Temporomandibular Disorders: A Critical Review of the Nature of Pain and Its Assessment

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Temporomandibular disorders is a common form of chronic pain affecting the head, face, and jaw. The distinguishing symptoms of this disorder include pain and impairment of the masticatory function, and frequent display of symptoms, ranging from aches in the head, neck, ears, and eyes, to atypical toothaches, throat symptoms, and occlusal changes. It is recognized that pain is a complex, multifactorial experience including not only sensory dimensions, but also affective and cognitive factors. Recent recommendations regard temporomandibular disorders as a dual-axis disorder with physical and psychologic dimensions, but little research has incorporated measures of multidimensional pain characteristics in the assessment of temporomandibular disorders. This article is a review of the literature on the psychophysiologic factors contributing to temporomandibular disorders and its limitations. Recommendations for future research are also given.
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Temporomandibular disorders (TMD) is characterized by orofacial pain, tenderness of the muscles of mastication and the temporomandibular joint (TMJ), restricted range of mandibular motion, and several types of joint sounds.[1] The dysfunctional aspects of this disorder and their assessment have been studied extensively. However, the multidimensional nature of pain experience in TMD, as implied in pain theories such as the earlier "gate control" theory of pain[2] and the current multidimensional model of pain,[3,4] is poorly elucidated, understood, and utilized in the assessment and management of patients suffering from TMD.[5,6]

Pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage."[7] According to this definition, which is based on the gate control theory of pain, both the sensory and suffering components, including a variety of psychologic influences, are important for the overall manifestation of pain perception. It is now accepted that pain experience consists of several intrinsic dimensions, including sensory-discriminative, cognitive-evaluative, and motivational-affective dimensions. All these dimensions interact and affect pain response, ie, pain behavior, to varying degrees. Further modifying influences on all of these factors are exerted by extrinsic factors such as environmental and sociocultural factors. To understand a patient's individual pain experience, expression, and response, an appreciation and understanding of these multiple influences is important for optimal pain control.

In the present study, an overview of pain theories and the present knowledge of both the sensory-discriminative and psychologic factors of relevance to pain in general and in particular to TMD are reviewed. Psychologic factors reviewed include the cognitive-evaluative (meaning, beliefs, attributions), motivational-affective (emotions), and behavioral factors and learning principles (environmental and sociocultural influences). Special emphasis will be given to the assessment of these different psychologic dimensions as they relate to TMD.

Pain Theories

Several theories on the mechanism of pain perception, including the "specificity" theory, the "pattern" theories, and the gate control theory, have been proposed, and several reviews regarding these theories are available.[2,8,9]

The specificity theory[10] has been the traditional theory of pain. It was based on the concept that pain is a "specific sense," just like hearing, and is transmitted directly from the periphery to the pain center in the brain via specific pain receptors. Consequently, it also has been termed the *alarm-bell theory* and the *push-button theory*. Free nerve endings were viewed as the specific pain receptors. Melzack and Wall[2] rejected the specificity theory on the basis of its narrow psychologic assumption; ie, they argued that there was no support for a one-to-one relationship between pain and stimulus intensity. As an example of this, they presented evidence of wounded soldiers, who "entirely denied pain from their extensive wounds or had so little that they did not want any medication to relieve it,"[11] and Pavlov's dogs, who were conditioned to receive electric shocks, burns, and cuts during feeding and eventually responded to these stimuli as signals for food and failed to show signs of pain.[12,13] However, they stated that the assumption of physiologic specialization, ie, that skin receptors have specialized properties, was valid.

Melzack and Wall[2] also rejected other common theories of pain perception such as the pattern theories, which include Goldscheider's central summation theory,[14] Livingston's conceptual model of reverberatory circuits,[15] and Noordenbos' sensory interaction theory.[16] The central proposition of these theories was that stimulus intensity and central summation are the critical determinants of pain and that all fiber endings are alike—speculation that has since been proven incorrect. Because of the limitations of the earlier theories (for review, see Melzack[8]) and to understand pain modulation, Melzack and Wall[2] proposed the gate control theory of pain.

According to the gate control theory, the neural influences are subjected to "gating" in the dorsal horn of the spinal cord substantia gelatinosa (SG). This gating was argued to be dependent on the relative activity of large-diameter (A-beta) and small-diameter (A-delta, C) fibers, ie, the inhibitory effect of SG was increased by the activity in large fibers and decreased by the activity in small fibers. The spinal gating mechanism was explained to be influenced also by descending influences from the brain.

The gate control theory has since been subjected to further conceptual elaborations,[3] especially in relation to central control processes. Melzack and Casey[3] noted that T-cell output after modulation in the gate control system was considered to relay information to both the sensory-discriminative system (into the thalamus and somatosensory complex via neospinothalamic fibers) and the motivational-affective system (reticular formation and limbic system via paramedial ascending system). Central control processes (the evaluative processes, such as cognitions) also influenced and interacted at all of these levels. The final product (motor mechanism), ie, behavioral response patterns, was therefore not only a direct physical reaction to a stimulus, but also a summation of all of these influences.

Although the gate control theory has been criticized on the basis of the specific neurophysiologic mechanisms involved,[9,17,18] conceptually it is still widely accepted and provides the basis for an understanding of the complex, multidimensional aspects of pain experience and pain modulation in relation to sensory, cognitive, emotional, and behavioral factors. These will be discussed subsequently; first, as a neurophysiologic model, and second, as psychologic models.

An Overview of Sensory-Discriminative Factors (Neurophysiologic Model)

Pain manifesting in the musculoskeletal parts of the cranium and upper cervical region (primarily TMD) is mainly transmitted along the afferent fibers of the trigeminal nerve. It should be noted that other nerves are involved also in the innervation of the oral and masticatory region and therefore can effect the total sensory input. These include the facial (VII), glossopharyngeal (IX),

vagus (X), accessory (XI), and hypoglossal (XII) cranial nerves; first, second, and third cervical spinal nerves as well as visceral nerves, including sympathetic and parasympathetic afferents receiving interoceptive stimuli.[19] Furthermore, neuromuscular mechanisms involved in mandibular function and dysfunction can also affect the total neuronal input by interconnections and projections between sensory and motor nuclei.[20]

The main nociceptive transmission pathway for orofacial pain, however, involves the trigeminal cranial nerve (V) and its peripheral receptors, the trigeminal (gasserian) ganglion (with afferent cell bodies of the trigeminal nerve) and the brain stem (and the second-order neurones of the trigeminal brain stem sensory nuclear complex), with sensory relay to the third-order neurones in the thalamus and the cerebral cortex. Sensory information relay from brain stem to cerebral cortex may also involve multisynaptic pathways, eg, via the reticular formation, or pass to other brain stem structures such as the cranial nerve motor nuclei, the spinal cord, or other regions or subnuclei of the complex. This ascending transmission is subjected to descending modulation from higher centers (including cognitive, affective, and motivational factors) and to several inhibitory and facilitatory processes that are mediated by neurotransmitters, neuromodulators, chemical agents, and ion channels. Many of these modulatory processes are still poorly understood, and the exact role of different mediators is not clear.

Only a few studies exist concerning the properties and neuromuscular pathways involved in mandibular function and dysfunction.[21,22] These involve movement receptors in muscles and TMJs, the mesencephalic nucleus (the main proprioceptive nucleus) of cranial nerve V, and the motor nucleus of cranial nerve V, with relays back to the periphery (reflex mechanisms) and to higher centers (such as the motor cortex). Several interconnections and mediators seem to exist between sensory and motor pathways, and as already highlighted, these are subjected to further influences from higher centers.

The factors further influencing the specific nature of pain in the orofacial region involve the properties and proportion of myelinated versus unmyelinated fibers, the organization of the central pathways such as the trigeminal brain stem system, and the large bilateral representation of somatosensory cortex for orofacial pain.[23]

It is well documented that the brain stem sensory nuclear complex of cranial nerve V receives afferent inputs both peripherally (fifth nerve; cranial nerves; cervical nerves) and centrally (cerebral cortex; reticular formation). Although the important center for the processing of this information seems to be in the subnucleus caudalis, several interconnections within the sensory nuclei and between the sensory and motor nuclei also exist.[22,24]

A more detailed description of the pathways and factors involved in the transmission of pain from the orofacial region has been provided in reviews by Sessle.[20,22,25] The complex interactions have been discussed further by Lund et al.[24] Factors involved in mandibular function and dysfunction have been discussed by Dubner et al,[21] Ramfjord and Ash,[26] and Bell.[19]

An understanding of the differences between acute and chronic pain is of importance in comprehending the neurophysiologic and psychologic influences in both pain perception and control. The acute concept refers to pain as a "warning signal," ie, pain with usually a well-defined cause, rapid onset (phasic component), and a characteristic time course (tonic component) whereby the pain disappears after healing. With chronic pain, the tonic (longer-lasting) component may persist after healing.[27,28] The neuropathologic mechanisms related to prolonged or chronic pain (as seen in nerve injuries) produce changes in nerve chemistry and physiology central to the damage, reorganization of receptive fields (such as spread of pain), and changes in connectivity associated with loss of inhibition.[29–31] More recent literature indicates that chronic pain is associated with shifts in central neuroregulatory mechanisms that become self-sustaining long after peripheral nociceptive input (associated with acute tissue damage) has resolved.[32,33] A more recent overview of the neurophysiologic factors and mechanisms involved in the modulation of orofacial pain has been provided by Fricton and Dubner.[34]

Assessment of Pain

Several assessment methods have been used to objectively measure the complex perceptual experience of pain by the patient or subject. In general, the pain experience can be quantified only indirectly.

Chapman et al[35] reviewed and critically examined the practice and theoretical basis of pain measurement in animal research, laboratory investigations using human subjects, and clinical studies. In laboratory studies using human subjects, the quantification of pain has been by psychophysical methods (thresholds), rating scale methods (visual

analog scales), magnitude estimation procedures (number assignment or hand grip force), and by performance behavior of laboratory tasks. Pain experience has been measured by physiology correlates, such as electromyographic measures, autonomic studies, evoked potentials, and electroencephalograph methods. In clinical pain assessment, the most common methods employed have been behavioral measurements, subjective pain reports (such as visual analog scales of pain) and word descriptors (such as the McGill Pain Questionnaire), as well as pain inventories. The critical analysis of these methods in general pain assessment has been attempted by Chapman et al.[35]

Assessment of Pain in TMD

The measurement of pain in patients with TMD has been generally based on an objective assessment of pain elicited on palpation of the muscles of mastication or the TMJs. Several indexes[1,36–38] have been constructed to ascertain a severity value for the responses obtained. Of these, the Research Diagnostic Criteria for Temporomandibular Disorders[1] deserves particular mention because it attempts to provide a comprehensive assessment of TMD signs and symptoms while at the same time emphasizing the potential role of psychologic variables in mediating the pain response. Similarly, the TMJ scale,[38] which is a self-report symptom inventory, tests for the clinical significance of pain report and joint dysfunction and has been found to have predictive value in detecting psychologic problems in TMD patients.[39–43] The subjective pain experience has been measured by anamnestic (history) indexes, symptom severity indexes, or by visual analog scales. Regardless of increasing interest in the field, the subjective element of a multidimensional pain experience as emphasized in the multidimensional model of pain[3,4] and the gate control theory of pain[2] has not been systematically studied in patients with TMD. The assessment of these variables will be reviewed in the following section.

Psychologic Models

According to the gate control theory[2] and the multidimensional model of pain,[3,4] the understanding of pain perception includes not only the sensory component, but also a variety of psychologic influences. Several psychologic models have been proposed to explain these influences. These latter factors include cognitive-evaluative, motivational-affective, behavioral, and sociocultural dimensions.

