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PAIN DISORDERS IN LITIGATION: PSYCHIATRIC UPDATE AND EVALUATION GUIDE

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FOREWARD

Pain disorders in developed countries of the Western world could be, as some have suggested, one of the epidemics of our times. This refers not so much to acute pain, but to chronic pain that persists beyond the expected time for recovery, and often takes a protracted course of symptoms and impairment. The phenomenon may be a reflection of greater awareness of these conditions together with the availability of more sophisticated treatment; personal, psychosocial, and cultural factors; a shift in the form of psychosomatic processes; or the reinforcing effect of available safety nets from disability claims and personal injury litigation. Pain management as an industry has become legitimized, even while conflicting schools of thought continue to express widely divergent, theoretical positions. In the last twenty years alone there has been an evolution of nomenclature dealing with pain disorders, and shifting back and forth between physiological and psychological perspectives. Even while espousing a more integrated concept of biopsychosocial factors, there is still an underlying and historically entrenched mind/body dichotomy that resists this integration. Newer research points to greater awareness of neurophysiological mechanisms, but also of psychological substrates in which pain disorders arise. At the same time, in the arena of compensation and litigation, opinions are routinely required in which a choice must be made about whether a pain disorder is caused by a physical injury or not, whether the patient is disabled or not, and whether a patient's claims are reliable or not. For psychiatrists or psychologists who enter into these disputes, an understanding of newer terminology and current, evidence based foundations for opinions offered is necessary. This psychiatric update and evaluation guide hopes to lend some clarity in this regard, and to provide a practical approach to the evaluation process.

This work is dedicated to the late Edmund V. Drukteinis, MD who was as compassionate a physician, and as sensitive to the pain of his patients, as anyone I have known.

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CHANGING CONCEPTS IN PAIN DISORDERS

Chronic pain disorders continue to be a major health problem worldwide, in spite of advances in the understanding of pain mechanisms and a wide variety of newer pain management techniques (1) (2). In the United States, musculoskeletal pain alone affects 36% of the population during any 12-month period of time, which translates to well over a billion workdays lost annually to disability (3). Prognoses for conditions such as chronic low back pain have required revision, with new data suggesting even greater individual and societal impact and a worse natural course than formerly believed (4)(5). It is also understood now that chronic pain disorders comprise a heterogeneous group of clinical conditions, only a minority of which are associated with structural pathology; many others coexist with pain at other unrelated anatomical sites (6). Therefore, it is not surprising that pain disorders are frequently the subject of litigation in workers' compensation, personal injury, and disability claims (7). For example, almost half of the Social Security disability disputes pending in federal courts involve claims of chronic pain (8). In addition, damages for "pain and suffering" often make up the largest component of personal injury awards (9)(10), and are the regular focus of tort reform (11).

Over the last two decades, the definition of pain disorders has undergone significant change, and has been tied to evolving medical and psychiatric concepts about pain. These changes have caused confusion, however, and have led to frequent misuse of, or inconsistencies in, terminology. Some of these changes are outlined below, to illustrate the disparities.

Pain disorders fall under the general class of Somatoform Disorders in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) of the American Psychiatric Association (APA); but definitions of these disorders have been edited in subsequent editions—specifically, from DSM-III-R (1987) (12), to DSM-IV (1994) (13), and DSM-IV-TR (2000) (14). Even the definition of Somatoform Disorders has not remained the same. Compare the definition in DSM-III-R to that in DSM-IV-TR, given below, respectively.

DSM-III-R defines Somatoform Disorders as: "The essential features of this group of disorders are physical symptoms suggesting physical disorder for which there are no *demonstrable organic findings* or known physiological mechanisms, and for which there is positive evidence, or a strong presumption, that the symptoms are linked to psychological factors or conflicts"(12).

DSM-IV-TR defines them as: "The common feature of the Somatoform Disorders is the presence of physical symptoms that suggest a general medical condition and are *not fully explained by a general medical condition*, by the direct effects of a substance, or by another mental disorder"(14).

In addition, the term Somatoform Pain Disorder was a subcategory of Somatoform Disorders in DSM-III-R, but is no longer used by DSM-IV or DSM-IV-TR. It was previously defined as pain with "*no organic pathology* or pathophysiological mechanism... or...[pain that is] *grossly in excess* of what would be expected from the physical findings"(12).

Somatoform Pain Disorder, therefore, was an actual mental disorder often synonymous with what was termed *psychogenic pain*, a concept that is not as readily accepted today.

Despite the changes in DSM, Somatoform Pain Disorder continued to be referenced in the *American Medical Association Guides to the Evaluation of Permanent Impairment* (hereafter, *AMA Guides*), 5th Edition (2000), which is used to rate pain disorders, and specifically indicated that Somatoform Pain Disorder should not be confused with Chronic Pain Syndrome, which was not considered to be a mental disorder (15).

The *AMA Guides*, 6th Edition (2008) no longer uses the term *Somatoform Pain Disorder* but does endorse Chronic Pain Syndrome, describing it as "pain that continues beyond the normal healing time for the patient's diagnosis and includes significant psychosocial dysfunction"(16).

As of DSM-IV, there is no longer the subcategory of Somatoform Pain Disorder; it was replaced by three types of Pain Disorders: Pain Disorder associated with psychological factors, Pain Disorder associated with both psychological factors and a general medical condition, and Pain Disorder associated with a general medical condition (13). Technically, the latter should not be included at all in DSM because it would typically not be diagnosed by a psychiatrist. Regarding the other two, the following distinctions were made:

Pain Disorder associated with psychological factors is a category used when the psychological factors are judged to have the *major role* in the onset, severity, exacerbation, or maintenance of the pain.

