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Cognitive Factors and Persistent Pain: A Glimpse into Pandora's Box[1]

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Conventional models of persistent pain have tended to be dichotomous in nature, with pain viewed as either physically or psychologically based. Inadequacies inherent in both of these views have resulted in alterative conceptualizations that focus on the integration of biomedical with cognitive, affective, and behavioral factors. During the past decade there has been a proliferation of research designed to examine the relative contributions of individuals' attitudes, beliefs, appraisals, self-perceptions, and coping strategies to the perception, experience, and response to noxious sensations as well as treatment, and how these are modified as a result of treatment. In this paper a cognitive-behavioral conceptualization of persistent pain is described and contrasted with sensory, psychogenic, motivational, and operant conditioning models. A number of cognitive assessment procedures and recent research on the role of cognitive schemata, cognitive processes, and ongoing cognition in chronic pain are briefly summarized. The central importance of negative cognition — "catastrophizing" — is emphasized. Once pandora's cognitive box has been opened, a range of important issues must be addressed or one may be consumed by unbridled enthusiasm for the development of instruments and correlational research. Several caveats regarding current research on cognitive mediators are raised, namely, confounds among the cognitive measures that have proliferated and between cognitive measures and measures of mood states, generalizability of results based on pain clinic samples, and adherence to "patient uniformity myths."

KEY WORDS: cognitive schema; cognitive processes; catastrophizing; self-efficacy; coping; tailoring treatment.

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PAIN: THE MAGNITUDE OF THE PROBLEM

Pain is a major health problem in American society that affects millions of people and costs society billions of dollars in health care and lost productivity. With such astronomical figures, it is all too easy to lose sight of the incalculable human suffering accompanying chronic pain for both the individual and his or her family. The amount of attention devoted to pain has been disproportionately small given the magnitude of the problem. One difficulty with pain is that it is a symptom associated with many diseases and syndromes, and may result from diverse sources of pathology or trauma. Persistent and recurrent episodes of acute pain pose particularly frustrating problems in health care despite great advances in biomedical knowledge.

UNDERSTANDING PERSISTENT PAIN AND DISABILITY: ALTERNATIVE CONCEPTUALIZATIONS

The traditional biomedical view of pain dates back several hundred years and is based on a simple linear view that assumes a close correspondence between symptom perception and actual biological state; as the physical pathology increases, its symptoms become increasingly diagnostic of the relevant underlying pathology and distressing to the patient. Thus, the extent of pain severity is presumed to be directly proportionate to the amount of tissue pathology.

There are several perplexing features of persistent pain complaints that do not fit the biomedical model. For example, (a) patients with objectively determined equivalent degrees and types of tissue pathology vary widely in their reports of pain severity; (b) asymptomatic individuals often reveal objective radiographic evidence of structural abnormalities; (c) conversely, patients with minimal objective physical pathology often complain of intense pain; (d) surgical procedures designed to inhibit pain by severing neurological pathways may fail to alleviate pain; and (e) patients with objectively the same tissue pathology and treated with the same intervention respond in disparate ways. These paradoxes have resulted in a search for alternative physiological models, each of which have also proved to be inadequate to explain the complex phenomenon of pain (see Melzack & Wall, 1983, for a review).

Psychogenic View. As is frequently the case in medicine, when physical explanations prove unsatisfactory, psychological alternatives are entertained. Several variants of psychogenic etiological models have been espoused. For example, a model of a "pain-prone" personality originally proposed by Engel (1959) and expanded by Blumer and Heilbronn (1982) suggests that persistent pain complaints occur in individuals who are *predisposed* to experience pain because of family history and specific psychological characteristics. The American Psychiatric Association (1987) has established a psychiatric diagnosis, Somatoform Pain Disorder, that is based largely on the absence of specific physical pathology or other psychiatric disorders in the presence of reports of pain. These psychogenic views are posed as alternatives to physiological models.

Motivational View. A variation of the dichotomous organic vs. psychogenic views is a conceptualization that is ascribed to by many third-party payers. They suggest that if there is insufficient physical pathology to justify the report of pain, the complaint is the result of symptom exaggeration or outright malingering. The assumption is that reports of pain without adequate biomedical evidence are motivated by financial gain.

Each of the views described above is based on a fundamental assumption, namely, a cause-effect relationship from pathology to pain report. Current clinical tests and diagnostic imaging procedures, however, often offer few clues to the precise source of pain. For example, the discriminative power of common physical examination signs of pathology have been questioned. Rowe (1969) reported that the prevalence of leg length differences, increased lumbosacral angle, spondylolisthesis, transitional lumbosacral vertebra, and spina bifida occulta in back pain patients were not significantly different from those of a control group.

The clinical significance of identifiable structural abnormalities has also been challenged by the results of a number of studies. For example, a number of studies have found that spinal radiographic abnormalities based on plain X-rays (Deyo, 1986; Nachemson, 1976), CAT scans (Wiesel et al., 1984), and magnetic resonance imaging (MRI; Boden, Davis, Dina, Patrones, & Wiesel, 1990) believed to be associated with pain can be found in asymptomatic patients and, therefore, cannot be used to validate the legitimacy of pain reports.