Cognitive Factors

Definitions and Theories. As implied by the gate control theory of pain, cognitions (the evaluative components of pain) play an important part in pain perception and control. Cognitions have been defined as "a generic term embracing the quality of knowing, which includes perceiving, recognizing, conceiving, judging, sensing, reasoning, and imagining."[44] Other terms relevant to cognitive theory include stressors (such as pain), appraisals (perceived consequences), and coping (efforts to manage).[45] Appraisals have been defined as "a dynamic process that changes according to the person's perceived or anticipated consequence of an event, its importance to his well being, and the perceived resources he has available to cope with the threat"[46]; and coping has been defined as "a constantly changing cognitive and behavioral effort made to manage specific internal and/or external demands that tax or exceed the resources of the individual."[45]

Conceptually, the gate control theory of pain[2,3] is considered to be most relevant to understanding the cognitive aspects of pain (such as attention and distraction, past experience, beliefs, and other evaluative dimensions) and the interrelationships between cognitive-evaluative and sensory-discriminative, motivational-affective, and behavioral responses. Other cognitive theories include the theory of cognitive dissonance[47,48]; attribution theory[49]; attribution of control[50]; cognitive control[46]; self-efficacy[51]; and the theory of "stress, appraisal and coping."[45]

According to the theory of cognitive dissonance, behaviors or cognitions that are dissonant with each other will lead to a state of tension, which, in turn, will motivate the person to reduce dissonance. The attribution theory relates to causes or explanations people seek for the observed events in their lives. The concept of control has been implied to be especially important in pain conditions. Studies have shown that attribution of internal rather than external loci of control is an important factor in the treatment of patients with chronic lower back pain.[50] Individuals with an internal locus of control perceive cause-and-effect relationships between their own behavior and expected outcome; those with an external locus of control relate the outcome to be dependent on external sources, such as powerful others, chance factors, fate, and luck.[52] Clinical and experimental studies

have proposed interrelationships among lack of control, increased anxiety, and decreased tolerance to pain.[53,54] Bandura[51] stated that effective coping depends on the person's assessment of his/her competence, ie, perceived control, and this has been viewed as an important factor in the treatment of pain. After reviewing theories relevant to control, Thompson[55] proposed that the "reaction to a potential stressful event depends on the meaning it has to the individual"; ie, control can change the meaning of an aversive situation from unendurable to endurable. Furthermore, it has been shown that a belief in the origin of pain can affect the pain perception, tolerance, and emotional and behavioral response to pain.[56,57] According to Ellis[58] and Beck,[59] belief systems influence cognitive appraisal, and they explained emotion as a consequence of the cognitive appraisal. Ciccone and Grzesiak[60] proposed mistaken beliefs as a key factor in chronic pain. Further interaction between cognitive appraisals and affective reactions include catastrophizing or overdramatizing and convictions of helplessness.[61–63]

Coping has been viewed from several points of view. Approaches and categorizations such as passive-active,[64] problem focused–emotion focused,[45] adaptive-maladaptive,[65] and cognitive coping–behavioral coping[66] have been described. Specific importance in coping, as discussed above, has been placed on perceived control, ie, a person's assessment of his/her competence,[51] or "expectancy of efficacy."[9] Fernandez and Turk[67] reviewed some of the classification systems used for the assessment of the efficacy of coping strategies in altering pain perception. They proposed six major classes of cognitive coping strategies, ie, external focus of attention, neutral imagings, pleasant imagings, dramatized coping, rhythmic cognitive activity, and pain acknowledgment.

Apart from pain, cognitive approaches have been applied to coping with stress,[45] anxiety,[68] and depression,[59,69] and to treatment of a variety of psychologic problems.[9,27,70,71]

Some of the more important elements in the cognitive control of pain seem to be the beliefs (appraisals), the perceived control, and the specific coping strategies used. It is still not known, however, which cognitive ingredients are of importance in different pain conditions.

Cognitive Factors and TMD. Cognitive processes may be of importance in understanding many of the puzzles of TMD as outlined in the following.

First, pain/discomfort or dysfunction affecting the masticatory apparatus can vary. It can be acute or chronic, intermittent or constant. The majority of patients presenting for treatment have experienced pain for months, often for years. It is well documented that the prevalence of what are considered signs of disturbance affecting the stomatognathic system have been as high as 88% in the general population, but only about 5% to 26% in the population samples have sought or have been considered to be in need of treatment. Pain has been shown to be a major reason for patients to seek treatment. Other possible factors may be the presence or increase in life stressors or of anxiety or depression. Adverse life events have been shown to be associated with certain pain descriptions in lower back pain[72] and have been positively associated with pain ratings in osteoarthritis.[73] Stressful life events have been proposed to be prevalent in patients suffering from TMD.[74-76] Likewise, increased anxiety and tension has been proposed in the development of parafunctional habits and muscle tension; however, the relationship is not clear. Turner et al[77] have argued that the lack of data concerning the relationship between stressful life events and pain problems may be the result of a lack of research that has considered patient differences in appraising and coping with stressful events.

Second, recent research has shown that individuals who experience pain develop ways to cope in an attempt to tolerate, minimize, or reduce their pain,[70,78] and this coping may be of importance in the way patients adjust to pain.[79,80] Likewise, understanding the cognitive dimensions of pain may be of importance in understanding the heterogeneity of symptoms in patients with TMD. Patients suffering from TMD have been classified traditionally according to morphopathologic dimensions. An alternative classification of patient differences was presented by Rudy et al,[81] who found that patients could be distinguished and classified according to the psychologic dimension rather than structural pathology. The groups identified included dysfunctional, adaptive copers, and interpersonally distressed. At the present time, these dimensions in TMD are still poorly understood and are not fully utilized in therapy.

Third, it has been well documented that 70% to 90% of patients suffering from TMD can be successfully managed by conservative methods, the most common of which is interocclusal appliance therapy.[82] The exact mode of action of the interocclusal appliance in producing a relief of symptoms is not clearly understood. It is rather interesting to note that one of the theories assessing the possible reasons for the efficacy of such interocclusal appli-

ances is proposed to be "increased cognitive awareness."[83] About 40% of positive therapeutic outcome has been related to effective counseling[84] concerning the nature of the problem and to providing the patients with self-management skills (ie, increasing self-efficacy) such as rest, soft diet, and physical therapy. Little is known, however, about the methods that patients with TMD use to cope with their problem. The reasons for the variable nature of the resolution have been interpreted to be related to psychosocial problems, but the exact nature of these is not well documented. Whether other psychologic dimensions may be operating is also not documented.

Cognitive-behavior therapies, including biofeedback and relaxation training, have been proposed and used in the management of TMD, but no controlled studies concerning the efficacy of these treatments exist. Experimental and clinical studies in pain conditions affecting other parts of the body have shown that training in the use of cognitive strategies for pain control can increase pain threshold and tolerance,[85] reduce pain and distress with medical and surgical procedures,[86–88] decrease headache activity,[89–91] and reduce pain ratings in chronic pain patients.[92–94] Some pain management programs now incorporate behavioral methods (such as operant conditioning) and pain coping skills training (such as relaxation, imagery, goal setting, and distraction) to teach patients adaptive ways to deal with their pain.[70,71] Recent reports suggest that cognitive-behavioral interventions to improve ways of coping are especially useful with psychologically oriented individuals who may represent a specific TMD subgroup.[95]

Assessment of Cognitive Factors. There has been a major problem in measuring coping.[96] This has been largely a result of the inward nature of the coping experience, and the measurements in the past have relied largely on self-reports and self-monitoring procedures. Another inherent problem has been the lack of taxonomy of coping strategies.

Two questionnaires represent potentially valuable tools for studying coping processes in chronic pain problems: the Coping Strategies Questionnaire (CSQ)[66] and the Ways of Coping Checklist (WCCL).[97] The CSQ examines coping strategies and appraisals with respect to pain control. The extent to which six cognitive strategies and one behavioral strategy are used to cope with pain are assessed. The WCCL examines appraisals of and coping strategies for the stressor that the patient identifies as most significant. In addition, the Vanderbild Pain Management Inventory (VPMI)[64,98] has been developed to assess passive and active coping strategies.

The CSQ was developed by Rosenstiel and Keefe[66] who used it to assess coping in a group of 61 patients with lower back pain. They found several factors: *(1)* cognitive coping and suppression, *(2)* helplessness and diverting attention, and *(3)* praying. Each of the factors was related to specific measures of adjustment to chronic pain. This factor structure was later replicated by Turner and Clancy[99] in a different group of patients with lower back pain. The latter authors also found significant associations between the use of certain types of coping strategies and measures of physical and psychosocial impairment. Treatment-related changes in types of coping strategies used and pain intensity and disability were found also. Increased use of praying and hoping was significantly related to decreased pain intensity, and decreased catastrophizing was significantly related to lower pain intensity and physical and psychosocial impairment.

The CSQ has been used in several pain patient populations, including patients with lower back pain prior to undergoing surgery, patients with myofascial pain, and patients with osteoarthritis. Gross[100] found that CSQ factors were associated with the ratings of patients' back pain prior to and 6 weeks following surgery. Two factors, self-reliance and loss of control, were found to be predictive of the postsurgical adjustment. In another study, Keefe and Dolan[101] compared a group of patients suffering from myofascial pain (MPD) and lower back pain. They found differences in the use of diverting attention and praying as well as in the overall use of different coping and behavioral strategies, but the factor structure was not reevaluated in this study. Keefe et al[102] found that of a group of patients with osteoarthritis, those who reported they were able to control their pain and who did not endorse catastrophizing responses had lower pain levels, better health status, and lower levels of psychologic distress. The CSQ has also been studied in relation to loci of control in a group of patients with chronic pain.[52] Patients depending on external or environmental factors and powerful others for help in pain control (the external locus or chance locus of control) reported more passive coping strategies, such as helplessness, diverting attention, and praying and hoping. These patients also felt more depressed and had higher overall levels of psychologic distress. Recent applications of the CSQ attest to its reliability and validity in the assessment of pain coping strategies.[103,104]

The revised WCCL[97] has been used to study the relationship between appraisal, coping, and psychologic distress in a number of populations, and

certain types of coping responses (eg, wishful thinking) have been significantly associated with depression (Vitaliano et al cited in Turner et al[77]). Turner et al[77] used the WCCL to study stressors, appraisals, and coping responses in chronic lower back pain. Only 43% identified pain or physical limitations as their primary stressor. These 43% differed significantly from those who did not identify pain or physical limitation as their primary stressor in a number of variables, including average pain during the last week, coping strategies, and appraisals of the stressor. It was found also that certain appraisals of the pain problem were associated with certain coping responses; eg, blaming oneself was negatively related to ratings of average pain during the previous week, and seeking social support was negatively related to present pain ratings.

The Vanderbild Pain Management Inventory (VPMI)[64] is a 27-item self-report measure of cognitive and behavioral strategies; patients rate the frequency with which they use the strategies when their pain reaches a moderate or greater level of severity on a five-point scale. Two factors were identified in a group of 361 patients with rheumatoid arthritis: active and passive coping. Patients who used active coping (such as distraction from pain or an active effort to function despite pain) reported less pain, less depression, less functional impairment, and higher general self-efficacy. Patients scoring high on passive coping (dependency on others for help in pain control) tended to report greater depression, pain and functional impairment, and lower general self-efficacy.[64]

Chronic pain populations appear to share many characteristics, including coping strategies, but few controlled studies of coping in patients with TMD have been published.[105,106] There has been a recent interest in assessing the beliefs of patients with pain in association with their coping strategies, but few methodologically sound studies exist (see De Good and Shutty[105]).

Motivational-Affective Factors

Theories. The most frequent affective concomitants of pain include anxiety, fear, and depression, but they may also include anger, aggression, guilt, and subservience.[107]

There are several difficulties associated with the assessment of affective dimensions of pain. These include the subjective experience of pain and emotional processes, individual differences in pain experience and behavior, and variations in pain experiences in different diseases.[108,109] Furthermore, it is still not clear whether affective processes should be conceptualized as causes or consequences of pain.[110] Craig[107] proposed that pain and emotion should be conceptualized "as multidimensional processes with reciprocal dependence on each other."

The affective reaction seems to be related to the type of pain experienced.[107] Phasic (short-duration) pain has been shown to be associated with fear and stress followed by recuperation that motivates rest and healing. There is also evidence that the immediate reaction to injury is further modulated by biologic, physical, and social context. Evidence for this is shown by athletes engaged in competitions and soldiers on the battlefield, who sustain injuries without complaint. Acute pain (including both the phasic and tonic components) tends to provoke fear and anxiety, while chronic pain is likely to generate depression, withdrawal, irritability, and somatic preoccupation (for review, see Craig[107]).