Pain Disorder associated with both psychological factors and a general medical condition is used when both psychological factors and a general medical condition are judged to have *important roles* in the onset, severity, exacerbation, or maintenance of the pain.

Although both of these conditions could be classified as mental disorders, the first (a) is closely related to the earlier concept of psychogenic pain, and is more likely a mental disorder; the second (b) is synonymous with chronic pain syndrome so that it is not purely a mental disorder.

Psychiatric concepts of pain disorders have also undergone notable evolution over the last 50 years. For much of the first half of the twentieth century, psychiatric expectations of unexplained pain disorders were heavily founded on psychoanalytical concepts (17). Pain was considered either a conversion symptom or akin to hysteria; with repressed guilt, aggression, and other unacceptable unconscious forces supplying the underlying mechanism of the disorder (17). An amplification of this same concept was expounded by Engel and others, who described a pain-prone personality whose origin was in the denial of unconscious conflicts and dependency needs, for which the pain serves as a pathological expression (18)(19). These theories seemed to fit well with the concept of psychogenic pain, which was introduced decades earlier to soften the negative implications of hysteria (20). With the advent of the behaviorists, however, conditioning became the focus of most of psychological pain research. Concepts such as respondent conditioning, operant conditioning, and conditioned fear of injury (rather than unconscious conflict) were the accepted way of understanding and dealing with chronic pain (21). Together with the theoretical construct of *gate control* (22), which introduced a neurological mechanism that increased or decreased pain signals and included central (brain) modulation, psychogenic pain began to decline as a relevant concept. The explosion of

neuroscience research toward the end of the twentieth century, and the difficulties establishing reliable methodology for empirical research on covert psychological mechanisms, led to a de facto dismissal of the importance of psychiatric understanding of the pain patient's personal history and psychological factors in precipitating or maintaining a chronic pain condition. From the standpoint of litigation of pain disorders, causation and long-term disability were merely a reflection of the disease process. Unfortunately, gains in treatment of chronic pain have not followed accordingly; and, slowly, a shift is occurring, back to a more comprehensive psychobiosocial framework, with research that supports complex and multilayered mechanisms. Personal characteristics of pain patients—including physiological mechanisms, coping styles, enhancing factors, the presence of mental disorders, personality characteristics, and somatoform processes—are being seen as more relevant than previously acknowledged. In addition, the psychosocial context and interaction patterns of the patient with others in his or her environment are also being increasingly regarded as relevant.

The following reviews the current state of research and understanding of pain disorders from a psychiatric perspective, and addresses issues in conducting a forensic psychiatry evaluation of individuals whose pain disorder is the subject of litigation.

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PERSONAL FACTORS

Although the term *psychosocial factors* is widely used in pain disorders and infers a broader context of analysis, a great deal of contemporary research has focused on individual and personal factors of the pain patient. These include a number of physiological and psychological mechanisms, each of which deserves some attention here.

Physiological Mechanisms

Much of the early research in physiological mechanisms of pain focused on *nociceptive pain*, meaning that which begins as a response to noxious stimuli at peripheral nerve endings—that is, nociceptors—and then travels through the peripheral nerves and spinal cord to the brain (22). Along the way, particularly in the spinal cord, the message is modulated through inhibitory and excitatory mechanisms, some of which come from the brain itself and its effect on neurochemical structures in the spinal cord (22). In effect, this was in line with the gate control theory of Melzak and Wall (21), in which pain was seen not just as a result of the degree of peripheral tissue harm but also as how much the pain message was allowed to pass through gates that would open or close in the main communication channels. Now we understand that there is another type of pain, neuropathic pain, which is also regularly implicated in chronic pain conditions, where the pain persists because of more complex neurophysiological mechanisms almost independent of any remaining harm at the peripheral tissue site (23). This is seen, for example, in reflex sympathetic dystrophy (renamed complex regional pain syndrome; see below) in which the mediation and persistence of the pain is through pathological activation of the sympathetic nervous system (24). Other studies point to a *brain network* for pain perception and the more active role of central neurophysiological processing (25). Differences can be seen, for instance, in how the brain network responds to acute versus chronic pain; and those differences have even been measured with functional magnetic resonance imaging (fMRI) (26). There is increasing evidence, as well, that chronic pain is not simply the prolonged presence of acute pain (27) (28), and that central nervous system structures such as the amygdala may play a crucial role (29). Specifically, it has been shown that the *amygdala* is prone to neuroplastic changes as a reflection of chronic "neuropathic" pain, and that this involves very different types of synaptic transmission than with acute pain (30). At this point, it is not clear whether these abnormal changes precede chronic pain perception or follow the onset of the chronic pain (25)(31). In addition, and perhaps not coincidentally, the amygdala is now well known to play a role in various psychiatric conditions (31).

Disturbance in the *hypothalamic-pituitary-adrenal (HPA) axis* has also been associated with chronic pain disorders (32)(33), and with the stress-related mechanisms within that axis. Even subjects who do not have chronic pain yet but who have identified abnormalities in HPA axis function (e.g., loss of diurnal rhythm, failure to suppress cortisol with dexamethasone) appear to have a higher risk of developing chronic pain (34). Abnormalities in HPA axis function have been seen, as well, in depression and fatigue (35)(36), both of which are linked to a well-known chronic pain condition, fibromyalgia (see below). One possible explanation is that traumatic events early in life may alter an individual's stress response, which then creates vulnerability to future traumatic events and leads to symptoms such as depression, fatigue, and chronic pain (32). Furthermore, neurotransmitter substances (e.g., noradrenaline, dopamine, and

serotonin), neuropeptides, and cytokines have been implicated in chronic pain disorders, also suggesting an endocrine and/or immunologic process (37)(38).