Operant Conditioning View. The operant conditioning model originally proposed by Fordyce (1976) stands in marked contrast to the sensory model of pain. The operant conditioning model proposes that when an individual is exposed to a stimulus that causes tissue damage, the immediate response is withdrawal and attempts to escape from the noxious sensations. This may be accomplished by avoidance of activity believed to cause or exacerbate pain, seeking help to reduce the symptoms, and so forth. These behaviors are observable and, consequently, subject to the principles of operant conditioning. The operant conditioning model does not concern itself with pain, an internal subjective experience, but rather with overt manifestations of pain and suffering — "pain behaviors" — such as limping, moaning, and avoiding activity. Positive reinforcers such as attention by spouse, health care providers, and financial compensation, as well negative reinforcement by avoidance of undesirable or feared activities may serve to maintain the pain behaviors even in the absence of nociception (processing of stimuli that are defined as related to the stimulation of specific receptors and capable of being experienced as pain). In this way, reflexive respondent behaviors that occur during an acute injury may be maintained by reinforcement after the injury has resolved.

Several studies have provided evidence that supports the underlying assumptions of the operant model. For example, Cairns and Pasino (1977) and Doleys, Crocker, and Patton (1982) demonstrated that pain behaviors and "well-behaviors" (e.g., activity) could be decreased by verbal reinforcement. Block, Kremer, and Gaylor (1980) demonstrated that pain patients reported differential levels of pain in an experimental situation depending on whether they knew that they were being observed by their spouses or by ward clerks. The operant model has also generated what has proven to be an effective treatment (for a review see Keefe & Williams, 1989). The operant model has, however, received some criticism for its exclusive reliance on motor behavior (e.g., Turk & Flor, 1987) and concerns about generalization and maintenance (Turk & Flor, 1987) as well as for problems with the acceptance of treatment by patients and patient dissatisfaction (Kotarba, 1983; Turk & Rudy, 1990).

Gate Control Model. Multidimensional views of chronic pain differentiate nociception from pain, which is a complex perceptual phenomenon (Melzack, 1986). Based on this view, Melzack and his colleagues (Melzack & Casey, 1968; Melzack & Wall, 1965) proposed the gate control theory of pain. The conceptual model of the gate control theory emphasizes the modulation of pain by peripheral as well as central nervous system processes and thus provides a physiological basis for the role of psychological processes in chronic pain. Melzack and Casey (1968) differentiate three systems related to the processing of nociceptive stimulation — motivational-affective, cognitive-evaluative, and sensory-discriminative dimensions — all thought to contribute to the experience of pain.

PHENOMENOLOGY OF CHRONIC PAIN

Chronic pain, by definition, extends over long periods of time. The average duration of the pain of many pain patients seen in pain clinics exceeds 7 years. Thus, even when the psychiatric evaluation identifies emotional problems, it is erroneous to infer that these are causal, as suggested by the psychogenic models. Psychological problems in chronic pain patients may be caused by a variety of factors, including iatrogenic complications, overuse of tranquilizers and narcotic medication, work disability, financial difficulties, prolonged litigation, disruption of usual activities, inadequate social support, and sleep disturbance.

Moreover, the experience of "medical limbo" — that is, having a painful condition that eludes diagnosis and carries the implication of either psychiatric causation or of an undiagnosed life-threatening disease — is itself the source of significant stress and can initiate psychological distress or aggravate a premorbid psychiatric condition. Living with chronic pain requires considerable emotional resilience and tends to deplete one's emotional reserve, and taxes the capacity of family, friends, and coworkers to provide support.

Biomedical factors, in the majority of cases, appear to instigate the initial report of pain. Over time, however, psychosocial and behavioral factors may serve to exacerbate and maintain levels of pain and, subsequently, disability. Following from this view, pain that persists over time should not be viewed as either solely physical or psychological, but rather as an experience maintained by an interdependent set of biomedical, psychosocial and behavioral factors.

COGNITIVE-BEHAVIORAL PERSPECTIVE

A comprehensive model of chronic pain needs to incorporate the mutual interrelationships of physiological, psychosocial, and behavioral factors and the changes among these relationships over time (Flor, Birbaumer, & Turk, 1990; Turk, Meichenbaum, & Genest, 1983). From this perspective, people with chronic pain, as is true for all individuals, are viewed as active processors of information. They have negative expectations about their own ability and responsibility to exert any control over their pain. Moreover, they often view themselves as helpless. Such negative, maladaptive appraisals about their situation and their personal efficacy may reinforce the experience of demoralization, inactivity, and overreaction to nociceptive stimulation. Such cognitive appraisals and expectations are postulated as having an effect on behavior, leading to reduced effort and activity and increased psychological distress.

The specific types of cognitive experiences relevant to pain perception are thought to include focus of attention, beliefs, attributions, expectations, coping self-statements, images, and problem-solving cognitions (Turk et al., 1983). The cognitive–behavioral perspective suggests that behavior and emotions are influenced by interpretations of events, rather than solely by characteristics of the event itself. Thus, pain, when interpreted as signifying ongoing tissue damage or life-threatening illness, is likely to produce considerably more suffering and behavioral dysfunction than if it is viewed as being the result of a minor injury, although the amount of nociceptive input in the two cases may be equivalent.

Patients' interpretations of nociception and their resources can have both direct and indirect effects on physiological processes that may maintain and exacerbate pain. Cognitive interpretations may have a direct effect on physiology by increasing autonomic and sympathetic nervous system arousal and, potentially, muscle spasm (Bandura, Taylor, Williams, Meffort, & Barchas, 1985; Flor, Turk, & Birbaumer, 1985), as well as an indirect effect by reducing physical activity, thereby leading to reduced muscle flexibility, strength, and tone.