The relationship between pain and depression is not clear, but nevertheless it is not uncommon to find concomitant depression in painful conditions.[111] Romano and Turner,[112] after reviewing estimates of the prevalence of affective disorders and various pain conditions, concluded that between 30% and 100% showed evidence of depression. However, the authors also highlighted several methodologic shortcomings in the assessments of both pain and depression, variations in the study populations, and absence of satisfactory assessment of base rates of depression in control populations. Although greater depression may be reported in pain patients, these reports may reflect a negative perceptual bias of these patients.[113] Further understanding of this relationship will also need an appreciation of both the variety of chronic pain disorders and the type of depression.[114]

The theoretical approaches to explain the relationship between emotion and pain include biologic, psychodynamic, cognitive, and behavioral models. Biologic theories focus on the disregulation of the key neurotransmitters that are thought to mediate neuroanatomic pathways in control of both pain and emotion.[115] According to the psychodynamic view, inability to modulate and express intense, unacceptable feelings such as anger[110] or feelings of guilt[116] may underlie this relationship. A cognitive view emphasizes thoughts of helplessness and lack of control,[117] while the behavioral view[118] emphasizes the role of severe reduction of activity in chronic pain. On the other hand, chronic pain may also be viewed as coping with an unsatisfactory existence.[107]

There is evidence of several interrelationships among affect, pain, and other psychologic dimensions. Patients with pathophysiologic conditions have been shown often to suffer psychosocial dysfunction.[119,120] Hence, psychologic processes cannot be ignored even if there is an organic basis for the disorder.[107] On the other hand, pain without the evidence of pathophysiologic disease may be evidence of somatization[121] or masked depression.[107] Psychogenic pains can also include hallucinations and conversion reactions.[122] Apart from anxiety and depression, some patients present with anger. These patients have been reported to show interpersonal alienation and manipulativeness.[123] Furthermore, the relationship between stress (environmental stressor), failure to cope, affective distress, and pain have been observed in several studies.[9,72,124] Also, the relationship among pain, anxiety, and tension has been suggested to underlie many musculoskeletal disorders, but the relationship is not clear. Further evidence for the association between emotional state and pain has been shown in studies that demonstrate positive emotional states and improved pain control.[125] Distressing events are also believed to underlie the initiation and exacerbation of many physical disorders previously referred to as psychosomatic or psychophysiologic.[107] Beutler et al[110] have indicated a possible role for the immune system in the relationship between stress and painful diseases. Evidence for the complex interactions between affect and pain are further highlighted in the neuromodulatory mechanisms, including serotoninergic and opioid pathways[126] and through cognitive factors (meaning, beliefs, loss of control[27]).

Motivational-Affective Factors and TMD. The review of personality and emotional factors in the etiology of TMD has shown diverse and conflicting results. The concept of a particular personality type involving several dimensions of the pain theory (cognitive, emotional-affective, pain behaviors) and the environment predisposing to a particular pain problem has not been supported in the published literature. This observation includes studies on patients with TMD and atypical facial pain, even when using similar methodologies. There is evidence, however, that subgroups of patients with facial pain may have personality characteristics, such as being a perfectionist or insecure, that make it difficult for them to deal with events of daily life.[127]

Many instruments have been used to study affective and personality dimensions in TMD, including the Minnesota Multiphasic Personality Inventory (MMPI and MMPI-2), Spielberger State-Trait Anxiety Scale, Beck and Hamilton depression scales, Symptom Checklist 90–Revised (SCL-90), and Cornell Medical Index (CMI), among others. The results so far have been varied (Table 1).

The earlier studies were empirical and descriptive in nature.[128–132] Moulton[133] reported that patients suffering from TMD were overly dependent emotionally, perfectionists, and "highly strung." Many also suffered from gastrointestinal disturbances, anxiety, and/or depression and had nervous habits, such as bruxing, to alleviate tension. Moulton[133] focused on the psychodynamic concept that the mouth is the first site for infantile satisfaction, as well as being the most primitive weapon of defense. Moulton[133] also emphasized that the mouth played an emotional role throughout life, contending that a gaping, helpless expression is a regression to a childlike state, whereas a rigid, tight-set mouth represents a controlling personality type. Kydd[134] used physiologic, emotional, and dental evaluations and found that only one of the 30 patients included in the study showed hyperfunction when emotional stress was absent. He interpreted the finding as evidence of an emotional response to stimuli the patients perceived as threatening in their daily lives. In 22 of the 30 patients, he found evidence of emotional conflict and anxiety. Lefer[135] reported that patients with TMD had lost an important adult early in life and inferred this as a separation anxiety. Unable to satisfy their dependency needs, the patients tended to brux to relieve frustration. He regarded this behavior as a symbolic reversion to the toothless state of infancy. Lupton[136] described another group of patients with TMD as responsible, generous, and managerial with a tendency to deny any submissive or dependent qualities. Burton[137] postulated that five psychologic phenomena could explain pain in TMD, ie, depressive equivalents, conversion reaction, hypochondriasis, psychophysiologic pain, and somatic delusion.

Other earlier studies have supported the presence of affective factors in patients with TMD. Lascelles[138] reported atypical depression in the majority of patients with facial pain. He further tested the effect of antidepressants in a double-blind trial and reported that those patients with active treatment improved significantly compared to those with placebo treatment. Fine[139] stated in his study that as many as 76% of a group of patients with TMD had psychiatric symptoms compared to 20% of controls. Small[140] grouped patients with TMD into "normal" and "abnormal" based on psychologic tests and implicated

Table 1 Psychologic Studies on Patients Suffering From TMD or Atypical Facial Pain (AFP)

	Subjects	Methods*	Findings
Engel, 1951[128]	20 AFP	Psychiatric interview	Hysterical conversion, masochistic, depressed, self-destructive, dependent, unhappy, unsatisfied
Moulton, 1955[133]	35 TMD	Psychiatric interview	20 of 35 anxious; 11 of 35 psychotic or prepsychotic; patients could be divided into two groups: *(1)* hostile, angry, dependent; *(2)* perfectionistic, obsessive, demanding, efficient
Lesse, 1956[129]	18 AFP	Psychiatric interview	Rigid, perfectionistic, domineering, obsessive-compulsive, masked depression
Kydd, 1959[134]	30 TMD	CMI, MMPI, EPP	20 of 33 emotionally disturbed, anxious, tense, apprehensive, overreacting to pain
McCall et al, 1961[130]	70 TMD 2 control	MMPI	Differences on 48 of 566 items: TMD characterized by somatic complaints, nervous anxiety, worry
Lascelles, 1966[138]	93 TMD	Physical examination, psychiatric interview, questionnaire	Majority had atypical depression
Lefer, 1966[135]	30 TMD	Psychiatric interview	Poor ego boundaries, utilized bodily reactions to reduce anxiety
Lupton, 1966[136]	37 TMD and control	MMPI IAC, TAT, interview	Overgenerous, autocratic, narcissistic, sadistic, dominant, hypernormal, responsible, managerial
Marbach et al, 1978[170]	170 TMD	Clinical interview, clinical and radiographic examination	Major life events preceded the treatment seeking in 62% of patients with an inconsistent sociomedical profile: sophisticated, individualistic, dominant, yet dependent, uninformed, and skeptical in health orientation
Millstein-Prentky and Olson, 1979[153]	135 TMD 41 successfully treated 33 unsuccessfully treated	MMPI, 29-item scale	Developed new scale, failed to predict treatment outcome in new patients, probably because of absence of consistent personality differences in patients
Schwartz et al, 1979[154]	84 TMD 42 successfully treated 42 unsuccessfully treated	MMPI	Similar configuration of MMPI profiles in successfully and unsuccessfully treated, but the latter group showed greater degree of emotional distress; profiles were diagnostic of a psychophysiologic disorder characterized by repression and somatization
Helöe et al, 1980[220]	113 TMD 46 control	Psychiatric interview, MMPI	No significant differences between groups in psychiatric disturbances; 24 of 113 patients had severely disturbed capacity for interpersonal contact
Marbach and Lund, 1981[171]	476 TMD 161 control	IPAT depression scale, physical anhedonia inventory, SAI	Few differences between patients and control subjects; clinical variables were correlated with depression but not anhedonia scores; psychologic variables did not distinguish between subgroups of facial pain subjects
Speculand et al, 1981[166]	3 TMD 11 AFP 10 facial neuralgia	IBQ	AFP higher on disease conviction, lower on psychologic versus somatic perception of illness
Fine, 1971[139]	50 TMD and control	Psychiatric interview	76% of TMD with psychotic symptoms; 20% of control subjects with psychotic symptoms
Solberg et al, 1972[169]	29 TMD and control	MMPI, clinical interview	Obsessive-compulsive, no common personality trait for TMD, anxiety within normal personality profile
Gessel, 1973[132]	23 TMD	Clinical interview	High standards, few indications of covert or overt depression, little social disability
Grieder, 1973[131]	100 TMD	Not specified	Internalized stress, conflicts between feelings of dependency and desire for dominance and aggression, denied and suppressed true emotions, perfectionistic, domineering, responsible, generous
Gross and Vacchiano, 1973[164]	56 TMD and control	CPFQ	TMD patients emotional, proper, imaginative, apprehensive, tense, anxious, neurotic, "drive the ego, restrain the id"
Molin et al, 1973[165]	27 TMD and control	MNTI, Eysenck somatic and psychic inventory, BDAI, CPFQ, SUSI	TMD patients higher in neurotism, psychic and somatic anxiety, muscular tension, aggression, super-ego strength; higher ratings on SUSI, emotionally unstable, insecure, hostile, worried

Table 1 (continued)

	Subjects	Methods*	Findings
Shipman, 1973[141]	176 TMD	MMPI	TMD patients high on coversion, hysteria, hypochondriasis, depression, psychopathic, deviate
Schwartz, 1974[142]	42 unsuccessfully treated	MMPI	Elevated on neurotic trial (hypochondriasis, depression, hysteria), excessive somatic concern, agitation, depression
Marbach et al, 1978[170]	87 TMD and control	Clinical and radiographic examination	No significant differences in state and trait anxiety
Stein et al, 1982[74]	16 TMD 8 control	SRSS	Patients scored higher on SRSS
Speculand et al, 1983[184]	100 TMD 100 control	IBQ	Patients had significantly increased levels of disease conviction, anxiety, or depression and were less likely to deny the existence of problems in their lives
Lipton and Marbach, 1984[208]	68 myofascial 61 organic TMJ 41 both 170 TMD	Subjective evaluation	Outcome of treatment related to sociocultural background, sociomedical orientation, symptom and treatment history, and behavior and attitudes toward pain
Feinmann and Harris, 1984[146]	50 TMD 43 AFP	CIS, MADRS, Eysenck personality questionnaire, life events	35% (33 of 93) had depressive neurosis, 22% (20 of 93) nondepressive neurosis; 82% adverse life events prior to the onset of pain; no difference between the two groups on these measures
Moss and Adams,1984[172]	10 TMD control	MMPI, SAI, BDI	No differences in personality, anxiety, or depression
Eversole et al, 1985[151]	156 TMD	MMPI	Subgroups (myofascial, internal derangement, AFP) differed in MMPI psychometric scales; myofascial and AFP significantly higher scores for hypochondria, depression, hysteria
Keefe and Dolan, 1986[101]	32 TMD 32 lower back pain	SCL-90 CSQ	Differences in coping strategies; both groups reported high levels of psychologic distress
Butterworth and Deardorff, 1987[149]	100 TMD	SCL-90	Three groups: normal, moderately distressed, and severely distressed
Gerschman et al, 1987[147]	368 chronic TMD or AFP	Eysenck personality inventory, Hamilton anxiety and depression scale, rapid symptom checklist	Half moderately anxious and/or depressed, about 17% severely anxious and/or depressed
Marbach et al, 1988[167]	151 TMD 139 control	Interview	TMD patients unsually distressed, beleaguered by physical illnesses and injuries as well as by pain, tend to attribute their fate to external factors, have fewer sources of emotional support
Bush et al, 1989[152]	85 TMD	Pain Dysfunction Questionnaire, VAS, emotion measures	MPD more psychologically distressed with higher ratings for anxiety and inability to endure pain
Lennon et al, 1990[79]	99 TMD 98 control	Psychologic interview	TMD patients more likely than control subjects to see occurrence of pain events outside of their control and as undesirable, and TMD patients used more problem solving and distraction than other negative events to cope with pain
Schnurr et al, 1990[221]	79 control with pain 71 pain free	SBPI, IBQ, Multidimensional Health Locus of Control, Perceived Stress Scale, WCC	No difference in personality type, response to illness, attitudes towards health care, or ways of coping with stress between TMD and control subjects
Stockstill and Callahan, 1991[168]	151 TMD 89 control	Measurements of hardiness (control, commitment, and challenge), SRRS, CES-D, Taylor manifest anxiety scale, seriousness of illness survey	Hardiness (control, commitment) significantly lower in TMD patients; challenge-factor of hardiness, anxiety, and depression did not differ between groups
Gerke et al, 1989[155]	43 TMD	Clinical assessment, IBQ	Grouping clinical and psychologic factors resulted in more than 80% successful prediction of treatment outcome

Table 1 (continued)