There is also debate whether chronic pain is tied to *genetic factors* (37)(39)(40). Evidence in experimental animals of genetic transmission of pain mechanisms such as nociception (41), and polymorphism of several specific gene loci, indicates they may be associated with pain processing (42). This includes, among other things, a possible link to the mu-opioid receptor gene (43) and to enzymatic correlates that respond to pharmacological pain treatment (44)(45). The association of pain response with genetics may be provocative, but to the extent that gene expression is determined to be a significant factor, it will undoubtedly not be independent of environmental factors.

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Coping Styles

Increasingly, research has shown that the way an individual copes with chronic pain can have a significant impact on his or her suffering and disability. Three important concepts involved in coping with chronic pain are: fear-avoidance, catastrophizing, and self-efficacy. (See also, [Table A, Damaging Coping Styles and Pain-Enhancing Factors.](#))

Fear-Avoidance

Fear-avoidance, sometimes known as *kinesiophobia*, refers to fear of movement after development of acute pain so as not to cause reinjury (46)(47). This then results in disuse, deconditioning, and other conditions that lead to chronic pain. In the short run, avoidance of movement may seem to have pain-reducing effects, and so is reinforced in the mind of the sufferer. However, paradoxically, it leads to greater long-term deleterious effects (48). Numerous studies have shown the association between fear-avoidance and chronic pain, but it is not clear whether fear-avoidance is a mediator, a cause, or simply an effect of chronic pain (49)(50). In addition, it does not appear to be the only variable.

Catastrophizing

Closely tied to the fear-avoidance model of chronic pain is the concept of catastrophizing, a maladaptive response to pain characterized by an experience of heightened pain intensity, increased disability, and difficulty disengaging from pain (51)(52). Although it can be seen in acute pain as well, its importance is the robust association with chronic pain (53). Interestingly, the effect of catastrophizing can be independent of negative affect or the presence of depression (54). Related to this is evidence that catastrophizing is correlated with suicidal ideation, also independent of depressive symptomatology or pain severity (54). Catastrophizing often includes worrying about the pain and fear for the uncertainty of one's future. Of course, worry by itself is not necessarily pathological, and may be the necessary first step toward problem solving. In chronic pain, though, worry can become problematic, interruptive, intense, and uncomfortable, at which point it no longer contributes to problem solving. In fact, worry is more often tied to feelings of helplessness and loss (55). The sense of loss resulting from chronic pain can mean more than the direct effects of physical disability and change in physical functioning; more indirect effects may include diminished role performance, inability to participate in recreational activities, and lack of desire to interact socially. Individuals who catastrophize tend to ruminate about what other adversities they may face, and thus become hypersensitive to further threats against their physical integrity. In part, this type of worry is fueled by the perceived insolubility of the pain problem, especially when the solution is framed as the elimination of pain. Tracking the severity of pain, vigilance to further pain threats, and repeated attempts to eliminate pain by cure intensify a negative focus on one's self and cause greater frustration-both of which have a deleterious effect on the pain process (56)(57).

Self-Efficacy

A reciprocal concept to fear-avoidance, catastrophizing, worry, and helplessness is that of self-efficacy. In other words, the more patients believe that they are still in control of their life (i.e., positive self-efficacy), the more successful will be their adaptation to chronic pain (58)(59)(60). In a general sense, self-efficacy may be related to the broader concept of resilience. This refers to the successful adaptation of an individual to debilitating adversity or life stressors (61)(62). Resilience includes conscious efforts at self-mastery, sometimes referred to as active coping, but also genetic, developmental, physiological, and other psychological attributes (63). Passive coping, on the other hand, involves assigning responsibility for pain management to an outside source, or allowing other areas of one's life to be adversely affected by pain (64)

(65). Passive coping is a strong and independent predictor of disabling pain, and is a marker for risk of disability. Studies have shown that self-efficacy and active coping with pain require an acceptance of chronic pain, with the understanding that even chronic pain sufferers can remain engaged in valued aspects of their life (66).

In line with that, pain management techniques appear to be more effective in individuals with chronic pain when they adopt active coping mechanisms and a self-management approach (67). Recently, an interesting study focused on a variation of active coping in which chronic pain patients could be helped by adopting a new self-perceived role identity, to replace that of someone whose life had been suspended by pain. In this new role, the patient implements strategies that target self-enhancement, encourage positive emotions related to social meaningfulness, and incorporate a self-concept of heroism in dealing with the unavoidable and unpleasant context of chronic pain (68). This new-identity approach appeared to reduce many of the negative and destructive emotions that are well known to reinforce chronic pain and helplessness.

Table A. Damaging coping Styles and Pain-Enhancing Factors

Coping Styles

Fear-Avoidance	Known as kinesiophobia, refers to fear of movement after Development of acute pain so as not to cause re-injury
Catastrophizing	A maladaptive response to pain characterized by an experience of heightened pain intensity, increased disability, and difficulty disengaging from pain
Low Self-Efficacy	Passive coping, or a belief of helplessness and dependence on external sources of pain control

Enhancing Factors

Fear and Anxiety	Known to modulate the pain response and cause greater suffering than the physically traumatic stimulus
Attention and Hypervigilance	Overdirected attention to painful stimuli, anticipating and tracking pain symptoms

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Enhancing Factors

Closely tied to an individual's coping mechanisms and their effect on chronic pain, several mental processes have been seen to enhance or amplify the pain experience in a number of research studies. These mechanisms are to a great extent interrelated and have shown to have functional correlates within central nervous system structures (see "Physiological Mechanisms," above). The first of these are fear and anxiety, which are known to modulate the pain response and cause greater suffering than the physically traumatic stimulus that initiated the pain (69)(70). In this context, fear is not just a pervasive phobic personality trait, but specific to the pain itself and what it signifies. Similarly, anxiety does not refer to generalized anxiety, but an affective reaction to the negative implications of uncomfortable physical sensations. Psychological questionnaires measuring both fear and anxiety about pain show that high responders are not only prone to chronic pain but demonstrate unique activation in the anterior cingulate and insular cortex of the brain, as well as regions of the thalamus, lateral prefrontal cortex, and parietal cortex (69)(71).