Cognitive interpretations also will affect how patients present symptoms to significant others, including health care providers. Overt communications of pain, suffering, and distress will enlist responses that may reinforce the pain behaviors and impressions about the seriousness, severity, and uncontrollability of the pain. That is, complaints of pain may lead physicians to prescribe more potent medications, order additional diagnostic tests, and, in some cases, perform surgery. Family members may express sympathy, excuse the patient from usual responsibilities, and encourage passivity. It should be obvious that the cognitive–behavioral perspective integrates the operant conditioning emphasis on external reinforcement contingencies and the respondent view of learned fear and avoidance within the framework of an information processing perspective.

COGNITIVE VARIABLES AS MEDIATORS OF PAIN PERCEPTION AND RESPONSE

If one accepts that chronic pain is a complex, subjective phenomenon that is uniquely experienced by each individual, then knowledge about idiosyncratic beliefs, appraisals, expectancies, and coping repertoires become critical for optimal treatment planning and for accurately evaluating treatment outcome. Cognitive activity of chronic pain patients may contribute to the exacerbation, attenuation, or maintenance of pain, pain behavior, affective distress, and dysfunctional adjustment to chronic pain (Turk & Rudy, 1986).

In order to facilitate adaptive coping in chronic pain patients, cognitive–behavioral interventions have been developed that attempt to (a) alter cognitions that may be associated with dysfunctional adjustment to chronic pain, such as perceived lack of self-efficacy in controlling pain, distortion in the interpretations of pain-related events, disease conviction, somatic preoccupation, and catastrophizing; (b) enhance patients' use of specific cognitive coping strategies; and (c) enhance patients' confidence in their ability to cope.

Three interdependent constructs have been described: cognitive structures, cognitive processes, and conscious cognitive products (Segal & Shaw, 1988). Separating these three constructs can be somewhat arbitrary as there is considerable overlap. We have chosen to discuss them separately but this may be more for ease of discussion rather than clear delineation between each of them. As applied to coping with pain, these constructs include (1) cognitive schema — general beliefs, appraisals, and expectations about pain; (2) cognitive processes — mental processes involved in pain control attempts; and (3) specific cognitive content about patients' circumstances and their attempts to cope with pain. Recently, a plethora of studies have appeared that are designed to assess cognitive structures (e.g., Jensen, Karoly, & Huger, 1987; Shutty & DeGood, 1990), cognitive processes (e.g., Gil, Williams, Keefe, & Beckham, 1990; Lefebvre, 1981), and the cognitive content (e.g., Rosensteil & Keefe, 1983) and to relate these constructs to treatment response. We will review the results and implications of a number of these studies in the remainder of this paper.

Cognitive Schema — Beliefs, Appraisals, and Expectations

Patients' beliefs, appraisals, and expectancies about their pain, their ability to cope, their social supports, their disorder, the medicolegal system, the health care system, and their employers are all important as they may facilitate or disrupt the patient's sense of control and ability to manage pain. These factors also influence patients' investment in treatment, acceptance of responsibility, perceptions of disability, adherence to treatment recommendations, support from significant others, expectancies for treatment, and acceptance of treatment rationale (Slater, Hall, Atkinson, & Garfin, 1991).

Individuals respond to medical conditions in part based on their subjective representations of illness and symptoms. When confronted with new stimuli, the individual engages in a "meaning analysis" (Cioffi, 1991) that is guided by the schemata that best match the attributes of the stimulus. When physical sensations are perceived, they are interpreted, labeled, and acted upon. People organize information into causal units and according to prior theories, beliefs, and expectations (Nisbett & Ross, 1980). We respond not only to "objective" reality, but to our private, subjective, idiosyncratic beliefs, fears, competencies, and goals (Leventhal, Meyer, & Nerenz, 1980).

Once a label has been assigned to a physical sensation (e.g., pain), other belief structures follow. People build fairly elaborate representations of their physical states, and these representations provide the basis for coping and plans of actions (Turk, Rudy, & Salovey, 1986). These representations are used to construct causal, covariational, and consequential information from their symptoms. These inferences in turn affect what people do in response to symptoms. For example, a cognitive schema suggesting that one has a very serious condition, that impairment is a necessary aspect of pain, that activity despite pain is dangerous, and that pain is an acceptable excuse for neglecting responsibilities will result in maladaptive responses (Schwartz, DeGood, & Shutty, 1985; Williams & Thorn, 1989). Similarly, if patients believe they have a serious condition that is quite fragile and a high risk for reinjury or exacerbation of pain, they may be afraid to engage in physical activities (Philips, 1987). Through a process of stimulus generalization, patients may avoid more and more activities, become more physically deconditioned, and more disabled.

Schwartz et al. (1985) suggest that the psychological dimension that appears most relevant to treatment planning is the patient's stability and willingness to understand and accept a chronic model of pain, along with its implications for rehabilitation. Consistent with this model, Herman and Baptiste (1981) noted that successes and failures in their program, defined according to several functional criteria, could be distinguished most prominently on the basis of changed vs. unchanged thought patterns related to the prospect of living useful lives despite pain. Clearly, it appears essential for patients with chronic pain to develop adaptive beliefs about the relation between pain and impairment, and to deemphasize the belief that pain per se can lead to dysfunction.