	Subjects	Methods*	Findings
McCreary et al, 1991[156]	112 TMD	BDI, SAI, MMPI	TMD subgroups identified on basis of scores on pain and distress measures, anxiety, depression, and somatic overconcern; differences supported by discriminant function analysis
LeResche et al, 1992[157]	36 chronic TMD 46 acute TMD	McGill, BDI, SAI, MMPI, SCL-90, CSQ, Daily Hassles Scale	Recent onset and chronic TMD cases did not differ on measures except for pain facial expression
McCreary et al, 1992[158]	95 TMD 73 control	McGill, BDI, SAI, MMPI	TMD patients scored higher for depression and anxiety; somatizers were less likely to respond to TMD treatment
Bush et al, 1993[159]	95 TMD	Orofacial Pain Symptom Checklist, McGill, IBQ	More females than males report psychologic disturbance with their TMD and are likely to seek treatment
Flor et al, 1993[160]	44 TMD 213 chronic back pain 38 control	Pain-Related Self Statements Scale, Pain-Related Control Scale	Four scales valid for chronic pain patients and related to pain intensity—catastrophizing, coping, helplessness, resourcefulness
Oakley et al, 1993[161]	116 TMD	Schedule of Recent Experience, BDI, SAI, subjective evaluation	TMD group exhibited mild depression, anxiety, recent life stress
Parker et al, 1993[162]	110 TMD	MMPI	Four personality profiles extracted from TMD patients—psychophysiologic, depressive, defensive, no diagnosis; TMD patients similar to other chronic pain patients
Jaspers et al, 1993[95]	53 TMD	WHYMPI, General Health Questionnaire, SCL-90, Coping With Specific Symptoms Questionnaire	Psychologic distress and pain severity were low in TMD patients, and there was little interference by pain with daily life
Schulte et al, 1993[163]	109 TMD	SCL-90	Three TMD subgroups were identified on the basis of the following symptoms: somatization; somatization + depression + anxiety; and the full range of psychopathology
Dworkin and Massoth, 1994[150]	261 TMD (functional and dysfunctional)	SCL-90	Dysfunctional TMD patients scored higher on depression and somatization than functional TMD patients

BDAI Buss Durkee Aggression Inventor
BDI Beck Depression Inventory
CES-D Centers for Epidemiologic Studies–Mood Depression Scale
C IS Clinical Interview Schedule
CMI Cornell Medical Index
CPFQ Cattell Personality Factor Questionnaire
CSQ Coping Strategies Questionnaire
EPP Edwards Personality Profile
IAC Interpersonal Adjective Checklist
IBQ Illness Behavior Questionnaire
IPAT IPAT Depression Scale
MADRS Montgomery-Asberg Depression Rating Scale
McGill McGill Pain Questionnaire
MMPI Minnesota Multiphasic Personality Inventory
MNTI Marke Nyman Temperament Inventory
SAI Spielberger State-Trait Anxiety Inventory
SBPI Symptomatology Basic Personality Inventory
SCL-90 Symptom Checklist 90–Revised
SRRS Social Readjustment Rating Scale
SUSI Situational Unpleasant Sensitivity Inventory
TAT Thermatic Apperception Test
VAS Visual Analog Scale
WCC Ways of Coping Checklist
WHYMPI West Haven–Yale Multidimensional Pain Inventory

evidence that psychologic factors play a part in the etiology of TMD. Shipman[141] and Schwartz[142] also reported depression in the MMPI profiles of their patients. In a comparative study, Olson and Schwartz[143] assessed the degree of depression in patients with TMD and in medical patients by the MMPI and reported depression in the former to be of reactive type, as a response to illness. Deardorff et al[144] have found preliminary support for the MMPI-2 in the assessment of chronic pain patient characteristics. Heloe et al[145] reported that those with a multiproblem TMD in Norway denied stress, had severe problems of interpersonal contact, ie, disturbed ability to express personal feelings, and showed greater levels of somatization. The problem with these studies is that they have been conducted in selected samples and generally lack in the definition of sampling techniques, adequate and/or matched controls, and statistical analysis. However, in a more recent study, Feinmann and Harris[146] supported the prevalence of depression in patients presenting with facial pain and success with antidepressant medication. Likewise, Gerschman et al[147] and Tversky et al[148] indicated that the role of depressive illness is of importance in the outcome of treatment in patients with TMD.

Recent attempts have been made to correlate psychologic factors to subgroups of TMD.[149,150] Eversole et al[151] compared patients with myofascial pain (MFP), temporomandibular joint internal derangement (TMJID), and atypical facial pain (AFP) using the MMPI. They found that patients with MPD and TMJID differed in age and personality when compared to patients with AFP. Patients diagnosed as suffering from MPD and AFP showed significantly more hypochondriasis, depression, and hysteria when compared to patients with TMJID. Bush et al[152] considered patients with MPD to be more psychologically distressed compared to patients with TMJID. Psychologic factors have been evaluated also in treatment outcome studies.[153–155] Recent studies have administered relatively large batteries of psychologic questionnaires to subjects with TMD and to control subjects, and the responses have been largely unanimous. Patients with TMD report higher levels of anxiety (state and trait), depression, and somatization than do control subjects.[156–163]

Studies that have compared patients with TMD to asymptomatic or other pain groups have either reported some differences[74,79,164–168] or no differences.[169–172] The comparative assessment of these studies is difficult, however, as a result of differing methodologies used.

Recently new testing instruments have been proposed.[173] McKinney et al[174] compared 78 patients with chronic TMD and 98 patients suffering from chronic pain but not TMD, using the Chronic Pain Battery (CPB). They found that patients with TMD had lower pain intensity and suffering, fewer vegetative symptoms, higher tolerance to pain, less impairment of activity, more likely a successful treatment outcome, lower health care utilization, but higher stress levels. Patients suffering from TMD therefore appeared to be less "handicapped" by their pain and differed in their perception of the disorder as compared to the chronic pain group. Before wider application of the many commercially available instruments, validity and reliability assessments are needed, especially if these instruments are applied cross-culturally.

There appears to be evidence of affective disturbance in patients suffering from TMD, especially in those with a poor response to therapy. The variability in motivational-affective factors in different studies could be the result of interindividual variability, which could be related to emotional factors, but also to cognitive, biophysiologic, and environmental factors that can interact and influence pain perception, disability, and control. The variability could be related also to the inadequate testing methods, selection of samples, and inadequate statistical analyses. For example, the validity of the commonly used testing by the MMPI has been questioned because of its inability to differentiate pain patients or reliably predict response to treatment.[175] Others, however, present a more optimistic view of the MMPI and its subscales in the assessment of chronic pain patients.[176]

Behavioral Factors

Definitions and Determinants. An additional factor to consider when assessing patients suffering from pain is illness behavior. The illness behavior concept was introduced by Mechanic[177] to refer to "the ways in which given symptoms may be differentially perceived, evaluated, and acted (or not acted) upon by different kinds of persons." Several factors have been considered to influence illness behavior, such as social class, social role, age, gender, learning, cultural factors, stress, interpersonal factors, even the type of illness.[177] Cultural factors, including family, social, and environmental factors, have been shown to be of importance in the way people respond to their pain. Zborowski's classic study[178] on cultural influences on pain showed that Jewish, Italian, Irish, and "old American" patients responded differently to pain. The Jewish and Italian tended to respond more emotionally by comparison with the other two groups. The response to pain thus seems to be influenced by the cultural context in which the patient and his/her family and the community react in socially modeled ways. Mechanic[177] stated that "it is necessary that we learn a good deal more about the various attitudes, values, and social definitions applied to symptoms, and how these influence the adoption of patient roles."

Pilowsky[179,180] reformulated the concept of illness behavior as "the ways in which individuals react to aspects of their own functioning which they evaluate in terms of 'health' and 'illness.'" He introduced a concept of abnormal illness behavior (AIB) as an extension to the sociologic model of "sick role." The sick role[181] refers to a "partially and conditionally legitimated state which an individual may be granted, provided he accepts that it is 'undesirable' and recognizes his obligation to cooperate with others for the purpose of achieving 'health' as soon as possible." Illness, on the other hand, was referred to as "a state of the organism which fulfills the requirements of an appropriate reference group, for admission to the sick role."[181] Pilowsky[180] elaborated on these earlier concepts

and defined AIB as "the persistence of an inappropriate or maladaptive mode of perceiving, evaluating and acting in relation to one's own state of health, despite the fact that a doctor has offered a reasonably lucid explanation of the nature of illness and the appropriate course of management to be followed, based on a thorough examination and assessment of all parameters of functioning and taking into account the individual's age, education and sociocultural background."[180]

The definition of illness behavior therefore encompasses several dimensions. It refers to not only behaviors, but also to thoughts (cognitive aspects) and feelings (affective aspects).[150,182] The behavioral aspects can be influenced by family and social setting, culture, and environment (modeling). Fordyce[118] has signified the behavioral dimensions and proposed new treatments that focus specifically on behavioral responses to illness. He has suggested the therapeutic use of positive (encouragement of well behavior) and negative reinforcers (discouragement of ill behaviors) in a family and social setting. The main affective (emotional) aspects of illness behavior include depression, anxiety, and anger. As already discussed in the previous section, the cognitive aspects can influence and interact with these dimensions.

Illness Behavior and TMD. One of the major problems in the treatment of TMD has been the lack of factors to predict treatment response. It has been proposed that about 5% to 10% of patients with TMD will be refractory to treatment.[183] Understanding the multiple elements that influence illness behavior has been shown to be important in the management of musculoskeletal disorders affecting other parts of the body, such as lower back pain.[66,99] It has been used also to study patients with TMD. Speculand et al[184] compared a group of 100 Australian patients with TMD and 100 control subjects. They found that patients with TMD showed increased disease conviction, increased affective disturbance, and decreased denial of other life problems apart from illness when compared to the control group. Nevertheless, these factors failed to differentiate between the TMD and control samples. When the TMD sample was compared to patients attending pain clinics, several differences were noted. This latter group was shown to have greater disease conviction and greater tendency to somatize. The percentage of patients showing abnormal illness behavior was 23% for the TMD group. In comparison, 19% in the control group showed abnormal illness behavior. The finding that the control and TMD samples could not be discriminated is of interest in relation to other comparative studies that have shown inconsistent findings in relation to personality, emotional, and psychogenic factors. Speculand et al[184] proposed that illness behavior assessment could be used as a screening device to identify those patients with an abnormal illness behavior and provide them with appropriate treatment in a multidisciplinary clinic.

Another approach to study behavior is by observing overt pain behavior. Fordyce[118] viewed pain behaviors as being ways for the patients to communicate to others that they experience pain. These behaviors included decreased activity, guarded movement, body posturing, and certain facial expressions. Some patients showed exaggerated or inconsistent pain behavior. Fordyce[118] explained these behaviors to be more related to conditioning and learning influences than organic pathology. They could be expressions for attention and sympathy to avoid unwanted home and work responsibilities. Pain behavior indexes, such as activity level or medication intake, were usually elevated. Several assessment instruments to measure these behaviors have been developed,[185] but the relationship between overt behavior and TMD is not known.

Assessment of Illness Behavior. For the assessment of illness behavior, an interview method (Illness Behavior Assessment Schedule [IBAS]) or a questionnaire method (the Illness Behavior Questionnaire [IBQ]) have been proposed.[180,186,187] Other observational methods have been developed also.[35,185]

When using the IBAS, interview evaluations are made of the patients' perception of the information they have received and their acceptance of it, their conceptualization of the type of illnesses they have, their awareness of symptoms and associated preoccupations or phobic attitudes, their ideas about etiology, the affective state, and the extent to which somatic illness is being used as a defense.

The IBQ was originally developed as a 52-item self-report questionnaire[186] and since has been expanded to include 62 items.[187] In the original factor analysis, seven parameters were identified, including general hypochondriasis, disease conviction, somatic versus psychologic view of illness, affective inhibition, affective disturbance, denial of life problems apart from illness, and irritability. The IBQ has since been used in several pain populations, including chronic pain patients,[186,188–191] general practice patients,[192] patients attending rheumatology, radiotherapy, pulmonary, and physiotherapy clinics,[186] patients with somatic illness such as myocardial infarction[193] and coronary artery bypass,[194] headache,[195] and TMD.[184] In

general, studies using the IBQ have shown that patients with intractable pain are more convinced of the presence of the disease and more somatically preoccupied.

In their original study, Pilowsky and Spence[186] studied 100 patients with chronic pain and 40 patients attending rheumatology, radiotherapy, pulmonary, and physiotherapy clinics who reported pain as a prominent symptom. Significant differences were found in disease conviction, indicating that patients with intractable pain were more convinced of the presence of illness, were more somatically preoccupied, and could not seem to accept reassurance from a doctor, ie, displayed abnormal illness behavior. These findings agreed with the earlier clinical observation by Smith et al,[196] who reported a high incidence of hypochondriasis in patients with atypical facial pain. These studies have shown that patients who are preoccupied with their symptoms and have difficulty expressing emotional distress are more significantly impaired by persistent pain than those who are less preoccupied and who express their feelings more openly. Pilowsky and Spence[197] related this observation to abnormal illness behavior and stated that psychologic and psychiatric interventions may be useful in the treatment of these patients.