In addition, greater *attention* to pain seems to be linked with chronic pain and disability (72). In fact, directed attention to painful stimuli increases synchronization between cortical structures (i.e., somatosensory and parasylian), as well as somatosensory and medial frontal. In contrast, *distraction* from pain shows much weaker synchrony (73). There is also growing recognition among pain researchers regarding the impact of dysfunctional attentional processes, sometimes labeled as *hypervigilance* (74). The more troubling question is whether attention and hypervigilance are learned responses or are inherent in the chronic pain experience. For example, it would make sense that individuals whose personal goal is to avoid or escape from the pain experience might become hypervigilant as a way of monitoring to what extent that goal is met. However, it is not clear how much of the process is conscious or unconscious, and by extension controllable or uncontrollable. It may be that attempting to teach people to divert attention away from the pain is not as successful as encouraging them to reshape their goals. There is supporting

literature to that effect, which describes the need for patients to challenge erroneous beliefs about pain, and to understand that a meaningful life is possible in spite of pain (46)(75). In addition, the provocative neurofunctional data has not resolved whether cortical brain correlates are the mechanisms to explain chronic pain, or are merely the result of chronic pain.

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Association with Mental Disorders

Distressing emotions understandably accompany chronic pain problems. At times these emotions are sufficiently severe to constitute a comorbid mental disorder. Estimates of the proportion of chronic pain patients with mental disorders have varied, although most studies have suggested figures of about 50% (76)(77)(78). Mental disorders associated with chronic pain are not unique to a particular type of pain; in large surveys they have been found to include patients with chronic back or neck pain, migraine, whiplash syndromes, and diffuse bodily pain (79)(80)(81)(82). There is also evidence that patients with chronic pain who have a comorbid mental disorder have a more impaired quality of life and greater disability (76)(83). Complicating the picture, treatment for chronic pain is markedly compromised when there is a comorbid disorder; and treatment of the mental disorder is equally difficult when the individual suffers with chronic pain (84)(85)(86).

The most common mental disorder to occur comorbidly with chronic pain is depression (79)(83). This can include a range of conditions, from Adjustment Disorder with depressed mood to Dysthymic Disorder to Major Depressive Disorder. Some researchers have even claimed that nonspecific chronic pain can be viewed as a type of depression—that is, "masked depression" (87). Along with depression, anxiety disorders are seen in chronic pain patients to a high degree; these can include Generalized Anxiety Disorder, Panic Disorder with or without agoraphobia, and Social Phobia (79). Patients with chronic pain also are more likely to suffer with alcohol and substance-related disorders, particularly Alcohol Abuse, perhaps as a consequence of the emotional distress of dealing with chronic pain or because of preexisting substance dependence issues, which then complicate the course of and recovery from chronic pain (79)(88). Posttraumatic Stress Disorder is also frequently associated with chronic pain, especially when a traumatic experience includes both a physical and a psychiatric impact (89)(90). Again, where both conditions are present, the potential impact on functional impairment is greater. That said, recent evidence suggests that individuals with Posttraumatic Stress Disorder may actually have a reduced sensitivity to pain (89). At the same time, there may be factors common to both Posttraumatic Stress Disorder and chronic pain that work to maintain both conditions (91), particularly when patients perceive themselves to be helpless and not in control of their life (90).

These associations, while strong, do not alone answer the question of whether the mental disorder is antecedent to the development of chronic pain, a consequence of the pain, or just incidentally comorbid (84). Certainly, there are studies that have shown, for example, that antecedent depression raises a risk factor for the development of chronic pain (92). However, this does not mean that the depression by itself is the causal factor. At the same time, preexisting anxiety disorders, substance abuse, and personality trait disturbance may be instrumental in leading to maladaptive coping mechanisms and/or amplification of a pain response. Unfortunately, empirical studies that try to address this issue may be limited by their methodology and by the need for a broader and more longitudinal understanding of the person in whom this comorbidity exists, rather than by simply focusing on a presumed singular association.

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Personality Characteristics

The relationship between personality characteristics and chronic pain has been studied extensively, perhaps because individuals with chronic pain frequently present with maladaptive behaviors and emotional distress that do not respond to conventional therapeutic interventions for mental disorders. Chronic pain patients, in fact, have a higher prevalence—over 40%—of personality disorders than psychiatric patients generally (93)(94). In studies that involved unstructured interviews, the most frequent diagnoses were dependent, passive-aggressive, histrionic, and compulsive personality disorders (94)(95). When the cluster classification of the *Diagnostic and Statistical Manual of Mental Disorders-IV-TR (DSM-IV-TR)* (14) was used, the most common cluster seen in chronic pain patients was cluster C: avoidant, dependent, and obsessive-compulsive personality disorders (96). In contrast, in structured interview studies, a higher

prevalence of paranoid, borderline, and obsessive-compulsive personality disorders were identified (97)(98)(99)(100). What emerges is the recognition that no one personality disorder is so clearly linked to chronic pain that it can either explain it or reliably predict its course. On the other hand, identification of unique personality characteristics or traits, apart from a personality disorder diagnosis, may have value. (See [Table B, Personality Theories and Chronic Pain.](#))

