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KEY NOTE LECTURE

F. Andrasik • H. Flor • D.C. Turk

An expanded view of psychological aspects in head pain: the biopsychosocial model

Abstract Traditionally, headache has been viewed from a limited perspective, both medically and psychologically. The authors propose that a more expanded view of headache that considers each perspective as important, as embodied in the biopsychosocial model, will greatly enhance understanding and be more useful in treatment planning. This model views pain as emanating from a complex interaction of biological, psychological and social variables. This paper describes the key behavioural, affective and cognitive influences and provides pertinent supporting examples from the literature.

Key words Biopsychosocial model • Headache • Atypical facial pain • Affect • Cognition

F. Andrasik (✉)
Institute for Human and Machine Cognition
University of West Florida
40 South Alcaniz Street
Pensacola, FL 32502, USA
e-mail: fandrasik@ihmc.us

H. Flor
Department of Clinical and Cognitive Neuroscience
University of Heidelberg
Central Institute of Mental Health
Mannheim, Germany

D.C. Turk
Department of Anesthesiology
University of Washington, Seattle, WA, USA

Introduction

The prevailing model to account for all forms of chronic pain, including headache, facial, and atypical facial pain, is best termed as the "biomedical model," and it is characterised as viewing pain as a direct transmission of impulses from the periphery to structures within the central nervous system [1]. As concerns headache, this model has led to a number of important insights into pathophysiological mechanisms and development of pharmacological treatments directed at modifying aberrant aspects. At the same time, this model has a number of limitations and has difficulty explaining the following: (1) pain that continues in the absence of identifiable pathology, (2) pathology that exists in the absence of pain, (3) the markedly varied individual responses to identical treatments, (4) the failure of potent medications to provide consistent pain relief, and (5) the absence of a strong relationship between pain, impairment and disability [1]. Some dismiss such limitations, attributing them to inadequate technology and claiming that these issues will be resolved in due time. Yet, an alternative viewpoint is that varied psychological factors play an important role in the genesis, exacerbation and maintenance of recurrent pain conditions and a proper explication of these factors can aid in understanding and ultimately treatment.

Early psychological models of pain and headache were unidirectional, oversimplified (e.g., pain in the absence of identifiable pathology was judged either to be motivated for secondary gain or was believed to be maintained by reinforcement contingencies), and had minimal impact upon the field. This view, like the biomedical model, also perpetuated an artificial dichotomy, that pain was either somatogenic or psychogenic [1]. A model that is more fruitful and heuristic is that which has been labelled the "biopsychosocial" or "biobehavioural" model (in the latter case, behavioural subsumes psychological and social factors). This model views pain (and any chronic illness) as emanating from a complex interaction among biological, psychological and social variables. From this perspective

[1] the diversity in illness expression (including severity, duration and consequences to the individual) can be accounted for by the complex interrelationships among predispositional, biological and psychological characteristics (e.g., genetics, prior learning history), biological changes, psychological status, and the social and cultural contexts that shape the individual's perceptions and response to disease. This model stands in sharp contrast to the traditional biomedical perspective that conceptualises disease in terms of more narrowly defined neurophysiological dimensions. This alternative model differs in other key ways, as it is dynamic and recognises reciprocal multifactorial influences over time. The text to follow expands further upon this more integrative model and provides some illustrative examples that demonstrate the psychosocial influences. Other papers within this series address biological influences, so these are not addressed further here.

For purposes of discussion, the psychosocial aspects are divided into the categories of behavioural, affective and cognitive influences.

Behavioural

The behavioural realm may be further subdivided into three types of learning: nonassociative, associative and social. Important processes to consider for nonassociative learning, but ones that are typically ignored when considering development of chronic pain, are habituation, defined as a decrease in the intensity of a response when the same stimulus is repeatedly presented, and sensitisation, defined as an increase in the intensity of a response when the same stimulus is repeatedly presented [2]. Patients who have continuous pain show sensitisation when exposed to painful stimuli, while nonpain patients (and patients with pain that is episodic) reveal habituation [3].

Associative learning consists of operant and respondent conditioning, with the former being studied most intently. Fordyce [4] was the first to comment on the role of operant learning regarding pain patients, pointing out how acute or episodic pain could develop into a more chronic form with attention from significant others including health care providers. He also noted how medication consumption, inactivity or avoidance of undesirable activities could be negatively reinforced (by terminating unpleasant states) and how well behaviours could concurrently decrease due to a relative lack of reinforcement. Research with chronic pain patients supports these basic tenets. For example, a number of studies have shown that pain patients exhibit more pain behaviour and report increased pain in the presence of solicitous spouses *vs*. the presence of more neutral parties and *vs*. controls [2]. Gentry and Bernal [5] have similarly pointed out how respondent conditioning can influence pain. Their model begins with the observation that acute pain (the unconditioned stimulus) is associated with sympathetic activation and increased muscle tension (the unconditioned response). With repeated pairings, stimuli that were previously neutral (environmental characteristics, body position, termed the conditioned stimuli) now come to elicit fear of pain and the sympathetic activation and the increased muscle tension mentioned before (now the conditioned response). Once established, the pain-tension cycle and anticipatory fear of pain may continue even in the absence of the original tissue damage [see also 6].

Finally, many responses are learned by observing and imitating the behaviour of others, and this is true for the expression and localisation of pain and ways of coping with pain [7, 8]. As a few examples, patients with solicitous spouses provide higher ratings of pain [9], reveal poorer performance on treadmills [10], and have lower pain thresholds and pain tolerance [11] when they believe they are being observed *vs*. not being observed. In a related fashion, pain patients are more likely to respond to marital conflict situations with increased pain behaviours [12]. Intervening in this realm can be complicated because these behaviours can serve adaptive functions [11]. Finally, even facial expressions can alter reports of pain [13].