Demjen and Bakal[195] tested the IBQ in another group of patients suffering from chronic headache and assessed illness behavior in relation to the severity of symptoms. Using a factor analytic procedure, they confirmed the original factor structure by Pilowsky and Spence[186] for the study of patients with chronic headache. They also found that those patients with the greatest headache activity and those with continuous pain viewed their disorder in somatic terms. Those with continuous pain also scored higher in denial when compared to patients with episodic pain. Compared to the study by Pilowsky and Spence,[186] patients with headache were found to differ from patients with intractable pain. The headache group scored higher in hypochondriasis and psychologic versus somatic perception of illness and significantly lower in disease conviction and denial, thus indicating greater acceptance of psychologic factors. The authors explained this to be a result of the episodic versus continuous nature of the pain experience in the two groups. Patients with continuous or near continuous headache patterns resembled more closely the intractable pain group by Pilowsky and Spence,[186] ie, had more somatic preoccupation and more denial of other problems apart from illness in their lives. The findings in this study were interpreted to demonstrate the utility of examining psychologic components of chronic headache syndrome from a severity perspective.

In a study of patients suffering from chronic lower back pain, Keefe et al[198] also found that scores on the IBQ were highly predictive of a variety of indexes of pain and pain behavior. Pilowsky et al[192] have used the IBQ also to compare two groups of general practice patients, one with an observable organic pathology and the other with absence of organic pathology. The latter group showed a greater disease conviction and a greater degree of anxiety, depression, and irritability; ie, somatizing patients were more likely to show disease conviction and affective disturbance. Males and females also differed, the former showing more disease conviction, somatic focusing, and hypochondriasis.

Recently in a study of 200 British patients suffering from chronic pain, Main and Waddell[191] challenged the validity of the original factor structure proposed by Pilowsky and Spence.[186] They constructed three new scales to replace the original ones, including affective and hypochondriac disturbance, life disruption, and social inhibition. Waddell et al[199] have also examined illness behavior in relation to behavioral symptoms and signs, objective physical impairment, pain and disability, and psychometric measures of distress and the IBQ. These factors were also related to the outcome of treatment. Their results showed that behavioral symptoms and signs were directly related to physical severity of the lower back pain problem, report of pain and disability, and the outcome of surgical treatment. The IBQ scores were also strongly related to measures of affective disturbance and psychologic distress. The disease affirmation scale (including disease conviction and psychologic versus somatic focusing scales) was important in relation to behavioral symptoms and signs. It was concluded that disease conviction should not be seen only as a function of disease process, but more as a psychologic coping mechanism for certain individuals under stress. In a study by Wichmann et al,[200] similar observations of the modifications needed to the original factor structure by Pilowsky and Spence[186] and as observed by Main and Waddell[191] were proposed.

It seems that understanding aspects of illness behavior is important in the management of patients suffering from a variety of pain problems, especially when the problem is resistant to treatment. Further, the understanding of environmental influences, such as ethnocultural and social factors (including family, community) in pain experience and expression are also of importance to better understand a pain response, the resolution of

symptoms, and the acceptance of certain therapy interventions.

Sociocultural Factors and TMD

Theories. It has been documented that cultural, ethnic, social, and family factors can play an important part in pain experience and expression.[201,202] They can influence all three dimensions of pain perception (sensory, cognitive, affective) and the response to pain to varying degrees, as well as health-seeking behavior.[203]

Zborowski[178] reported major interethnic differences in Irish, Italian, Jewish, and Old American patients in his much quoted study. The data were collected by observation and interview, and the results indicated that Jewish and Italian patients were more emotional and more expressive about their pain than the other two groups. Zborowski's findings were supported by Zola,[204] who reported interethnic differences in responses and attitudes to pain among Italian Catholic, Irish Catholic, and Anglo-Saxon Protestant patients. Others have found only few[205,206] or no[207] interethnic differences. The study by Weisenberg et al[205] comprised black, white, and Puerto Rican patients with acute dental pain. They found that the Puerto Rican group had a greater tendency to avoid dealing with pain either by using denial or by trying to cure it, but found no differences between the other two groups. In their study of racial differences among the blacks and whites, Garron and Leavitt[206] only found differences in the pain description. All of these studies, however, lack in the definition of sampling, methodology, and statistical testing. The results also lack in the analysis of other intraethnic differences that may influence the results reported.

In a more recent study, Lipton and Marbach[208] found that ethnic groups (in this study, Irish, Italian, Jewish, and Puerto Rican patients with facial pain) differed with respect to factors that influence pain response, but not in the actual responses to pain. Statistically significant differences were found in quality and intensity of pain, search for meaning and cause of pain, emotionality of responses, interference in daily functioning, medical care sought, and behaviors and attitudes comprising the "pain patient role." So, while intraethnic heterogeneity dominated factors that influence the experience, interethnic homogeneity was present for most aspects of pain experience.

There are several factors to consider that may cause variability both within and between ethnic groups.[203,208] These include acculturation, socioeconomic class, degree to which cultural aspects are adhered, social situation and situational demands, as well as the way ethnic patterns may be selectively expressed. Both Craig and Wyckoff[203] and Lipton and Marbach[208] cautioned against stereotyping. A further aspect to be considered is that both the pain sufferer and the health provider will perceive pain according to their specific sociocultural learning. Recent reviews by Bates et al[202] and Ecandela[209] stress that pain intensity can best be examined within the framework of the attitudes and beliefs of one's ethnic group affiliation. The West Haven–Yale Multidimensional Pain Inventory (WHYMPI[210]) has proven useful in assessing the relationship between psychosocial variables and pain (see Kerns and Jacob[211] for a review) and may prove to be an effective instrument in recording the psychosocial factors relevant in TMD.

Bates[212] presented a biocultural model that incorporated aspects of the gate control theory of pain, the social learning theory of Bandura,[51] and the theory of social comparison of Festinger.[213] This model was developed to illustrate variability in pain-response patterns and biocultural influences and to show that ethnocultural values, by determining attitudes and attention, can influence the biologic inhibitory control system.

Ethnicity and culture have been suggested to influence health beliefs (meaning of symptoms) and illness behavior (through social modeling), including the ways of perceiving, labeling, responding and communicating symptoms, and health care utilization. Nevertheless, factors such as social situation, intraethnic variation, degree of acculturation, and social class can influence the expression of these ethnic patterns

Psychiatric Disorders. Psychiatric factors, apart from influencing the complex pain experience, can also be present in patients without demonstrable organic pathology. The theoretical aspects were discussed in an earlier section.

Several psychiatric conditions have been diagnosed in patients suffering from orofacial pain conditions. In the study by Gerschman et al,[147] which included 138 patients from heterogenous ethnic groups treated in a multidisciplinary pain clinic, the most frequent psychiatric diagnoses included neurotic disorder, depressive illness, major affective disorder, and personality disorder. In another study by Remick and Blasberg,[214] psychiatric diagnoses in 121 patients with atypical facial pain included affective disorder, somatoform disorder, adjustment disorder, psychosis, anxiety disorder, psychologic factor affecting physical con-

dition, personality disorder, barbiturate dependence, dementia, and malingering. Psychiatric diagnoses in other societies include the study by Hampf[215] in which 38 Finnish patients with atypical facial pain were diagnosed with major psychiatric disorders such as personality disorder, atypical psychosis, and psychogenic pain disorder.

Generally, these studies illustrate the variety of psychiatric conditions that could be present in patients with chronic TMD. Rome et al[216] divided the major psychiatric diagnoses in TMD into the following groups: adjustment disorder; dysthymia (chronic depression) or major depression; and posttraumatic stress disorder. The adjustment disorder mainly manifested as a reaction to the painful disorder and presented as (generally mild) depression/anxiety with usual manifestations of limited activity, sleep disturbance, and increased awareness of bodily sensations. If the condition persisted, dysthymia or major depression/anxiety could follow. As Biondi and Picardi[217] and Milliner[218] pointed out, thorough diagnoses of TMD take into account the patients' personality types, their psychosocial backgrounds, and importantly, the presence or absence of psychopathologic symptoms concurrent with the TMD.

Critical Summary of the Assessment of Pain in Patients With TMD and Proposals for Further Research

In the present review, research concerning different methods of assessing pain and psychologic factors in patients with TMD have been discussed. An examination of these studies has raised the following questions:

Are the Subjects in Previous Studies Representative of Patients With TMD?

Studies of patients with TMD who have used acceptable statistical selection criteria (random design) representative of the clinic population are rare. The majority of psychologic assessment studies have been conducted cross-sectionally in selected clinic populations. Generally, these studies have implicated a wide variety of psychologic factors in patients with TMD both within and between samples. This variability between studies could be a result of not only the sample selection, methodology, and study designs, but also to individual differences, which could be related to sensory (threshold, tolerance), cognitive (beliefs, meanings, attributes), affective (emotions), and behavioral (environment) dimensions.[6] Presently, caution needs to be expressed in the generalization of the findings in psychologic studies because findings may only be valid for those samples studied.

Are the Studies Comparative and Repeatable?

Standards for diagnosis and classification of patients with TMD have varied in the past and between research groups. In the earlier studies, there was a lack of detailed reporting of the selection and diagnostic criteria to allow for critical comparison between different studies. A further problem in comparative assessment of different studies has been the variety of testing methods used. These have included one-dimension approaches and assessment by different instruments. Most earlier studies were descriptive and lacked adequate controls to allow for comparison, repeatability, or clinical application of the results reported. The majority of psychologic surveys, even to date, lacks the reporting of scientific statistical principles such as validity and reliability assessments of the instruments used. Before reliability assessments can be made, the commercially available instruments should be more adequately tested, especially if they are to be applied cross-culturally. The absence of psychometric testing in TMD research has been noted recently.[219]

Do Patients With TMD Differ From Control Subjects?

According to the literature reviewed, reports on psychologic factors between patients with TMD and control subjects have not been consistent.[6] A variety of different psychologic profiles have been reported. The earlier studies, based on interviews and descriptive data, indicate psychologic disturbances in patients with TMD, but these studies lacked control subjects and random selection of patients. The more recent studies often have lacked adequate control samples. In those reports that have included acceptable control subjects (of either normal population or other pain populations), findings have varied. Some have reported few or no differences between patients with TMD and control subjects,[169–171,220,221] while others have reported a difference.[74,79,101,167,168,184] Factors such as affective disturbances, illness behavior, differences in coping, attributions, and hardiness have been reported. The role of these factors in the etiology of TMD is not clear, however. As raised in the first and second questions, many factors may

contribute to this variability, such as sample selection, diagnostic and assessment methods, data analysis, and interindividual differences.

What Has Been Learned About the Assessment of Pain in Patients With TMD?

The assessment of pain is a complex area of research. The subjective and private nature of pain experience means that it can be measured only indirectly, ie, by how it is described by the patient or by observing the patients' behavior. Patients with pain generally form a heterogenous population. Most present with differing disease status; eg, the intensity of pain may vary, not only between, but also within individuals at different times.[222] It is now recognized, however, that pain, including pain in TMD, is a complex, multifactorial experience including not only sensory-discriminative dimensions, but also motivational, affective, and cognitive factors that all interrelate and affect the pain response and expression by the patients. From a theoretical basis, if adopting the multidimensional pain model,[3,4] the question of measuring the multiple factors contributing to the experience of pain is a difficult one. Much of the early research in this field has been empirical and analyzed only one or few dimensions of pain and its psychologic correlates. There has been a lack of studies that have used validated instruments or psychometrically tested the validity and reliability of the instruments used in patients with TMD. Many have lacked longitudinal designs; eg, it is not known how different psychologic dimensions change with time.

Thus far there has been a lack of evidence for specific psychologic profiles for patients with TMD. The results reported in different studies have indicated a multifactorial basis for TMD, and thus a need for the assessment of patients with TMD to be from a multifactorial perspective. Few studies exist where multiple factors have been studied systematically. Multivariate statistical principles have been applied only recently to analyze the contribution of multiple variables in factors associated with patients suffering from TMD.

Based on the critical summary of the assessment of pain, the following proposals are suggested for further research. The assessment should be done in large randomized samples, including matched control case studies, with validated instruments. The selection, diagnosis, and exclusion criteria, as well as the drop-out rate or intend-to-treat groups should be well documented.[150] If it is accepted that TMD is multifactorial, appropriate statistics should be employed to study the influence of multiple variables.