Historically, one of the earliest proposed theories was that of the *pain-prone personality*, which demonstrated prominent features of guilt, a history of suffering and defeat, an intolerance of success, a strong unfulfilled aggressive drive, and the development of pain subsequent to a loss or threatened loss (18). The pain was thought to reduce the guilt and shame of depression in a face-saving way. This posited theory was expanded further by subsequent researchers who believed that many chronic pain patients, from an early age, had unmet dependency needs and a desire to be cared for; that those needs were concealed; and, instead, these individuals took on an outward personality style of independence, relentless activity (*ergomania*), and excessive caring for others (87). Again, with a significant loss, disappointment, or injury, these patients shifted to assume an invalid role, which is the expression of their old unrecognized dependency needs.

Although these theories have been criticized for lack of compelling empirical data to support them (101)(102), anecdotally, and when longitudinal histories of patients are accurately obtained, similar personality characteristics, with dramatic shifts in levels of premorbid and postmorbid activity, are often seen.

A number of other personality characteristics, which have been observed and studied separately from personality disorders, deserve some attention here. One is the theoretical construct of *alexithymia*, which refers to patients who have difficulty identifying and describing their feelings, and who tend to be concrete, physical reality-based, and have an impoverished inner emotional or fantasy life (103)(104). Such individuals appear to have a predisposition to psychosomatic symptoms, including chronic pain.

Another personality dichotomy that has received attention in pain disorders is that of *extraversion versus introversion*. Hypothetically, individuals with extraversion have higher pain thresholds and are more tolerant to pain. They also may demonstrate adaptive reactions that help inhibit a pain experience, seek out greater social support, express feelings more readily, and maintain more social activity to counter passive invalidism (105). In contrast, individuals with introversion may have a tendency to brood and withdraw from social contact, obsess about their pain, and demonstrate neurotic patterns of adaptation (105). Closely tied is the observation that those individuals who show optimism deal with the pain experience in a more positive way and have a more favorable course, both in terms of subjective symptoms and personal functioning (106).

Chronic Pain Models and Personality

One model of chronic pain in relationship to personality is that of *diathesis-stress*, in which personality predispositions, diatheses, coupled with the stress of pain and its consequences, lead to what appears to be a personality disorder that perpetuates the pain and the disability (107). Proponents of this model cite the fact that patients and their families will frequently report no signs of a preexisting personality disorder, which, by definition, would then disqualify them from a personality disorder diagnosis according to *DSM-IV-TR*. The model assumes a biological/genetic vulnerability interacting with an environmental stressor such as an injury-producing pain, which then takes a chronic course. The difficulty with this model is obtaining an accurate history, because individuals who have become fixated on their pain and/or injury may reconstruct their own story in an erroneous way, or may not have sufficient psychological insight to recognize a preexisting personality disturbance. Furthermore, the biological underpinnings of the diathesis-stress model may not sufficiently take into account nonbiological factors, underlying psychological needs, and a more active role in maintaining invalidism than the individual may admit to.

A more recent theory of personality and chronic pain has incorporated an earlier psychobiological model, which views personality from the standpoint of *temperament dimensions and character dimensions* (108). Temperament dimensions are thought to be more biologically based, whereas character dimensions represent differences in goals, values, attitudes, and self-concept. Character dimensions tend to be more variable and psychologically governed. When measuring these factors as they relate to chronic pain patients, two significant findings were seen:

The temperament dimension of *harm-avoidance* was significantly correlated with chronic pain patients, even when controlling for comorbid mental disorders (109). This could reflect a neurobiological predisposition to the early emotion of fear and its related automatic behavior, which can help explain the self-defeating fear-

avoidance (see above) characteristic in many of these patients.

The character dimensions of *self-directedness* and *cooperativeness* were inversely correlated with chronic pain patients (109). Here, low self-directedness means that an individual does not expect to be able to control and positively influence a distressing situation, and does not participate cooperatively in his or her own rehabilitation. Again, these character dimensions appear to involve higher cognitive processes and represent less of a neurobiological predisposition (108). Also, high harm-avoidance and low self-directedness and cooperativeness were significantly related to diagnosed personality disorders within this study; they were seen in over 40% of the subjects (109).

The identification of distinctive personality characteristics that are associated with chronic pain remains a challenge, and no firm conclusions can be drawn beyond the fact that a number of specific characteristics may be important. Of greater relevance is how those characteristics exert their influence in the context of the entire psychobiological makeup of the person and his or her life history.

Table B. Personality Theories and Chronic Pain

Pain-Prone Personality	An earlier questionably validated theory that pain serves to reduce guilt and shame of depression; and/or a reflection of unmet dependency needs including a desire to be cared for
Ergomania	Relentless activity, and often excessive caring for others; pain serves as an expression of unrecognized dependency needs
Alexithymia	Refers to patients who have difficulty identifying and describing their emotional state; and have a predisposition to psychosomatic symptoms including pain
Introversion	Unlike extroversion, which can help adaptive reactions to pain, there is a tendency to brood and withdraw, and be pessimistic
Diathesis-Stress	A personality predisposition, not previously seen, but coupled with the stress of pain and its consequences leads to personality disintegration
Temperament and Character Dimensions	A psychological model in which temperament dimensions, e.g., harm-avoidance, are more biologically based; whereas character dimensions, e.g., self-directedness and cooperativeness, represent psychological differences in goals, values, attitudes, and self-concept

