor track record of the psychogenic model across medicine cautions against the assumption that psychological factors play a significant role for most people with chronic pain. And for those patients who are affected by psychological, behavioral or lifestyle factors, I spell out the guiding premises of CBT in pain management.

**Origin of the psychogenic model**

The history of psychiatry stretches back millennia. Hippocrates and Galen pondered obsessive-compulsive behavior, depression, and the medical importance of the psyche. Fifth century Jerusalem could boast a hospital devoted to mental disorders. But it was Freud in the 1880s who produced the first comprehensive clinical theory of the emotions and their effect on the body. In the kaleidoscopic universe of half-hidden conflicts and desires pictured by Freud, a conversion process provided the music for the dance between mind and body. At the center of this process, Freud envisioned a kind of psychological gatekeeper. The job of this gatekeeper was to limit the entry of anxiety-producing thoughts into consciousness. One way to accomplish this was through a psychic transmutation whereby disturbing emotion-laden thoughts were permitted to enter consciousness but only in the guise of physical symptoms. This was conversion, a concept that was to prove as enduring as it was intriguing. Over the next century, the idea that psychological phenomena can metamorphose into physically referable signs and symptoms – Freud's "mysterious leap" (p. 5) – permeated both medicine and popular thought. And the concept of psychogenesis gained a foothold in medicine that continues to the present day.

Psychoanalysts working with headache patients fleshed out many of Freud's general concepts. A major theme they developed was the role played by repressed emotion in the origin of headaches. Repressed hostility directed at the mother attracted particular attention. Weiss and English described a 35-year-old man with weekly migraines who deeply resented the financial support he felt obliged to contribute to his mother following the death of his father. Another of their migraine patients roiled with unconscious anger at the cruelty he experienced in a so-called religious household. The other major theme in the psychoanalytic headache literature revolved
around early emotional trauma. One author detected a headache-initiating trauma in infancy. Sadger speculated that while being carried to bed as a baby, her patient experienced the weight and pressure of her father's body as sexual arousal and that this conflict-laden unconscious response became the basis for her later migraines (summarized in English in Alexander).

Psychoanalytic writers also investigated the personality characteristics of migraine sufferers. Did a common set of personality traits define this disorder? Knopf concluded that women with chronic migraine were domineering, resentful, and humorless due to conflicted gender roles, overweening ambition, and emotional hypersensitivity. Women in another study were seen as tense worriers who tired easily, slept poorly, carried too much responsibility, and were, because of their perfectionism, readily unsettled by anything out of the ordinary.

One way to gauge the authority of the psychogenic model over time is through the writings of Harold Wolff. Wolff was the dean of American headache research in the middle of the last century. The eight editions of the textbook which still bears his name provide a window into leading-edge thinking on headache over the last 60 years. In the first three editions of the text (1948-1972), Wolff and his successors endorsed a moderate version of psychogenesis. They cited the occurrence of early childhood rejection, residual anger toward parents, and an endless striving for approval through achievement in (mostly female) headache sufferers who were driven, fastidious, and perfectionistic. Wolff cautioned, however, that these features were not pathognomonic of migraine. The next three editions (1980-1993) continued to find a significant role for personality in migraine, but only in select patients. In 2001 everything changed. The seventh edition of Wolff's text marked a turning point in the psychological understanding of migraine. Here the authors presented a fully mature biopsychobehavioral conception of migraine that had been chipping away at the psychogenic model for 30 years.

Origin of the biopsychobehavioral model

Behaviorism began to challenge psychoanalysis in the 1930s and 1940s. Grounded in the laboratories of Pavlov, Watson and Skinner, behaviorism installed conditioning as the prime mover of psychological development in place of Freud's innate drives and unconscious motivations. The clinical extensions of behaviorism were behavior modification, which psychologists used for childhood habit disorders such as bedwetting, and behavior therapy, which focused on adults.