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References

1. Dworkin SF, LeResche L. Research Diagnostic Criteria for Temporomandibular Disorders: Review, Criteria, Examinations and Specifications, Critique. J Craniomandib Disord Facial Oral Pain 1992;6:301–355.
2. Melzack R, Wall PD. Pain mechanisms: A new theory. Science 1965;150:971–979.
3. Melzack R, Casey KL. Sensory, motivational, and central control determinants of pain: A new conceptual model. In: Kenshalo DR (ed). The Skin Senses. Springfield, IL: Thomas, 1968.
4. Rugh JD. Psychologic components of pain. Dent Clin North Am 1987;31:579–594.
5. Gamsa A. The role of psychologic factors in chronic pain. I. A half century of study. Pain 1994;57:5–15.
6. Gamsa A. The role of psychologic factors in chronic pain. II. A critical appraisal. Pain 1994;57:17–29.
7. Merskey H. Pain terms: A list with definitions and notes on usage. Recommended by the IASP Subcommittee on taxonomy. Pain 1979;6:249–252.
8. Melzack R. The Puzzle of Pain. New York: Basic Books, 1973.
9. Weisenberg M. Pain and pain control. Psychol Bull 1977; 84:1008–1044.
10. von Frey M. Beitrage zur Sinnesphysiologie der Haut. Ber d kgl sachs Ges d Wiss, math-phys Kl 1895;47:181. Cited in: Melzack R. The Puzzle of Pain. New York: Basic Books, 1973.
11. Beecher HK. Measurement of Subjective Responses: Quantitative Effects of Drugs. New York: Oxford University Press, 1959.
12. Pavlov IP. Conditioned Reflexes. New York: Milford, 1927. Cited in: Melzack R. The Puzzle of Pain. New York: Basic Books, 1973.
13. Pavlov IP. Lectures on Conditioned Reflexes. New York: International Publishers, 1928. Cited in: Melzack R. The Puzzle of Pain. New York: Basic Books, 1973.
14. Goldscheider A. Über den Schmerz in Physiologischer und Klinischer Hinsicht. Berlin: Hirschwald, 1894.
15. Livingston WK. Pain Mechanisms. London: MacMillan, 1943.
16. Noordenbos W. Pain. Amsterdam: Elsevier Press, 1959.
17. Melzack R, Dennis SG. Neurophysiological foundations of pain. In: Sternbach RA (ed). The Psychology of Pain. New York: Raven Press, 1978.
18. Wall PD. The gate control theory of pain mechanisms: A re-examination and re-statement. Brain 1978;101:1–18.
19. Bell WE. Orofacial Pains, ed 4. Chicago: Year Book Medical, 1990.

20. Sessle BJ. Anatomy, physiology and pathophysiology of orofacial pain. In: Jacobson AL, Donlon WC (eds). Headache and Facial Pain. Diagnosis and Management. New York: Raven Press, 1990.
21. Dubner R, Sessle BJ, Storey AT. The neural basis of oral and facial function. New York: Plenum Press, 1978.
22. Sessle BJ. The neurobiology of facial and dental pain: Present knowledge, future directions. J Dent Res 1987;66:962–981.
23. Fields H. Depression and pain: A neurobiological model. Neuropsychiatr Neuropsychol Behav Neurology 1991;4: 83–92.
24. Lund JP, Donga R, Widmer CG, Stohler CS. The pain–adaptation model: A discussion of the relationship between chronic musculoskeletal pain and motor activity. Can J Physiol Pharmacol 1991;69:683–694.
25. Sessle BJ. Recent developments in pain research: Central mechanisms of orofacial pain and its control. J Endod 1986;12:435–444.
26. Ramfjord SP, Ash MM. Occlusion, ed 3. Philadelphia, PA: Saunders, 1983.
27. Weisenberg M. Cognitive aspects of pain. In: Wall PD, Melzack R (eds). Textbook of Pain. Edinburgh: Churchill Livingstone, 1984.
28. Sternbach RA. Behavior therapy. In: Wall PD, Melzack R (eds). Textbook of Pain. Edinburgh: Churchill Livingstone, 1984.
29. Sternbach RA, Janowsky DS, Huey LY, Segal DS. Effects of altering brain serotonin activity on human chronic pain. In: Bonica JJ, Albe-Fessard D (eds). Advances in Pain Research and Therapy, vol 1. New York: Raven Press, 1976.
30. Melzack R, Loeser JD. Phantom body pain in paraplegics: Evidence for a central 'pattern generating mechanism' for pain. Pain 1978;4:195–210.
31. Wall PD. Mechanisms of acute and chronic pain. In: Kruger L, Liebeskind JC (eds). Advances in Pain Research and Therapy, vol 6. New York: Raven Press, 1984.
32. Sessle BJ, Hu JW. Mechanisms of pain arising from articular tissues. Can J Physiol Pharmacol 1991;69: 617–626.
33. Sessle B. Brain stem mechanisms of orofacial pain. In: Fricton JR, Dubner R (eds). Advances in Pain Research and Therapy, vol 21. New York: Raven Press, 1995.
34. Fricton JR, Dubner R (eds). Advances in Pain Research and Therapy: Orofacial Pain and Temporomandibular Disorders, vol 21. New York: Raven Press, 1995.
35. Chapman CR, Casey KL, Dubner R, Foley KM, Gracely RH, Reading AE. Pain measurement: An overview. Pain 1985;22:1–31.
36. Helkimo M. Epidemiological surveys of dysfunction of the masticatory system. In: Zarb GA, Carlsson GE (eds). Temporomandibular Joint Function and Dysfunction. Copenhagen: Munksgaard, 1979.
37. Fricton JR, Schiffman EL. The craniomandibular index: Validity. J Prosthet Dent 1987;58:222–228.
38. Levitt SR, McKinney MW, Lundeen TF. The TMJ scale: Cross-validation and reliability studies. J Craniomand Pract 1988;6:17–25.
39. Levitt SR, McKinney MW, Willis WA. Measuring the impact of a dental practice on TM disorder symptoms. J Craniomand Pract 1993;11:211–216.
40. Levitt SR. Predictive value: A model for dentists to evaluate the accuracy of diagnostic tests for temporomandibular disorders as applied to a TMJ scale. J Prosthet Dent 1991;66:385–390.
41. Spiegel EP, Levitt SR. Measuring symptom severity with the TMJ scale. J Craniomandib Disord Facial Oral Pain 1991;4:177–185.
42. Levitt SR. The predictive value of the TMJ scale in detecting psychologic problems and non-TMJ disorders in patients with temporomandibular disorders. J Craniomand Pract 1990;8:225–233.
43. Levitt SR. Predictive value of the TMJ scale in detecting clinically significant symptoms of temporomandibular disorders. J Craniomandib Disord Facial Oral Pain 1990; 4:177–185.
44. Stedman's Concise Medical Dictionary, ed 2. Baltimore: Williams and Wilkins, 1994:206.
45. Lazarus RS, Folkman S. Stress, Appraisal, and Coping. New York: Springer, 1984.
46. Roskies E, Lazarus RS. Coping theory and the teaching of coping skills. In: Davidson PO, Davidson SM (eds). Behavioral Medicine: Changing Health Lifestyles. New York: Brunner/Mazel, 1980.
47. Zimbardo PG, Cohen AR, Weisenberg M, Dworkin L, Firestone I. Control of pain motivation by cognitive dissonance. Science 1966;151:217–219.
48. Zimbardo PG, Cohen AR, Weisenberg M, Dworkin L, Firestone I. The control of experimental pain. In: Zimbardo PG (ed). The Cognitive Control of Motivation. Glenview, IL: Scott Foresman, 1969.
49. Nisbett RE, Schachter RS. Cognitive manipulation of pain. J Exp Soc Psychol 1966;2:227–236.
50. Gottlieb H, Strite LC, Koller R, Madorsky A, Hockersmith V, Kleeman M, Wagner J. Comprehensive rehabilitation of patients having chronic low back pain. Arch Phys Med Rehabil 1977;58:101–108.
51. Bandura A. Self-efficacy: Towards a unifying theory of behavioral change. Psychol Rev 1977;84:191–215.
52. Crisson JE, Keefe FJ. The relationship of locus of control to pain coping strategies and psychological distress in chronic pain patients. Pain 1988;35:147–154.
53. Bowers KS. Pain, anxiety and perceived control. J Consult Clin Psychol 1968;32:596–602.
54. Staub E, Tursky B, Schwartz GE. Self-control and predictability: Their effects on reactions to aversive stimulation. J Pers Soc Psychol 1971;18:157–162.
55. Thompson SC. Will it hurt less if I can control it? A complex answer to a simple question. Psychol Bull 1981; 90:89–101.
56. Lowery BJ, Jacobsen BS, Murphy BB. An exploratory investigation of causal thinking of arthritics. Nurs Res 1983;39:157–162.
57. Turk DC, Rudy TE. Assessment of cognitive factors in chronic pain: A worthwhile enterprise? J Consult Clin Psychol 1986;54:760–768.
58. Ellis A. Reason and Emotion in Psychotherapy. New York: Lyle Stuart, 1962.
59. Beck AT. Cognitive therapy and the emotional disorders. New York: International Universities Press, 1976.
60. Ciccone DS, Grzesiak RC. Cognitive dimensions of chronic pain. Soc Sci Med 1984;19:1339–1345.
61. Langer E. The Psychology of Control. London: Sage, 1983.
62. Pennebaker J. The Psychology of Physical Symptoms. New York: Springer-Verlag, 1982.
63. Turk DC, Meichenbaum D, Genest N. Pain and behavioral medicine: A cognitive-behavioral perspective. New York: Guilford Press, 1983.
64. Brown GK, Nicassio PM. Development of a questionnaire for the assessment of active and passive coping strategies in chronic pain patients. Pain 1987;31:53–64.