There are many laboratory studies demonstrating that perceived controllability of aversive stimulation reduces its impact considerably (for a review see Thompson, 1981). In chronic pain patients the perceived lack of personal control is likely related to ongoing but unsuccessful efforts to control pain. Furthermore, uncontrollability augments the perception of pain intensity (Miller, 1981).

Self-Efficacy. Pain patients' beliefs about their capabilities appear to be predictive of their behavior. A central construct in the cognitive–behavioral model of chronic pain is self-efficacy (Bandura, 1977). A self-efficacy expectation is defined as a personal conviction that one can successfully execute a course of action to produce a certain outcome. Bandura suggested that given sufficient motivation to engage in a behavior, it is an individual's self-efficacy beliefs that determine the choice of activities that the individual will initiate, the amount of effort that will be expended, and how long the individual will persist in the face of obstacles and aversive experiences. From this perspective, the occurrence of coping behaviors is conceptualized as being mediated by the individual's beliefs that situational demands do not exceed coping resources.

Self-efficacy beliefs are influenced by four sources of information: performance or enactment experiences, vicarious experiences, verbal persuasion (or social persuasion), and emotional or physiological arousal (Bandura, 1977). Mastery experiences gained through performance accomplishments are hypothesized to have the greatest impact on establishing and strengthening expectancies because they provide the most information about actual capabilities.

For chronic pain patients, physiological states are a particularly important source of self-efficacy information. In evaluating their physiological states, people often make inferences about their personal capabilities. Although arousal and tension are perceived as common antecedents of debilitating performance, activities such as exercise, which demand strength or stamina, can result in certain amounts of pain and fatigue that may be interpreted as signs of personal inefficacy. In order to boost efficacy cognitions in these situations, interpretations of physiological states need to be altered or reinterpreted as a natural, progressive stage in achieving an increase in fitness. It becomes important then for physical therapists to educate patients about the relationship of physiological responses like muscle fatigue. That is, they need to inform patients that these physiological responses are inherent to exercise and such signs are to be expected rather than feared. Successful vs. unsuccessful physical therapy may be distinguished by the presence vs. the absence of changes in perceived self-efficacy in conjunction with physical improvements in tolerance, strength, and endurance.

Support for the importance of self-efficacy as specifically related to pain has been demonstrated in laboratory studies (e.g., Bandura, O'Leary, Taylor, Gauthier, & Gossard, 1987; Litt, 1988), with headache patients (e.g., Holroyd et al., 1984), temporomandibular pain disorders (Hijzen, Slangen, & Van Houweligew, 1986), back pain (Council, Ahern, Follick, & Cline, 1988; Dolce, Crocker & Doleys, 1986), arthritis (Lorig, Chastain, Ung, Shoor, & Holman, 1989), and heterogeneous clinical populations (e.g., Dolce et al., 1986; Kores, Murphy, Rosenthal, Elias, & North, 1990). For a review see Turk and Rudy (1991). Several illustrative studies will be noted as they demonstrate the important role of self-efficacy in chronic pain.

In a set of studies, Schmidt (1985a, 1985b) demonstrated that low-back-pain patients demonstrated poor behavioral persistence on various exercise tasks and that their performances on these tasks were independent of any physical parameters or actual self-reports of pain. These patients appeared to have a negative view of their abilities and expected increased pain if they performed physical exercises. Thus, the rationale for their avoidance of exercise was not the presence of pain but their *anticipation* of heightened pain. Schmidt postulated that these negative perceptions of their capabilities for physical performance form a vicious circle, with the failure to perform activities reinforcing the perception of helplessness and incapacity.

Dolce et al. (1986) reported that beliefs regarding ability to exercise predicted improvement in work status and exercise levels 6–12 months after a physical restoration program. The interrelated role of fear avoidance and self-efficacy was illustrated in a study reported by Council et al. (1988). They found that actual physical performance of back pain patients was best predicted by self-efficacy ratings, which appeared to be determined by pain response expectancies. The authors interpreted these results as suggesting that daily pain experience determines pain response expectancies for specific movements. Pain response expectancies appear to influence performance and associated pain behavior through their effects on efficacy expectancies. These findings also indicate that pain response expectancies associated with specific movements are based on generalized expectancies drawn from daily experiences and suggest that chronic pain patients have well-established ideas of how much pain they will experience in different situations. These beliefs about the results of activity may influence patients' avoidance of certain activities for fear of the consequences, including the belief that they may become more functionally impaired (Philips, 1987).

Learned Helplessness. Learned helplessness refers to the belief that effective solutions are not available to eliminate or reduce the source of stress. In the extreme, this belief can lead to emotional, motivational, and cognitive deficits in coping with stressful situations. Several authors have suggested that the seemingly unpredictable and uncontrollable waxing and waning of physical symptomatology of rheumatoid arthritis (RA) patients and its impact on functional capabilities can induce learned helplessness, with associated behavioral and emotional consequences (Nicassio, Wallston, Callahan, Herbert, & Pincus, 1985). In the face of severe or worsening disease and the associated increasing discomfort and decreasing ability to engage in valued activities, RA patients who view themselves as helpless would be most likely to be depressed. Thus, it is not the impact of the illness itself that produces depression but rather the patient's interpretation of it as being uncontrollable.