The advent of two new treatments – biofeedback and cognitive-behavioral therapy – turned the attention of behavior therapists to headache. Psychologists initially used biofeedback as a laboratory tool for studying the conditionability of physiological responses that were not normally under voluntary control. During a blood flow modification study in the late 1960s, one of the participants developed a migraine. The attack ended at precisely the moment her hand temperature rose 10°F due to an increase in blood flow. Soon "handwarming" became a watchword in the treatment of migraine. The development of cognitive therapy and its eventual melding with behavior therapy established a more expansive platform for treating headache. The goal of treatment would now shift from blood flow modification and relaxation to nothing less than a comprehensive change in lifestyle. The new treatment rested squarely on the proposition that how and what we think largely determine what we feel and do.
Dysfunctional thoughts are the constants of cognitive-behavioral therapy. Therapist-guided self-questioning and behavioral experimentation are its levers of change. Behavioral assignments abound. Patients may be asked to practice daily goal-setting or to schedule specific activities. It is important to note that in a medical context this therapy may address fear, guilt, anger and other emotional issues. However, in contrast to psychoanalysis, the cognitive-behavioral model makes no etiological claims for psychological factors. Emotional difficulties and dysfunctional beliefs are seen as the consequences – and sometimes exacerbators – but not the cause of physical illness. Moreover, there is no expectation that even a complete amelioration of emotional distress will eliminate physical symptoms. While symptomatic relief is certainly welcomed, improved function remains the primary goal of treatment. Viewed through a cognitive-behavioral lens, the main components of migraine are headache triggers, exacerbating factors such as drug misuse, and the emotional difficulties that can result from chronic pain. Thoughts and behaviors are given equal weight. The medication-overuse skeptic, for example, might be taught how to challenge the belief that simple aspirin could not possibly transform episodic migraine into chronic daily headache, while at the same time committing to a firm tapering schedule.

A comparison of a psychoanalytic study from the 1930s with a contemporary cognitive-behavioral report brings the differences between the psychogenic and the biopsychobehavioral views of migraine into sharp relief. Weber's early psychoanalytic study describes a patient whose rejecting parents, wounded pride and pervasive narcissism produced "strategic" headaches that compelled compassion from her mother in childhood and her husband later in life. This emphasis on core personality and symptom as strategy typified the psychogenic position in medicine through much of the 20th century. In sharp contrast, Martin's prototypical cognitive-behavioral treatment focuses almost exclusively on the thoughts and behavior of his migraine patient. She tracks daily thoughts and headache triggers, practices muscle relaxation and pain-attenuating imagery, and learns to identify, challenge, and eventually alter unrealistic thinking about her children, her ability to cope with severe headaches, and her guilt over being chronically ill.

Retreat of the psychogenic model

The psychobehavioral approach described by Martin gradually supplanted the psychogenic model throughout medicine in the later decades of the last century. Advances in biomedical research played a large part in this shift. As new mechanisms and treatments came to light, the psychogenic model receded in condition after condition. The reconceptualization of asthma and peptic ulcer are especially instructive examples of the decline of psychogenic thinking.

In the 1940s, the psychodynamics of asthma were well-known. Most analysts placed the onset of the disorder in childhood. Fearful that mother would discover forbidden sexual or aggressive impulses (toward a sibling, in particular), the child developed a lifelong preoccupation with abandonment. The asthma attack at whatever age became a symbolic expression of an infant's rage on being separated from its mother. In essence, the asthma sufferer was reduced to a "shrieking, helplessly sprawling newborn child with blood-red, swollen face" (p. 245). Based on the observation that the confession of wrongdoing to a parent can relieve a child's guilt,
treatment for many asthma patients in the middle of the last century focused on the expression of anxiety-related desires to a therapist acting as an accepting mother substitute.