65. Turner JA. Coping strategies of patients with chronic pain [abstract]. Pain 1990;5(special issue):251.
66. Rosenstiel AK, Keefe FJ. The use of coping strategies in chronic low back pain patients: Relationship to patient characteristics and current adjustment. Pain 1983;17: 33–44.
67. Fernandez E, Turk DC. The utility of cognitive coping strategies for altering pain perception: A meta analysis. Pain 1989;38:123–135.
68. Beck A, Emery G, Greenberg RL. Anxiety disorders and phobia. New York: Basic Books, 1985.
69. Beck AT, Rush AJ, Shaw BF, Emery G. Cognitive Therapy of Depression. New York: Guilford Press, 1979.
70. Tan SY. Cognitive and cognitive-behavioral methods for pain control: A selective review. Pain 1982;12:201–228.
71. Turner JA, Chapman CR. Psychologic interventions for chronic pain: A critical review. II. Operant conditioning, hypnosis and cognitive-behavioral therapy. Pain 1982; 12:23–46.
72. Leavitt F, Carron DC, Bieliauskas LA. Psychologic disturbance and life event differences among patients with low back pain. J Consult Clin Psychol 1980;48:115–116.
73. Lichtenberg PA, Skehan MW, Swenson CH. The role of personality, recent life stress and arthritic severity in predicting pain. J Psychosom Res 1984;28:231–236.
74. Stein S, Loft G, Davis H, Hart DL. Symptoms of TMJ dysfunction as related to stress measured by the social readjustment rating scale. J Prosthet Dent 1982;47: 545–548.
75. Moody PM, Calhoun TC, Okeson JP, Kemper JT. Stress-pain relationship in MPD syndrome patients and non-MPD syndrome patients. J Prosthet Dent 1981;45: 84–88.
76. Speculand B, Hughes AO, Goss AN. Role of recent stressful life events experience in the onset of TMJ dysfunction pain. Community Dent Oral Epidemiol 1984;12:197–202.
77. Turner JA, Clancy S, Vitaliano PP. Relationships of stress, appraisal and coping, to chronic low back pain. Behav Res Ther 1987;25(special issue: Chronic pain): 281–288.
78. Copp LA. The spectrum of suffering. Am J Nurs 1974; 74:491–495.
79. Lennon MC, Dohrenwend BP, Zautra AJ, Marbach JJ. Coping and adaptation to facial pain in contrast to other stressful life events. J Pers Soc Psychol 1990;59: 1040–1050.
80. Creisser ME, Robinson ME, Henson CD. The Coping Strategies Questionnaire and chronic pain adjustment: A conceptual and empirical reanalysis. Clin J Pain 1994; 10:98–106.
81. Rudy TE, Turk DC, Zaki HS, Curtin HD. An empirical taxometric alternative to traditional classification of temporomandibular disorders. Pain 1989;36:311–320.
82. Greene CS, Laskin DM. Long-term evaluation of treatment for myofascial pain-dysfunction syndrome: A comparative analysis. J Am Dent Assoc 1983;107:235–238.
83. Clark GT, Jacobson R, Beemsterboer PL. Interdental thickness discrimination in myofascial pain dysfunction subjects. J Oral Rehabil 1984;11:381–386.
84. Epstein JB. Understanding placebo in dentistry. J Am Dent Assoc 1984;109:71–74.
85. Beers TM, Karoly P. Cognitive strategies, expectancy, and coping style in the control of pain. J Consult Clin Psychol 1979;47:179–180.
86. Kendall PC, Williams L, Pechacek TF, Graham LE, Shisslak C, Herzoff N. Cognitive-behavioral and patient education interventions in cardiac catheterization procedures: The Palo Alto medical psychology project. J Consult Clin Psychol 1979;47:49–58.
87. Pickett C, Clum GA. Comparative treatment strategies and their interaction with locus of control in the reduction of postsurgical pain and anxiety. J Consult Clin Psychol 1982;50:439–441.
88. Wernick RL, Jaremko ME, Taylor PW. Pain management in severely burned adults: A test of stress inoculation. J Behav Med 1981;4:103–109.
89. Mitchell KR, White RG. Behavioral self-management: An application to the problem of migraine headaches. Behav Res Ther 1977;8:213–222.
90. Anderson NB, Lawrence PS, Olson TW. Within subject analysis of autogenic training and cognitive coping training in the treatment of tension headache pain. J Behav Ther Exp Psychiatry 1981;12:219–223.
91. Kremsdorf RB, Kochanowicz NA, Costell S. Cognitive skills training versus EMG biofeedback in the treatment of tension headaches. Biofeedback Self Regul 1981;6:93–102.
92. Turner JA. Comparison of group progressive-relaxation training and cognitive-behavioral group therapy for chronic low back pain. J Consult Clin Psych 1982;50: 757–765.
93. Engstrom D. Cognitive behavioral therapy methods in chronic pain treatment. In: Bonica JJ, Lindblom U, Iggo A, Jones LE, Benedetti C (eds). Advances in Pain Research and Therapy, vol 5. New York: Raven Press, 1983.
94. Moore JE, Chaney EF. Outpatient group treatment of chronic pain: Effects of spouse involvement. J Consult Clin Psychol 1985;53:326–334.
95. Jaspers JPC, Heuvel F, Stegenga B, de Bont LGM. Strategies for coping with pain and psychologic distress associated with temporomandibular joint osteoarthrosis and internal derangement. Clin J Pain 1993;9:94–103.
96. Tunks E, Bellissimo A. Coping with the coping concept: A brief comment. Pain 1988;34:171–174.
97. Folkman S, Lazarus RS. An analysis of coping in a middle-aged community sample. J Health Soc Behav 1980; 21:219–239.
98. Brown GK, Nicassio PM, Wallston KA. Pain coping strategies and depression in rheumatoid arthritis. J Consult Clin Psychol 1989;57:652–657.
99. Turner JA, Clancy S. Strategies for coping with chronic low back pain: Relationship to pain and disability. Pain 1986;24:355–364.
100. Gross AR. The effect of coping strategies on the relief of pain following surgical intervention for lower back pain. Psychosom Med 1986;48:229–241.
101. Keefe FJ, Dolan E. Pain behavior and pain coping strategies in low back pain and myofascial pain dysfunction syndrome patients. Pain 1986;24:49–56.
102. Keefe FJ, Caldwell DS, Queen KT, Gil KM, Martinez S, Crisson JE, et al. Pain coping strategies in osteoarthritis patients. J Consult Clin Psychol 1987;55:208–212.
103. Hill A. The use of pain coping strategies by patients with phantom limb pain. Pain 1993;55:347–353.
104. Swartzman LC, Gwadry FG, Shapiro AP, Teasell RW. The factor structure of the Coping Strategies Questionnaire. Pain 1994;57:311–316.
105. De Good DE, Shutty MS. Assessment of pain beliefs, coping, and self-efficacy. In: Turk DC, Melzack R (eds). Handbook of Pain Assessment. New York: Guilford Press, 1992:214–234.

106. Jensen MP, Turner JA, Romano JM, Karoly P. Coping with chronic pain: A critical review of the literature. Pain 1991;47:249–283.
107. Craig KD. Emotional aspects of pain. In: Wall PD, Melzack R (eds). Textbook of Pain. Edinburgh: Churchill Livingstone, 1989.
108. Craig KD. Modelling and social learning factors in chronic pain. In: Bonica JJ (ed). Advances in pain research and therapy. New York: Raven Press, 1983.
109. Dubuisson D, Melzack R. Classification of clinical pain descriptors by multiple group discriminant analysis. Exp Neurol 1976;51:480–487.
110. Beutler LE, Engle D, Oro-Beutler ME, Daldrup R, Meredith K. Inability to express intense affect: A common link between depression and pain. J Consult Clin Psychol 1986;54:752–759.
111. Weickgenant AL, Slater MA, Patterson TL, Atkinson JH, Grant G, Garfin SR. Coping activities in chronic low back pain: Relationship with depression. Pain 1993; 53:95–103.
112. Romano JM, Turner JA. Chronic pain and depression: Does the evidence support a relationship? Psychol Bull 1985;97:18–34.
113. Krause SJ, Wiener RL, Tait RC. Depression and pain behavior in patients with chronic pain. Clin J Pain 1994; 10:122–127.
114. Krishnan KRR, France RD, Pelton S, McCann UD, Davidson J, Urban BJ. Chronic pain and depression. II Symptoms of anxiety in chronic low back pain patients and their relationship to subtypes of depression. Pain 1985;22:289–294.
115. Fields HL, Besson J-M. Pain modulation. In: Progress in Brain Research, vol 77. Amsterdam: Elsevier, 1988.
116. Gaffney A, Dunne EA. Children's understanding of the causality of pain. Pain 1987;29:91–104.
117. Flor H, Haag G, Turk DC, Koehler G. Efficacy of EMG biofeedback, pseudotherapy, and conventional medical treatments for chronic rheumatic pain. Pain 1983;17: 21–32.
118. Fordyce WE. Behavioral Methods for Chronic Pain and Illness. St Louis: Mosby, 1976.
119. Woodforde JM, Merskey FG. Some relationships between subjective measures of pain. J Psychosom Res 1971;16:173–178.
120. Heaton RK, Getto CJ, Lehman AW, Fordyce WE, Brauer E, Groban SE. A standardized evaluation of psychosocial factors in chronic pain. Pain 1982;12:165–174.
121. Katon W, Kleinman A, Rosen G. Depression and somatization: A review (parts I and 2). Am J Med 1982;72: 127–247.
122. Merskey H. Psychologic aspects of pain. Postgrad Med J 1968;44:297–306.
123. Timmermans G, Sternbach RA. Factors of human chronic pain: An analysis of personality and pain reaction variables. Science 1974;184:806–808.
124. Sternbach R. Pain Patients: Traits and Treatment. New York: Academic Press, 1974.
125. Horan JA, Dellinger DK. 'In vivo' emotive imagery: A preliminary test. Percept Mot Skills 1974;39:359–362.
126. Price DD, McHaffie JG. Effects of heterotopic conditioning on first and second pain: A psychophysical evaluation in humans. Pain 1988;34:245–252.
127. Rugh JD. Psychologic factors in the etiology of masticatory pain and dysfunction. In: Laskin D, Greenfield W, Gale E (eds). The President's Conference on the examination, diagnosis and management of temporomandibular disorders. Chicago: American Dental Association, 1983.
128. Engel GL. Primary atypical facial neuralgia. Psychosom Med 1951;13:375–396.
129. Lesse S. Atypical facial pain syndromes of psychogenic origin. J Nerv Ment Dis 1956;124:346–351.
130. McCall JCM, Szmyd L, Ritter RM. Personality characteristics in patients with temporomandibular joint symptoms. J Am Dent Assoc 1961;62:694–698.
131. Grieder A. Psychologic aspects of prosthodontics. J Prosthet Dent 1973;30:736–744.
132. Gessel AH. Parometric diagnosis and treatment of temporomandibular joint syndrome [abstract 69]. J Dent Res 1973;52(special issue):76.
133. Moulton R. Psychiatric considerations in maxillofacial pain. J Am Dent Assoc 1955;51:408–414.
134. Kydd WL. Psychosomatic aspects of temporomandibular joint dysfunction. J Am Dent Assoc 1959;59:31–44.
135. Lefer L. A psychoanalytic view of a dental phenomenon: Psychosomatics of the temporomandibular joint pain-dysfunction syndrome. Contemp Psychoan 1966;2:135–150.
136. Lupton DE. A preliminary investigation of the personality of female temporomandibular joint dysfunction patients. Psychother Psychosom 1966;14:199–216.
137. Burton RC. The problem of facial pain. J Am Dent Assoc 1969;79:93–101.
138. Lascelles RG. Atypical facial pain and depression. Br J Psychiatry 1966;112:651–659.
139. Fine EW. Psychologic factors associated with nonorganic temporomandibular joint pain dysfunction syndrome. Br Dent J 1971;131:402–404.
140. Small EW. An investigation into the psychogenic bases of the temporomandibular joint myofascial pain dysfunction syndrome. In: Bonica JJ, Albe-Fessard DG (eds). Advances in Pain Research and Therapy. New York: Raven Press, 1976.
141. Shipman WG. Analysis of MMPI test results in women with MPD syndrome [abstract 82]. J Dent Res 1973;52 (special issue):79.
142. Schwartz RA. Personality characteristics of unsuccessfully treated MPD patients [abstract 291]. J Dent Res 1974;53:127.
143. Olson RE, Schwartz RA. Depression in patients with myofascial pain-dysfunction syndrome [abstract 434]. J Dent Res 1977;56:160.
144. Deardorff WW, Chino AF, Scott DW. Characteristics of chronic pain patients: Factor analysis of the MMPI-2. Pain 1993;54:153–158.
145. Helöe B, Helöe LA, Heiberg A. Relationship between sociomedical factors and TMJ-symptoms in Norwegians with myofascial pain-dysfunction syndrome. Community Dent Oral Epidemiol 1977;5:207–212.
146. Feinmann C, Harris M. Psychogenic facial pain: Presentation and treatment. Br Med J 1984;288:436–438.
147. Gerschman JA, Wright JL, Hall WD, Reade PC, Burrows GD, Holwill BJ. Comparisons of psychologic and social factors in patients with chronic oro-facial pain and dental phobic disorders. Aust Dent J 1987;32:331–335.
148. Tversky J, Reade PC, Gerschman JA, Holwill BJ, Wright J. Role of depressive illness in the outcome of treatment of temporomandibular joint pain-dysfunction syndrome. Oral Surg Oral Med Oral Pathol 1991;71:696–699.