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Somatoform Processes

Pain as a manifestation of psychiatric disturbance has received attention for over a century, especially since the advent of psychoanalytical theory (110). Terms such as *hypochondriasis*, *hysteria*, *somatization*, and *psychosomatic disorder* have been variably used to characterize chronic unexplained pain symptoms (110)-although to counter the negative implications of these terms, some researchers prefer to use the rubric, "psychogenic pain" (111). Here, pain was seen as a manifestation of underlying anxiety, fear, depression, or as a defense mechanism for unacceptable impulses (e.g., hostility and resentment). Troubled life histories of pain patients seemed to offer support for this concept. As biological underpinnings of pain pathways became apparent, however, the focus shifted away from psychogenic to *neurogenic*. In addition, the role of psychological factors was difficult to identify, even in well-designed empirical studies (81)(82). Recently, an integrated biopsychosocial model has become more popular (112). Under this model, multilevel interactions are assumed, including genetic and biological factors, personal history, social context, and behavioral reinforcers. As a result, the category of Somatoform Disorders in *DSM-IV-TR* reflects this change and, as indicated above, describes these conditions as "not fully explained" by medical pathology, replacing the earlier description of "without" medical pathology (14). Unfortunately, this newer trend sometimes relegates psychological factors to a less important role, or simply attributes them to an excessive reaction to a pain experience. In spite of nominal attempts at integration, there is also a subtle, lingering mind/body dichotomy-that it must be one or the other, psyche or soma. Nevertheless, contemporary studies are demonstrating powerful psychological variables, preexisting and otherwise, that are closely tied to chronic pain and are measurable in well-constructed research methodology, which include

the identification of neural substrates to these factors.

Certainly, *cognitive-behavioral models* of chronic pain have by now been well documented. Pain-related avoidance stemming from fear of movement or injury, passive coping styles, and a negative outcome expectancy all result in a poor prognosis for chronic pain (46)(113)(114). This vicious cycle appears to be based on anxiety, not necessarily because of pain but also as anticipation of pain. This pattern independently will predict treatment outcome, and addressing it in a multidisciplinary program greatly improves the prognosis. A dramatic illustration of the deleterious effect of overfocusing on pain, and fear of pain from a prior memory of pain experience, may be seen in the anecdotal reports in the literature of two chronic pain patients who had substantial pain reduction when they forgot their pain (115). The authors reporting this phenomenon described the two patients as having long histories of chronic pain and opioid dependence. Then, incidentally, one suffered multiple epileptic seizures, after which her pain was relieved. The other had a motor vehicle accident and head injury, with retrograde amnesia, after which his pain was relieved as well. The authors also reported on earlier studies with electroconvulsive therapy and chronic pain syndromes, which raised similar questions about the role of memory and/or a direct effect of such amnesic experiences on central pain processing (116).

Another important phenomenon from earlier psychiatric literature, which is now garnering new attention in the understanding of chronic pain, is that of *attachment and loss*. Attachment theory holds that interpersonal experiences in early childhood and onward, such as loss or unavailability of important figures, contribute to the development of one's own self-image (i.e., model of self), as well as perception of others (i.e., model of others) (117). The attachment styles that are so formed govern one's responses to threat in the future. Recent studies have shown that individuals with insecure attachment are more likely to experience chronic pain and more distress from pain (118). Those with a negative model of self tend to appraise pain as more threatening, and so engage in fear-avoidance, catastrophizing, and hypervigilance to pain (119). Those with a negative model of others are less likely to find comfort in closeness, which can result in worse adaptation to pain (120). In addition, an insecure attachment style has an effect on caregivers who, with attachment styles of their own, may then engage in nonoptimal responses to the patient, causing a downward-cycling pattern (121).

Closely tied are observations about the effect of loss on pain, in particular permanent loss. For example, in the extreme, death and bereavement are known to increase maladaptive biological changes; but similar biological effects are seen even in times of crisis or separation from a partner (122). Some have described these biological/bodily changes in relationship to social and interpersonal events as a disruption of homeostasis (123); or have referred to a construct of *psychopathology of the somatic*, in which a physical symptom such as pain is itself the expression of an individual's personal disregulation (124). The propensity for disregulation is, theoretically, established by an initial insecure attachment style. (See [Figure A, Attachment and Psychopathology of the Somatic.](#))

Masked depression can also be an explanation for chronic pain. The association between pain and depression is well known, but an international survey by the World Health Organization also showed that a large percentage of patients with depression reported only somatic symptoms: half had multiple somatic symptoms, which were unexplained, and a significant number denied depression, even though they met the criteria for it (125). Many patients, of course, do perceive that their pain complaints are exacerbated by depression; but they may be less likely to understand that their depression and their pain may be synonymous. Therefore, simply questioning patients about symptoms of depression may not always be a reliable way to understand its association with pain. More specific scrutiny is needed into the criteria that constitute a patient's depressive disorder—when the depression actually began and what factors are fueling it.

Early adverse experiences as a precursor to the development of chronic pain have been studied for decades. Now, data from a large sample in the National Comorbidity Survey appear to confirm this relationship (126). In this study, childhood abuse was clearly associated with more reported pain. Even though childhood abuse is associated with depression, depression was not found to be the primary factor for these increased pain reports. Rather, the childhood abuse and depression seem to contribute independently to the pain. Other epidemiological studies similarly confirm that early adverse experiences (e.g., child abuse, neglect, and family dysfunction) are associated with a significant increased risk for adverse health outcomes generally (127)(128)(129). While the mechanism for this is not entirely clear, recent advances in developmental neurosciences suggest that early-life stressors induce persistent changes in neural circuitry that are implicated in the integration of emotional processing and endocrine/autonomic control (130)(131). This could result in an increased reactivity to the environment, cognitive impairment, pain sensitivity, and predisposition to emotional problems.