A number of investigators (e.g., Flor & Turk, 1988; Nicassio et al., 1985; Smith, Peck, & Ward, 1990) have demonstrated that greater feelings of helplessness were significantly correlated with both psychological distress and physical disability. Flor and Turk (1988) also noted that perceived helplessness was predictive of reports of pain and the number of physician visits per year. In a longitudinal study, Stein, Wallston, Nicassio, and Castner (1988) reported that changes in helplessness over a period of 6 months were associated with changes in depression, pain severity, and global ratings of health status. Moreover, helplessness predicted depression, global heath ratings, psychological distress, and arthritis symptom severity up to 2 years later.

Cognitive Processes

Cognitive processes are integrally related to cognitive schemata. They are involved in the actual manipulation of information. It is through cognitive processes that individuals operate on information, select action plans, and execute responses. Cognitive processes are responsible for the transformation of new information and the modification of internal representations. When confronted with new information, individuals do not engage in an exhaustive search of all relevant information but may react to cues that are already contained in relevant cognitive schemata. Efficient processing of information relies on the use of preconceptions and automatic thoughts that occur without conscious awareness. These preconceptions influence what evidence is used in making inferences.

Automatic cognitive distortions can have important emotional and behavioral effects. There is a high incidence of depression among chronic pain patients. Several studies have demonstrated that cognitive distortions seem to be particularly important in mediating the pain–depression association. For example, Smith, Follick, Ahern, and Adams (1986) reported data suggesting that the level of cognitive distortion is reliably associated with the degree of disability reported by patients with chronic low back pain. Dufton (1989) reported that persons experiencing chronic pain had a tendency to make cognitive errors related to the emotional difficulties associated with living with pain, rather than the pain intensity alone, and those who made such errors were more depressed.

Lefebvre (1981) developed the Cognitive Errors Questionnaire (CEQ) to assess cognitive distortions (i.e., "catastrophizing," "overgeneralization," "personalization," and "selective abstraction") in chronic back pain patients. Lefebvre found that patients who reported engaging in high levels of cognitive distortion were more depressed than patients who did not. A number of studies have confirmed the link between depression and cognitive distortions (e.g., Keefe & Williams, 1990; Slater et al., 1991; Smith, Peck, Milano, & Ward, 1988).

The results of Lefebvre induced Ingram, Atkinson, Slater, Saccuzzo, and Garfin (1990) to examine whether subgroups of pain patients varied on the basis of cognitive patterns associated with depression. Ingram et al. (1990) found no differences among pain groups in demographics, pain intensity, duration, orthopedic diagnosis, or disease severity; however, patients experiencing depression reported more maladaptive automatic thoughts than did nondepressed subjects. These authors suggested customizing treatment to patterns of maladaptive thinking. They proposed that chronic pain patients who are depressed would be appropriate for cognitive–behavioral methods that focus on correcting cognitive distortions and on modifying negative automatic thinking with more adaptive positive thoughts. Alternatively, for patients who are not depressed, these results suggested that cognitive–behavioral approaches aimed primarily at identifying and correcting negative automatic thinking and cognitive distortions are less appropriate.

Gil et al. (1990) developed an Inventory of Negative Thoughts in Response to Pain (INTRP) that comprised three factors: negative self-statements, negative social cognitions, and self-blame. Respondents to the INTRP indicate how frequently they have each negative thought during a flare-up of pain. Higher scorers on negative self-statements and negative social cognitions reported more severe pain and psychological stress. Gil et al. suggested that patients who view flare-ups negatively may become inactive, withdraw from family and social relationships, and suffer heightened anxiety, depression, and preoccupation with physical symptoms.

In sum, cognitive processes characterized by negative thoughts appear to predict long-term adjustment to chronic pain, may mediate the relationship between disease severity and adjustment, and make a unique contribution to predicting adjustment. The increased interest in the processing of information by chronic pain patients has resulted in the development of a number of conceptually related instruments such as the CEQ and INTRP.

Cognitive Content

The specific thoughts and feelings that patients experience prior to exacerbations of pain and during an intense episode of pain, as well as following a pain episode, can greatly influence the experience of pain and subsequent episodes. Moreover, the methods patients use to control their emotional arousal and symptoms have been shown to be important predictors of both cognitive and behavioral responses (e.g., Flor & Turk, 1988; Reesor & Craig, 1988).

A number of recent studies have attempted to examine directly the ongoing thoughts and coping techniques used by chronic pain patients (e.g., Boston, Pearce, & Richardson, 1990; Flor & Turk, 1988; Vlaeyen et al., 1990). These studies all asked patients to indicate from a list what they found themselves thinking when they were experiencing pain. Some representative findings suggest that more positive thoughts correlate negatively with current pain distress and pain-related behavioral disruption (Ressor & Craig, 1988), scores on scales measuring helplessness and hopelessness factors are correlated positively with pain measures (Boston et al., 1990), and reporting more negative thoughts is associated with greater frequency of physician visits (Flor & Turk, 1988).

Cognitive and Behavioral Coping Strategies: Coping with the Coping Literature. The process by which individuals react to and actively manage the impact of pain in their lives is referred to as *coping.* Self-regulation of pain and pain's impact on them depends upon individuals' specific ways of dealing with pain, adjusting to pain, and reducing or minimizing pain and distress caused by pain, and, therefore, on their coping strategies. Coping is assumed to be manifested by spontaneously employed purposeful and intentional acts. Cognitive strategies are thought to act to alter the perception of pain intensity and one's ability to manage or tolerate pain and to continue everyday activities (Turk et al., 1983).