149. Butterworth JC, Deardorff WW. Psychometric profiles of craniomandibular pain patients: Identifying specific subgroups. J Craniomand Pract 1987;5:225–232.
150. Dworkin SF, Massoth DL. Temporomandibular disorders and chronic pain: Disease or illness? J Prosthet Dent 1994;72:29–38.
151. Eversole LR, Stone CE, Matheson D, Kaplan H. Psychometric profiles and facial pain. Oral Surg Oral Med Oral Pathol 1985;60:269–274.
152. Bush FM, Whitehill M, Martelli MF. Pain assessment in temporomandibular disorders. J Craniomand Pract 1989;7:137–143.
153. Millstein-Prentky S, Olson RE. Predictability of treatment outcome in patients with myofascial pain-dysfunction (MPD) syndrome. J Dent Res 1979;58:1341–1346.
154. Schwartz RA, Greene CS, Laskin DM. Personality characteristics of patients with myofascial pain-dysfunction (MPD) syndrome unresponsive to conventional therapy. J Dent Res 1979;58:1435–1439.
155. Gerke DC, Richards LC, Goss AN. Discriminant function analysis of clinical and psychologic variables in temporomandibular joint pain dysfunction. Aust Dent J 1989;34:44–48.
156. McCreary CP, Clark GT, Merril RL, Flack V, Oakley ME. Psychologic distress and diagnostic subgroups of temporomandibular disorder patients. Pain 1991;44:29–34.
157. LeResche L, Dworkin SF, Wilson L, Ehrlich KJ. Effect of temporomandibular disorder pain duration on facial expressions and verbal report of pain. Pain 1992; 51:289–295.
158. McCreary CP, Clark GT, Oakley ME, Flack V. Predicting response to treatment for temporomandibular disorders. J Craniomandib Disord Facial Oral Pain 1992; 6:161–170.
159. Bush FM, Harkins SW, Harrington WG, Price DD. Analysis of gender effects on pain perception and symptom presentation in temporomandibular pain. Pain 1993;53:73–80.
160. Flor H, Behle DJ, Birbaumer N. Assessment of pain-related cognitions in chronic pain patients. Behav Res Ther 1993;31:63–73.
161. Oakley ME, McCreary CP, Flack VF, Clark GT. Screening for psychologic problems in temporomandibular disorder patients. J Orofacial Pain 1993;7:143–149.
162. Parker MW, Holmes EK, Terezhalmy GT. Personality characteristics of patients with temporomandibular disorders: Diagnostic and therapeutic implications. J Orofacial Pain 1993;7:337–344.
163. Schulte JK, Anderson GC, Hathaway KM, Will TE. Psychometric profiles and related pain characteristics of temporomandibular disorder patients. J Orofacial Pain 1993;7:247–253.
164. Gross SM, Vacchiano RB. Personality correlates of patients with temporomandibular joint dysfunction. J Prosthet Dent 1973;30:326–329.
165. Molin C, Schalling D, Edman G. Psychologic studies of patients with mandibular pain-dysfunction syndrome: I. Personality traits in patients and controls. Sven Tandläk Tidskr 1973;66:1–13.
166. Speculand B, Goss AN, Spence ND, Pilowsky I. Intractable facial pain and illness behavior. Pain 1981; 11:213–219.
167. Marbach JJ, Lennon MC, Dohrenwend BP. Candidate risk factors for temporomandibular pain and dysfunction syndrome: Psychosocial, health behavior, physical illness and injury. Pain 1988;34:139–151.
168. Stockstill JW, Callahan CD. Personality hardiness, anxiety, and depression as constructs of interest in the study of temporomandibular disorders. J Craniomandib Disord Facial Oral Pain 1991;5:129–134.
169. Solberg WK, Flint RT, Brantner JP. Temporomandibular joint pain and dysfunction: A clinical study of emotional and occlusal components. J Prosthet Dent 1972;28: 412–422.
170. Marbach JJ, Lipton JA, Lund PB, Delahanty F, Blank RT. Facial pains and anxiety levels: Considerations for treatment. J Prosthet Dent 1978;40:434–437.
171. Marbach JJ, Lund P. Depression, anhedonia and anxiety in temporomandibular joint and other facial pain syndromes. Pain 1981;11:73–84.
172. Moss RA, Adams HE. The assessment of personality, anxiety and depression in mandibular pain dysfunction subjects. J Oral Rehabil 1984;11:233–235.
173. Fricton JR, Nelson A, Monsein M. IMPATH: Microcomputer assessment of behavioral and psychosocial factors in craniomandibular disorders. J Craniomand Pract 1987;5:372–381.
174. McKinney MW, Lundeen TF, Turner SP, Levitt SR. Chronic TM disorder and non-TM disorder pain: A comparison of behavioral and psychological characteristics. J Craniomand Pract 1990;8:40–46.
175. Love AW, Peck CL. The MMPI and psychological factors in chronic low back pain: A review. Pain 1987;28: 1–12.
176. Prokop CK. Chronic pain. In: Greene RL (ed). The MMPI: Use With Specific Populations. Philadelphia: Grune and Stratton, 1988:22–49.
177. Mechanic D. The concept of illness behavior. J Chronic Dis 1962;15:189–194.
178. Zborowski M. Cultural components in responses to pain. J Soc Iss 1952;8:16–30.
179. Pilowsky I. Abnormal illness behavior. Br J Med Psychol 1969;42:347–351.
180. Pilowsky I. Pain as abnormal illness behavior. J Human Stress 1978;4:22–27.
181. Parsons T. Social structure and personality. London: Collier MacMillan, 1964.
182. Dworkin SF. Illness behavior and dysfunction: Review of concepts and application to chronic pain. Can J Physiol Pharmacol 1991;69:662–671.
183. Gerschman JA, Burrows GD, Reade PC. Chronic orofacial pain. In: Bonica JJ (ed). Advances in Pain Research and Therapy, vol 3. New York: Raven Press, 1979.
184. Speculand B, Goss AN, Hughes A, Spence ND, Pilowsky I. Temporo–mandibular joint dysfunction: Pain and illness behavior. Pain 1983;17:139–150.
185. Keefe FJ, Wilkins RH, Cook W. Direct observation of pain behavior in low back pain patients during physical examination. Pain 1984;20:59–68.
186. Pilowsky I, Spence ND. Pain and illness behavior: A comparative study. J Psychosom Res 1976;20:131–134.
187. Pilowsky I, Spence ND. Manual for the Illness Behavior Questionnaire (IBQ), ed 2. Adelaide: University of Adelaide, 1983.
188. Pilowsky I, Chapman CR, Bonica JJ. Pain, depression, and illness behavior in a pain clinic population. Pain 1977;4:183–192.
189. Chapman CR, Sola AE, Bonico JJ. Illness behavior and depression compared in pain center and private practice patients. Pain 1979;6:1–7.
190. Skevington SM. Activities as indexes of illness behavior in chronic pain. Pain 1983;15:295–307.

191. Main CJ, Waddell G. Psychometric construction and validity of the Pilowsky Illness Behavior Questionnaire in British patients with chronic low back pain. Pain 1987;28:13–25.
192. Pilowsky I, Smith QP, Katsikitis M. Illness behavior and general practice utilisation: A prospective study. J Psychosom Res 1987;31:177–183.
193. Byrne D, Whyte H, Lance G. A typology of responses to illness in survivors of myocardial infarction. Int J Psychiatry Med 1978;9:133–145.
194. Pilowsky I, Murrell TG, Gordon A. The development of a screening method for abnormal illness behavior. J Psychosom Res 1979;23:203–207.
195. Demjen S, Bakal D. Illness behavior and chronic headache. Pain 1981;10:221–229.
196. Smith DP, Pilling LF, Pearson JS, Rushton JG, Goldstein NP, Gibilisco JA. A psychiatric study of atypical facial pain. Can Med Assoc J 1969;100:286–291.
197. Pilowsky I, Spence ND. Patterns of illness behavior in patients with intractable pain. J Psychosom Res 1975; 19:279–287.
198. Keefe FJ, Crisson JE, Maltbie A, Bradley L, Gil KM. Illness behavior as a predictor of pain and overt behavior patterns in chronic low back pain patients. J Psychosom Res 1986;30:543–551.
199. Waddell G, Pilowsky I, Bond MR. Clinical assessment and interpretation of abnormal illness behavior in low back pain. Pain 1989;39:41–53.
200. Wichmann E, Nilges P, Gerbershagen HU, Gamber J, Scheifling I. The illness behavior questionnaire, psychometric properties and validity of a German version [abstract 644]. Pain 1990;5(special issue):336.
201. Sternbach RA. The Psychology of Pain, ed 2. New York: Raven Press, 1986.
202. Bates MS, Edwards WT, Anderson KO. Ethnocultural influences on variation in chronic pain perception. Pain 1993;52: 101–112.
203. Craig KD, Wyckoff MG. Cultural factors in chronic pain management. In: Burrows G, Elton D, Stanley R (eds). Handbook of Chronic Pain Management. Amsterdam: Elsevier Science, 1987.
204. Zola I. Culture and symptoms—An analysis of patients presenting complaints. Am Sociol Rev 1966;31: 615–630.
205. Weisenberg M, Keindler M, Schachat R. Pain: Anxiety and attitudes in black, white, and Puerto Rican patients. Psychosom Med 1975;37:123–135.
206. Garron D, Leavitt F. Demographic and affective covariates of pain. Psychosom Med 1979;41:525–534.
207. Flannery R, So SJ, McGovern P. Ethnicity as a factor in the expression of pain. Psychosomatics 1981;22:39–50.
208. Lipton JA, Marbach JJ. Ethnicity and the pain experience. Soc Sci Med 1984;19:1279–1298.
209. Ecandela JA. Social science and the study of pain since Zborowski: A need for a new agenda. Soc Sci Med 1993;36:783–791.
210. Kerns RD, Turk DC, Rudy TE. The West Haven–Yale Multidimensional Pain Inventory (WHYMPI). Pain 1985;23:345–356.
211. Kerns RD, Jacob MC. Assessment of the psychosocial context of the experience of chronic pain. In: Turk DC, Melzack R (eds). Handbook of Pain Assessment. New York: Guilford Press, 1992:235–253.
212. Bates MS. Ethnicity and pain: A biocultural model. Soc Sci Med 1987;24:47–50.
213. Festinger LA. A theory of social comparison processes. Hum Relat 1954;7:117–140.
214. Remick RA, Blasberg B. Psychiatric aspects of atypical facial pain. J Can Dent Assoc 1985;51:913–916.
215. Hampf G. Dilemma in treatment of patients suffering from orofacial dysaesthesia. Int J Oral Maxillofac Surg 1987;16:397–401.
216. Rome HP, Harness DM, Kaplan HJ. Psychologic and behavioral aspects of chronic facial pain. In: Jacobson AL, Donlon WC (eds). Headache and Facial Pain. Diagnosis and Management. New York: Raven Press, 1990.
217. Biondi M, Picardi A. Temporomandibular joint pain dysfunction syndrome and bruxism: Etiopathogenesis and treatment from a psychosomatic integrative viewpoint. Psychother Psychosom 1993;59:84–98.
218. Milliner EK. Biopsychosocial dentistry: The interface with psychiatric assessment. J Orofacial Pain 1994; 8:241–242.
219. Levitt SR, McKinney MW. Appropriate use of predictive values in clinical decision making and evaluating diagnostic tests for TMD. J Orofacial Pain 1994;8: 298–308.
220. Heloë B, Heiberg A, Krogstad BS. A multiprofessional study of patients with myofascial pain-dysfunction syndrome. I. Acta Odontol Scand 1980;38:109–117.
221. Schnurr RF, Brooke RI, Rollman GB. Psychosocial correlates of temporomandibular joint pain and dysfunction. Pain 1990;42:153–165.
222. Raphael KG, Marbach JJ. A year of chronic TMDS: Evaluating patients' pain patterns. J Am Dent Assoc 1992;123:53–58.

Resumen

Dolor temporomandibular y disfunción: Revisión crítica de la naturaleza del dolor y su evaluación

El desorden de disfunción-dolor temporomandibular es una forma común de dolor crónico que afecta la cabeza, la cara y la mandíbula. Los síntomas característicos de este desorden incluyen el dolor y el deterioro de la función masticatoria, y el despliegue frecuente de síntomas, que varían entre los dolores de cabeza, cuello, oídos y ojos; hasta odontalgias atípicas, síntomas en la garganta y cambios oclusales. Se reconoce que el dolor es una experiencia compleja, multifactorial que incluye no solo las dimensiones sensoriales, pero también los factores afectivos y cognoscitivos. Las recomendaciones recientes consideran el desorden de disfunción-dolor temporomandibular como un desorden de doble eje con dimensiones psicológicas y físicas, pero muy pocas investigaciones han incorporado las medidas de las características del dolor multidimensional en la evaluación del desorden de disfunción-dolor temporomandibular. Este artículo es una revisión de la literatura sobre los factores psicofisiológicos que contribuyen al desorden de disfunción-dolor temporomandibular y sus limitaciones. También se dan recomendaciones para investigaciones futuras.

Zusammenfassung

Myoarthropathien des Kausystems und Schmerzen: Eine kritische Übersicht über die Natur der Schmerzen und ihre Beurteilung

Myoarthropathie-Schmerzen des Kausystems sind eine häufige Form von Schmerzen im Bereich des Kopfes, des Gesichts und der Kiefer. Die verschiedenen Symptome dieser Störung beinhalten Schmerzen und Behinderung der Kaufunktion und häufig eine ganze Reihe von weiteren Symptomen, angefangen von Schmerzen im Kopf über solche in Nacken, Ohren und Augen bis hin zu atypischen Zahnschmerzen, Halssymptomen und okklusalen Veränderungen. Es ist bekannt, dass Schmerz eine komplexe, multifaktorielle Erfahrung ist, die nicht nur die sensorische Dimension, sondern auch affektive und kognitive Faktoren umfasst. Aktuelle Empfehlungen betrachten Myoarthropathie-Schmerzen des Kausystems als Störung mit zwei Achsen, eine Störung mit physikalischer und psychologischer Dimension, aber wenige Forschungsarbeiten haben Messungen dieser multidimensionalen Schmerzcharakteristik in die Untersuchung der Myoarthropathien des Kausystems einbezogen. Dieser Artikel bietet einen Überblick über die Literatur zu den psychophysiologischen Faktoren, die zu den Myoarthropathie-Schmerzen des Kausystems beitragen, und deren Grenzen. Es werden Empfehlungen für zukünftige Forschungsarbeiten abgegeben.