Affective

The International Association for the Study of Pain [14] defined pain thus: "It is unquestionably a sensation in a part or parts of the body, but it is also always unpleasant and therefore also an emotional experience." There is no doubt that pain and negative affectivity (anxiety, depression and anger) go hand-in-hand [15–17]. Knowledge of this leads to a simple question, "how does affect interact with pain?" Although it is asked simply, the answer is not nearly so easily provided. Fernandez [17] points out there are six primary models to consider in addressing this question: affect as a (1) correlate of pain, (2) predisposing factor, (3) precipitating factor, (4) exacerbating factor, (5) consequence and (6) maintaining factor. Which one (or ones) best applies to a given patient has a direct bearing on the treatment that might be selected (as outlined in Table 1).

Fernandez [17] discusses methodological aspects that warrant consideration in future investigations and consideration of which might provide further important insights. These concern study of expanded affective types or newly recognised diagnostic entities, a greater focus on individual symptoms, consideration of complications due to Berkson's Paradox [18] (the spurious association that is observed because persons with two or more disorders are more likely to enter treatment than an individual with one disorder), greater attention to potential biasing factors (co-occurrence as an artefact of overlap in diagnostic criteria or measures, point of diagnosis *vs*. point of onset, etc.), and more careful attention to sample source.

Table 1 Treatment suggestions based on operative affect model (adapted from [17])

Affect as	Treatment approach
Predisposer	Characteriological change
Precipitant	Short-term intervention for affective trigger itself
Exacerbator	Focused intervention to defuse the aggravator
Perpetuator	Alter environmental conditions maintaining the pain
Consequence	Provide minimal attention to affect
Correlate	Third superordinate factor may be responsible; look elsewhere
Reciprocal relationship	Target both

Cognitive

Melzack's Neuromatrix Theory of Pain [19] proposes that a variety of cognitive factors can play a significant role in the experience of pain. Among these are attention, coping styles, beliefs, expectations, and memories about pain [20]. There is a growing literature suggesting that individuals who experience recurrent pain have an attentional bias that leads them to be over-responsive to pain-related stimuli [21]. Pain interrupts and demands attention, because it is an alarm signal for action [22], and this may be especially so for people with a high level of fearfulness about pain [23]. Studies employing high-resolution functional magnetic resonance imaging have shown that focusing attention on pain leads to activation in the periaqueductal grey region, whereas distraction leads to decreased activation in several areas involved with pain regulation (thalamus, insula and parts of the anterior cingulate) [24, 25].

The way people cope with pain has a bearing on the experience of pain. Consider for example the negative approach of catastrophising, which involves rumination about an aversive or negative event that is accompanied by a magnification of the extent of the problem and an attendant feeling of helplessness. Catastrophising is widely linked to chronic pain, particularly the subjective experience of pain [26] and this has been found in headache patients [27]. Headache patients tend to use other maladaptive strategies for coping (withdrawal, avoidance, self-criticism) as well [26, 28]. Another strategy, acceptance which is characterised by a state of remaining in touch with one's feelings and thoughts without making an active effort to change or attend to them, has been shown to be related to lower levels of pain, avoidance, depression and disability in patients who suffer from chronic pain [29–32].

Beliefs, appraisals and expectancies held by patients regarding possible consequences of pain and their abilities to deal with them can impact functioning both directly (by influencing mood, which can in turn affect muscle tension and biochemical processes) and indirectly (by altering attempts to cope) [2]. Beliefs can even affect pain severity. Newton and Barbaree [32] sampled the thinking of headache patients during and following a headache episode, both before and after treatment. Compared to the untreated control group, the treated group revealed a reduction in negative appraisals ("the headache is getting worse"; "there is nothing I can do") and a corresponding increase in positive appraisals. The authors noted that patients who reported the greatest shift in positive appraisals obtained the greatest reductions in headache activity. Further, intense pain was associated with more negative appraisals of headache episodes.

A laboratory study provides evidence that expectation alone can produce head pain. In this investigation, Bayer et al. [33] led participants to believe they would receive an undetectable current, which was "safe but often painful", applied to the forehead to measure the effect upon reaction time. Over one-half of the participants reported experiencing a headache and the reported pain level was in direct proportion to the displayed intensity setting.

A related concept is self-efficacy, or beliefs regarding a person's ability to engage in action that can influence the outcome of a headache episode. Headache self-efficacy beliefs (as assessed by the Headache Specific Self-Efficacy Scale [34]) have been shown to be related to positive coping responses, active efforts to manage and prevent pain, and increased pain tolerance. Self-efficacy can also mitigate the relationship between stress and headache; as self-efficacy increases, the correlation between headache and stress decreases [35]. Bandura and colleagues [36] have demonstrated that self-efficacy beliefs can activate opioid-mediated pain inhibitory mechanisms and increase pain tolerance.

Finally, patients are more likely to remember pain when they are in a pain state [37, 38] and they focus on pain when asked to report life experiences in autobiographical memory tasks [39]. These memory processes may lead to a selective focus on stimuli that have been associated with pain in the past, which in turn can lead to avoidant behaviour. Further, activation of painful or stressful memories can instigate physiological arousal, along with hypervigilance [2]. Thus, merely thinking about pain [40], discussing painful experiences [41] or observing others performing activities that

patients themselves are fearful of [42] can lead to increases in muscle tension, heart rate and skin conductance.

Thus, all forms of recurrent pain, including typical and atypical facial pain, are influenced by a number of interacting factors, biological and psychosocial. The most complete understanding will be obtained by incorporating a biopsychosocial viewpoint.

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