Figure A Attachment and Psychopathology of the Somatic

Pain as Emotion

Perhaps the most interesting area of research has been in conceptualizing pain as an emotion, and the identification of distinct central nervous system structures that are common to both pain and other emotional experiences (122). Here, of course, it is chronic pain rather than acute pain that shows this association. For example, one study noted that in chronic back pain patients who were diagnosed with fibromyalgia, cortical and subcortical regions of the brain were more active on fMRI than those of controls when presented with a pain stimulus (132). In addition, it appears that some of the areas activated in chronic pain patients are the same as those regularly activated in emotional tasks (133). The suggestion is that the brain areas activated by these chronic pain patients are also related to observed coping styles, such as catastrophizing, overattention, and negative evaluation (134). Moreover, it may be that pain as an emotion may depend in part on an individual's capacity for emotional awareness, both neuroanatomically and psychologically (135).

The hypothesis that follows states that emotional awareness is a cognitive skill, which undergoes a developmental process similar to Piaget's process of concept formation. This could, therefore, result in variations in the degree of differentiation of emotional processing, possibly dependent on how emotions were processed and what emotional information was available throughout development. The proponents of this model suggest five levels of emotional awareness, in ascending order, that have counterparts in both neuroanatomical sites and psychological sophistication (136):

Level I, *Sensorimotor reflexive*: Individuals are primarily aware of bodily sensations, apart from which they may not experience feelings. This is a more visceral activation, originating in the brain stem. Level I, as well as Level II, may be similar to the concept of alexithymia, described above.

Level II, *Sensorimotor inactive*: Individuals are aware of their body in action, enabling them to experience both bodily sensations and an action tendency; however, the ability to experience emotion as a conscious feeling may be absent. This action tendency appears to originate from the diencephalon.

Level III, *Preoperational*: Individuals are aware of separate feelings, but with little gradation and little capacity to appreciate the emotions of others. This more discreet emotion characteristic appears to originate in the limbic system.

Level IV, *Concrete operational*: Individuals experience a more expansive range of emotional experience, and can appreciate blends of feelings. There is a greater capacity to modulate emotional extremes and to engage in more complex emotional relationships. The ability to experience blends of emotions appears to originate in the paralimbic system.

Level V, *Formal operational*: Here, individuals are aware of blends of blends of feelings. There is much greater differentiation and integration in one's appreciation of the experience of others in the context of a differentiated awareness of one's own experience. They can make subtle distinctions between emotions, and their descriptions of such emotions are more novel. This blends of blends characteristic appears to originate from the prefrontal cortex.

With regard to pain, individuals on Levels I and II will thus experience a psychologically (psychogenically) induced bodily sensation only as a change in their physical condition, and be unable to connect it to the psychological trigger (137)(138)(139). This model, then, may provide a neurobiologically founded theoretical framework to understand somatoform or psychosomatic processes, and support the concept of pain as an emotion in a substantial group of patients.

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PSYCHOSOCIAL FACTORS

The term *psychosocial factors* is widely used in pain research and in the characterization of nonbiological variables that are either evident or inferred when dealing with chronic pain patients. Often the term is used generically and there is no articulation of what the psychosocial factors actually are (140)(141). In its broadest use, it can refer to many of the individual factors that are described above. Here, the focus will be more on external correlates from the environment of individuals with chronic pain, which may or may not impact the intensity and course of the pain experience. They include interpersonal, social context, and cultural factors.

Interpersonal Factors

Familial interactions with the individual who has chronic pain, particularly spousal interactions, have been identified as significant in promoting either disability or health. There is evidence, frequently cited, that spousal *solicitousness* is associated with greater reports of disability (142). These findings are said to be consistent with operant behavioral theory, which posits that solicitous behaviors on the part of the spouse serve as a positive reinforcer for pain behavior dependence and disability. The counterpart to this, then, might be presumed to be that less attention from a spouse should promote greater independence and less disability. This simple paradigm is not always accurate, however; more recent studies have shown that there are, in fact, a number of different types of spousal behavioral responses, giving rise to variable effects (142). For example, spousal solicitousness in association with greater disability is seen as more relevant in male, rather than female, patients. At the same time, many pain patients, both male and female, with solicitous spouses frequently reported experiencing negative feelings in response to the overattention. The range of spousal behaviors also can include: encouraging task persistence; helping to problem-solve; discouraging or, conversely, encouraging, pain talk; shielding the pain patient; offering to help but failing to provide it, ignoring cries for help, providing help, providing help but projecting hostility; expressing frustration; and attempting to distract the pain patient from his or her discomfort (142). It is also true that spouses who project helplessness and engage in catastrophizing in dealing their partners' pain may suffer more distress themselves, as well as reinforce a pain focus in their partners (143); conversely, true *empathy* with the sufferer can have a healing effect, and promote intimacy between the partners (144) (145). Clearly, the response of family members to a pain patient can be complex, hence does not always lead to a predictable effect; but the type of interpersonal reaction is nonetheless important to identify.