Several lines of research, including experimental laboratory studies of acute pain with normal volunteers and field studies with clinical patients suffering chronic pain, have indicated that catastrophizing (negative thoughts likely to increase distress) and adaptive coping strategies are important in reacting to pain. Two findings from laboratory studies are particularly important. Individuals who spontaneously utilize less catastrophizing self-statements and/or more adaptive coping strategies rate experimentally induced pain as less painful and tolerate painful stimuli longer (e.g., Heyneman et al., 1990; Spanos, Horton, & Chaves, 1975). In addition, in a myriad of studies it has been shown that if instructed in the use of adaptive coping strategies, the rating of intensity of pain decreases and tolerance of pain increases (see review by Fernandez & Turk, 1989). As we will see, the most important factor in poor coping appears to be the presence of catastrophizing rather than differences in adaptive coping strategies (e.g., Heyneman et al., 1990; Martin, Nathan, Milech, & Van Keppel, 1989).

Clinical studies with patients have reported parallel findings to the laboratory research. Rosensteil and Keefe (1983) found that cognitive coping and suppression (adaptive strategies) and helplessness and catastrophizing were predictive of adjustment. Similarly, Turner and Clancy (1986) found that adaptive strategies were related to less handicap in response to pain and that catastrophizing was related to greater disability in back pain patients. Generally, the strongest predictions were possible with measures of catastrophizing.

As noted earlier, Flor and Turk (1988) found in low-back-pain sufferers and arthritis patients that between 32% and 60% of the variance in pain and impairment, respectively, was accounted for by cognitive factors that the authors labeled catastrophizing, helplessness, coping, and resourcefulness. In both the low-back-pain and the arthritis groups, the cognitive variables of catastrophizing and adaptive coping had substantially more explanatory power that did disease variables.

The Coping Strategies Questionnaire (CSQ) was developed by Rosensteil and Keefe (1983) to measure the frequency that patients report using cognitive and behavioral coping strategies and the degree to which these strategies were perceived as effective. Rosensteil and Keefe found that reported frequency of using specific coping strategies was predictive of pain, functional status, and psychological distress after controlling for demographic and medical status variables. Those high on helplessness were significantly more depressed and anxious that those low on this strategy. Diverting attention and praying were associated with higher levels of pain and more functional impairment.

Parker et al. (1988) demonstrated that following a cognitive–behavioral intervention, RA patients were found to have significantly improved scores on the CSQ subscales that make up the pain control rational thinking (PCRT) factor. The results of that study demonstrated that cognitive–behavioral interventions can significantly increase RA patients' confidence that they can manage their pain.

To date, the large majority of studies using the CSQ have failed to consider the actual pain intensity of the patients completing the questionnaire. Since patients are asked about their typical coping strategies when they are in pain, it might be expected that current level of pain would influence the strategies selected. Using a Finnish translation of the CSQ, Estlander and Harkapaa (1989) found similar relationships between coping strategies and pain to those reported by Rosensteil and Keefe (1983) and Romano, Turner, Syrjala, and Levy (1987) but with some important qualifications. The level of pain (mild vs. severe) influenced the nature of the coping strategies employed regardless of the level of disability. Catastrophizing was reported significantly more often in severe pain than in mild pain regardless of the degree of disability. For patients with "not very severe pain," catastrophizing scores were significantly associated with higher levels of perceived disability.

Importance of Catastrophizing

Research findings suggest that cognitive methods might be more effective if they were aimed at reducing the frequency of using negative strategies rather than increasing the frequency of using positive strategies.

Chaves and Brown (1987) and Rosensteil and Keefe (1983) suggest that successful coping is a consequence of avoiding catastrophizing. This is further supported by Turner and Clancy (1986), who showed that during cognitive-behavioral treatment reductions in catastrophizing were significantly related to reductions in pain intensity and physical impairment.

It may be true that it is more important for patients to avoid or interrupt cognitions of feeling helpless than to engage in some form of more active coping. The most important factor in poor coping both in laboratory and clinical pain appears to be the presence of catastrophizing rather than differences in adaptive coping strategies (e.g., Estlander & Harkappa, 1989; Heyneman et al., 1990; Turk et al., 1983; Turner & Clancy, 1986).

COGNITIVE-BEHAVIORAL TREATMENT OF CHRONIC PAIN

Although cognitive-behavioral interventions have been found to be generally effective in reducing psychological distress in chronic pain patients (Bradley et al., 1987; Keefe et al., 1990; Kerns, Turk, Holzman, & Rudy, 1986; Moore & Chaney, 1985; Turner & Clancy, 1988), there has been little research identifying which cognitions change, and how these changes relate to treatment improvements or better adjustment to persistent pain.

Newton and Barbaree (1987) used a modified thought sampling procedure to evaluate the nature of patients' thoughts during and immediately following headache, both prior to and following treatment. Their results indicated significant changes in certain aspects of headache-related thinking in the treated groups compared to the control group. The treated patients demonstrated significant reductions in negative appraisals and significant increases in positive appraisals. Additionally, the treated patients reported experiencing significantly fewer headache days per week and lower intensity of pain. The subjects who reported the largest shift toward less negative appraisals also reported the greatest reduction in headache intensity.