During the same period on a parallel track, medical researchers advanced the biology of asthma. They recognized the condition as a probable hereditary disorder marked by bronchial and airway hyperresponsiveness and inflammation as early as the 1940s. Advances followed in neurotrophin research, genetics, and the use of immunological technologies. Today asthma is understood as a chronic inflammatory disorder caused by a combination of genetic and environmental factors. The fall of the psychogenic model was even more dramatic in the case of peptic ulcer. Over many decades the notion that an unconscious dependency conflict caused autonomically induced changes in the stomach became etiologic dogma in the ulcer literature. Peptic ulcer became the paradigm of a psychologically-based physical disorder. With the discovery of *H. pylori* in the 1980s, the psychogenic model swiftly collapsed.

**Clinical considerations**

The decline of the psychogenic model across medicine serves as a cautionary tale. It reminds us that an insight can have a half-life and an inflection point. The idea that a psychological cause and cure could be found for most physical illness offered great promise at the beginning of the last century. Within a few decades, however, the arrival of modern experimental methods for identifying disease mechanisms and therapies prompted a subtle but significant reworking of the concept of psychogenesis. The result has been a biopsychobehavioral model in which physical disorders have a biological basis. At the same time, this model recognizes that psychological, behavioral, and lifestyle factors may influence a given physical disorder in a given patient at a particular point in time. This turn from psychogenic to biopsychobehavioral thrust CBT front and center into pain management. From that position a number of practice points come into view.

1. **Patient education**

The practitioner making a referral for pain management training should provide patient education focused on the etiological orientation, goals, and procedures of CBT. This will help ensure a common understanding between patient and practitioner of the differences between CBT and traditional psychotherapy.

2. **Etiology**

Although typically carried out by a psychologist, CBT does not assume that psychological factors play a significant role for all people with chronic pain. Nor does CBT view a nonresponse to medication or physical therapy as evidence of psychological involvement. CBT focuses first and foremost on thinking and behavior. Emotional issues may draw only passing attention, depending upon individual circumstances. Such clarification can strengthen the therapeutic alliance with patients who may have been told directly or by innuendo that their headaches had an emotional origin.

3. **Goals**
The primary goal of CBT is to help patients gain a sense of control over their pain. The emphasis is on improved functioning rather than pain reduction. However, as patients develop the ability to pace activities, increase productivity, and minimize dysfunctional thinking, they often experience improvements in both mood and pain tolerance.

4. Procedures

CBT typically employs a variety of research-based procedures. Training in relaxation, coping skills, activity pacing, and cognitive restructuring are the mainstays. These procedures are sometimes supplemented with biofeedback.

5. Candidates

CBT should be considered for people who report or manifest consequential levels of depression, anxiety, or stress. For these patients, CBT can mean the difference between a life marred by pain and a life crushed by pain. Patients wishing to learn pain management skills to supplement or possibly replace drug therapy are also good candidates.

Conclusion

The psychosomatic handbook of record no longer endorses a psychological etiology for migraine, asthma, ulcers or any of the "psychogenic" conditions in its 1940s counterpart. The concept of conversion continues but mostly across a highly circumscribed set of disorders. These are relatively uncommon and sometimes dramatic neurological disturbances – paralyses, seizures, amnesia, blindness – often marked by traumatic onset, short duration, and abrupt disappearance. Bringing a handbook up-to-date, whether yesterday's or today's, is a never-ending process. The coming decades will surely see new therapies and new insights into both specific disorders and pain in general. Will the next breakthrough in chronic pain come from genomics? Will a ghrelin agonist unlock the pathophysiology of functional gastrointestinal disorders? Such advances would certainly reduce the opportunities for psychological diagnosis by default. But will the coming decades also produce a deeper understanding of the interaction of mind and body in disease? The move from a psychogenic to a biopsychobehavioral perspective has not necessarily moved us closer to the ultimate nature of psyche and soma. A truly unitary biopsychobehavioral or biopsychosocial model, in which each of the elements loses its separate identity, may eventually prevail. But for now, at least, we have a model that gives psychological factors their due. No more, no less.

References