These results indicate that a cognitive shift does occur following cognitive-behavioral therapy. Significant improvement in headache symptomatology accompanied cognitive shifts. These results also support the argument that changes in cognitive reactions to headache might underlie headache improvement following a variety of different treatments (Holroyd & Andrasik, 1982). The strongest evidence points toward a reduction in negative appraisal as representing the potential change mechanism. As noted above, a reduction in negative thinking rather than an increase in positive self-talk or use of coping strategies was associated with therapeutic improvement.

Mechanisms of Change

Several mechanisms may account for the influence of cognitive variables in pain perception and patients' responses to treatment. Some research has suggested that there is a direct effect of cognition on physiology. For example, Flor et al. (1985) reported that thinking about stress and pain resulted in increased muscular arousal at the specific site of pain for a subset of patients with low back pain. Bandura and his colleagues reported the direct effects of self-efficacy beliefs on stress hormones (Bandura et al., 1985) and levels of endogenous opioids — "endorphins" (Bandura et al., 1987).

Longitudinal studies of changes in coping strategies and adjustment to pain over time are needed to validate the cognitive hypothesis that the use of particular cognitive coping strategies will determine the development, maintenance, and exacerbation of psychological distress in chronic pain patients (Turk et al., 1983). Assessment of cognitive activity during headache, as examined by Newton and Barbaree (1987), addresses the important question: What cognitive processes mediate pain perception? If a cognitive mechanism does underline therapeutic improvement, then changes in cognitive measures should occur during the course of treatment. A number of studies reviewed earlier (e.g., Lorig et al., 1989; O'Leary, Shoon, Lorig, & Hohman, 1988; Parker et al., 1989) suggest that changes in various cognitive measures did correspond with changes in pain, dysphoric mood, and disability.

In considering the efficacy of biofeedback, Blanchard (1987) speculated that the maintenance of treatment effects endures in spite of almost universal cessation of regular home practice of biofeedback because the self-perpetuating cycle of chronic headache has been broken. The experience of headache serves as a stressor to cause, in part, a future headache. It may also serve to maintain improper analgesic medication consumption, the cessation of which can also lead to "rebound headache." By the end of treatment when the patient has experienced noticeable headache relief, it is as if the patient redefines himself or herself as someone able to cope with headaches. As a consequence, one source of stress is removed and the patient copes with recurrences more adaptively. Similarly, in considering the efficacy of biofeedback with back pain patients, Nouwen and Solinger (1979) concluded that ". . . simultaneous accomplishment of muscle tension reduction and lowering reported pain convinced patients that muscle tension, and subsequently pain, could be controlled. . . . As self-control could not be demonstrated in most patients, it seems plausible that feelings of self-control, rather than actual control of physiological functions or events is crucial for further reductions" (p. 110). Supporting empirical evidence for the hypothesized role of self-efficacy in biofeedback treatment has been reported by several investigators (e.g., Hijzen et al., 1986; Holroyd et al., 1984; Litt, 1988; Nouwen & Solinger, 1979). For example, in a study of migraines treated by cephalic vasomotor biofeedback, Gauthier, Cote, and Drolet (1985) found a significant association between increases in perceived self-efficacy and headache relief but no association between psychophysiological change and headache relief.

What are the mechanisms that account for the observed association between self-efficacy and behavioral outcome? Cioffi (1991) suggested at least four psychological processes could be responsible: (a) As perceived self-efficacy decreases anxiety and its concomitant physiological arousal, the person may approach the task with less potentially distressing physical information to begin with; (b) the efficacious person is able to willfully distract attention from potentially threatening physiological sensations; (c) the efficacious person perceives and is distressed by physical sensations but simply persists in the face of them (stoicism); and (d) physical sensations are neither ignored nor necessarily distressing but rather are relatively free to take on a broad distribution of meanings (change interpretation). These and other cognitive mechanisms need to be examined in more depth.

Caveats

After glimpsing into Pandora's box of cognitive factors, some important qualifications need to be made when interpreting the results reviewed above.

Confounds of Cognitive Measures. A caveat is especially warranted when we consider the proliferation of cognitive measures that have been developed. There tends to be an implicit assumption that the various cognitive measures are relatively independent. Research needs to be conducted to determine if in fact the plethora of cognitive measures being developed are truly measuring unique cognitive constructs.

Little attention has been given to the overlap of these measures and whether they are measuring different constructs or some common latent construct (e.g., negative mood). For example, all three scales and summary ratings from the INTRP (Gil et al., 1990) were significantly correlated with the Catastrophizing scale from the CSQ (Rosensteil & Keefe, 1983), described above. Moreover, the negative thoughts from the INTRP were also all significantly correlated with the Global Severity Index of psychological distress from the SCL-90R. Similarly, the negative self-statements identified by Boston et al. (1990) were all significantly correlated with state anxiety.

The problem of confounding of pain measures with anxiety has been described by Gross and Collins (1981).

Cognitive measures used in pain also may be significantly associated with measures of depression. For example, Sullivan and D'Eon (1990) reported a statistically significant association between measures of catastrophizing and depression. Additionally, when items reflective of symptoms of depression were removed from the Catastrophizing scale of the CSQ, none of the remaining subscales were significantly correlated with depression. In other words, it appears that the Catastrophizing scale of the CSQ is conceptually and operationally confounded with depression. Further, Sullivan and D'Eon suggest that depression might entirely explain the relationship between the use of coping strategies and disability. In sum, the issue of independence of cognitive measures and the confounds between cognitive measures and mood states needs greater attention.

Generalizability. There are a number of factors that limit the generalizations that can be drawn from treatment outcome studies that have used psychological modalities with chronic pain patients. Turk and Rudy (1990) have drawn attention to the uniqueness of the sample of patients referred to pain clinics. They also note that a substantial number of patients who are evaluated at such clinics never enter treatment either because of the exclusion criteria used by the investigators, or simply because patients chose not to enter treatment (e.g., no third-party coverage, inconvenient, unmotivated). Moreover, a subset of patients drop out of treatment or are dropped from treatment. Several epidemiological studies also have demonstrated higher levels of psychological distress for patients treated at pain clinics in contrast to patients recruited from primary care settings or newspaper advertisements.

Matching of Patients to Treatments. Chronic pain patients have tended to be treated as a homogeneous group either based on the medical diagnosis or as suffering from a generic "chronic pain syndrome" (Black, 1975), for whom the same treatment is prescribed. This approach to patients implicitly ascribes to a "pain patient uniformity myth" (Turk, 1990). This myth has been opposed by the Institute of Medicine (Osterweis, Kleinman, & Mechanic, 1987), which wrote, "Because of the considerable differences in types of pain and patients, it is inappropriate to speak of 'the' chronic pain patient as if there were only one type . . . which implies a homogeneity among conditions that are actually quite different" (pp. 12-13). Given the complexity of the phenomenon and the wide range of individual variation of patients, it is hardly surprising that generic treatments have not proven to be uniformly successful.

Reviews of the literature on the efficacy of coping strategies tend to be equivocal (e.g., Fernandez & Turk, 1989). However, it should be noted that many of these studies combined subjects with important individual differences, for example, in regard to catastrophizing. Thus, studies with equivocal effects may suffer from the patient uniformity myth in that subject heterogeneity adversely affects the statistical power of the tests used.

Within any given pain treatment setting, particular referral sources and treatment orientations may create perceptions of relative homogeneity among patients. However, the danger of generalizing from limited samples of patients in terms of treatment and research is that the specificity of diagnoses and treatment approaches for one setting or subgroup of patients will be applied uniformly to all chronic pain patients. The challenge to clinicians is to sort out the complex biomedical, psychosocial, and behavioral factors and to develop tailored treatment approaches that fit the idiosyncrasies of each patient (Turk, 1990).

Perhaps the more important and appropriate questions for treatment studies is not whether the treatment is successful but how successful the treatment is for which patients with what characteristics (Turk & Rudy, 1988). A reasonable strategy might be to maximize the commonalties (nonspecifics) for all patients, but simultaneously individualize treatments to specific physiological, psychosocial, and behavioral characteristics. The exclusive reliance on group effects may mask important subject-by-treatment interactions.

For example, Flor et al. (1985) reported that a subset of back pain patients demonstrated site-specific muscular response to psychological stress. Medical status variables did not predict which patients demonstrated the abnormal muscular response; however, depression did predict responders. Thus, not all back pain patients would be expected to respond to paraspinal EMG biofeedback designed to modify stress reactivity. In other words, it would seem to make little sense to offer site-specific biofeedback to the subset of patients who did not display baseline elevations in paraspinal EMG or stress-induced reactivity, except perhaps to increase self-efficacy expectations.

Another example of the importance of matching specific components of treatment to patient characteristics is revealed by examining the treatment outcome study reported by Moore and Chaney (1985). The authors examined the additive contribution of spouse inclusion in a group cognitive–behavioral intervention. Both treatment groups benefitted significantly from the treatment; however, there were no between group differences. The authors concluded from these results that spouses did not contribute to treatment effects. Flor, Turk, and Rudy (1989), however, demonstrated that quality of the marital relationship mediated the effects of spouse reinforcement of pain behaviors. Thus, some consideration of the quality of the marriage should be considered when interpreting the benefits of spouse improvement.

Collapsing treatment responders and nonresponders together in between-group designs might account for some of the confusion in the treatment outcome literature. As in other areas of treatment research, there has been a tendency for investigators into the impact of cognitive factors on chronic pain to rely on group means and treat patients as a homogeneous group when performing statistical analyses. However, a number of studies have demonstrated that there are distinct subgroups of chronic pain patients (see Turk, 1990, for a review). It is quite possible that subgroups of patients make use of quite different coping strategies, with different results. Moreover, these subgroups might respond differently to treatments customized to their individual characteristics (Heyneman et al., 1990; Turk, 1990). The use of patient subgroup characteristics as blocking factors or covariates in statistical analyses or explicitly in the design of studies may help to address many of the ambiguities in the pain treatment outcome literature.

CONCLUSION

In an earlier paper (Turk & Rudy, 1986) we raised the question as to whether assessment of cognitive factors in chronic pain patients was a "worthwhile enterprise." Since that paper, a plethora of studies have been published designed implicitly to answer our question. The results to date seem to lead to a conclusion in the affirmative. However, once the cognitive box has been opened, like Pandora's box, what escapes can never be put back. The diversity of measures of cognitive structures, processes, and conscious content that have appeared have not only answered our questions but raised many new ones. The evidence as to the importance of cognitive factors in self-reports of pain, emotional responses to pain, disability, and behavioral responses seems incontrovertible. Future studies need to refine these measures, address the issue of the independence among measures of putatively different cognitive constructs, and examine the mechanisms by which these cognitive variables produce their effects.

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