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Social Context Factors

Two factors are relevant to understanding the course of pain and the functional impairment of the sufferer: the social context in which pain arises and where the injury occurred. It is well known that *occupational injuries* that produce a pain condition are associated with poorer outcomes than nonoccupational injuries (146)(147). A number of possible explanations have been proposed for this, including dissatisfaction with the job or the employer, adversarial and secondary gain issues arising out of workers' compensation, preexisting risk factors, differences in treatment availability, and a higher prevalence of malingering (148). Closely linked is the potential reinforcing effect of *disability claims* generally, even outside of an occupational injury. Here, too, job or career dissatisfaction may play a role, as may a lack of motivation, for a variety of personal and psychological reasons, to remain in the workforce (149). Pain that is the subject of damages in *personal injury litigation* can be reinforced by the hope of a large monetary award or by the suggestive influence of the patient's attorney (150). An important distinction needs to be made between pain and disability, even though the two are frequently and closely connected in such settings. Pain alone is rarely sufficient reason for disability, without some type of objective measurement of structural impairment (16). Typically, pain is considered too subjective to establish impairment, and there are marked differences in the degree of functional impairment cross-culturally, as well as in governmental/legal venues where a pain condition may not be recognized for purposes of disability (151).

Another important social context is the impact of the *healthcare system*. For one, the attitudes and beliefs of healthcare practitioners regarding pain can greatly affect outcome in their patients (152). For example, promoting only a biomedical approach, or reinforcing passive treatment, tends to reinforce chronic pain and longer periods of disability, especially where no objective medical pathology exists. Also, there is a

suggestive effect possible from health practitioners who work in pain treatment centers, facilities that have become an industry in and of themselves (150)(153).

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Cultural Factors

Cultural and ethnic differences with regard to the experience of pain have been demonstrated for over 50 years (154). One well-known study looked at differences between Irish Americans, Jewish Americans, Italian Americans, and elderly Americans, and found that elderly Americans and the Irish tended to hide their pain experience through avoidance and minimization, while the Jewish and Italians were much more expressive and willing to seek relief. Ethnic disparities in pain have recently gained increasing attention, as well (155). One such study found that African American and Hispanic American groups that had a strong ethnic identity reported lower pain thresholds and tolerances than controls (156). The reasons for these differences remain unclear; however, possible explanations include some combination of psychological and sociocultural factors, among them: attitudes, gender, language, acculturation, learning and cultural conditioning, degree of expressiveness, chronic stress, education, religion; and socialization of pain expression, heightened attention to painful stimuli, and pain coping styles (157). Some have postulated that the differences arise as a consequence to the lower socioeconomic status among those groups, thereby complicating the healthcare outcome. But lower socioeconomic status does not alone explain the differences (156). Besides the importance of these findings for the treatment of pain conditions, the even greater significance is that the pain experience itself can be shaped by culture. It may be that shared experiences within a culture affect not only beliefs and practices related to pain but also underlying neurobiological processes involved in sculpting it (156).

Culture also may be instrumental in legitimizing pathological conditions, thereby suggesting a condition itself, or at least its form. A look at psychosomatic illnesses, for example, shows that nineteenth-century conditions such as hysterical paralysis and neurasthenia have given way to twentieth-century conditions such as chronic pain and chronic fatigue (158). Statistics certainly demonstrate that at least in Western countries the prevalence of chronic pain has increased dramatically, and may be a reflection of the meaning individuals and culture give to pain within contemporary society (159). To the extent that Western culture emphasizes science and technology in dealing with pathological conditions, the relevance of meaning may be lost, and the potential for cultural suggestibility obscured (150).

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FORENSIC PSYCHIATRY EVALUATIONS

Historical Assessment

In addition to obtaining a standard psychiatric history along the lines of a traditional assessment, the evaluation of patients with pain disorders presents unique challenges, and so requires a specialized approach. The threshold problem in these evaluations is *patient resistance* (22). Although seeing a psychiatrist may not carry quite the same stigma as it did years ago, because people today, in general, have had greater exposure to mental health intervention, many still are understandably apprehensive, embarrassed, or reluctant to tell a total stranger, even a mental health professional, about their personal difficulties. In addition to these factors, patients with pain disorders commonly demonstrate resistance when referred to a psychiatrist or psychologist; and this extends to the evaluation itself and to the difficulty in obtaining an accurate history.

There are a number of possible explanations for patient resistance in these circumstances. First, many patients who have been distressed over pain have medical pathology, or in some cases undiagnosed medical pathology. Therefore, they sense intuitively that something is wrong with them, so that exploration of psychiatric issues may appear irrelevant. Second, when the referral from a medical or surgical treatment provider is first broached, the patient may assume that he or she is no longer believed as having a genuine pain problem. Third, if there are independent psychological problems, personality trait disturbance, lack of motivation, or secondary gain factors, a patient may be afraid that the psychiatric evaluation will expose issues that will undermine the validity of his or her pain complaints and/or pain impairment. Of course, psychological resistance in a psychoanalytic context has been described extensively in early twentieth-century literature (160)(161), and breaking this type of resistance in order to reach unconscious conflicts is

known to require careful interpretations over a considerable period of time, during which a psychotherapeutic relationship is built (162). Therefore, to the extent that a pain disorder has unconscious or conscious psychological factors that help maintain it, it would be presumptuous to assume that breaking the resistance would be any easier. It is likely that this is one of the reasons that modern psychological pain management has shifted to a more cognitive-behavioral approach, which is more time-efficient, less intrusive, and more palatable to patients. Sometimes, resistance can be extremely intense, and be accompanied by powerful emotional displays. As long as the psychiatrist has not triggered this with an unempathic approach, the intensity of the resistance does not necessarily validate the legitimacy of the pain. In fact, for those pain patients who demonstrate some degree of malingering, intense resistance is frequently used to counter any implication of invalidity.

It should be clear, then, that the *psychiatrist's attitude* is an important consideration, and can have a major impact on the historical assessment (160). It is helpful to inform patients up front that the purpose of the evaluation is not to discount or discredit their pain, and explain to them that psychiatric evaluations are commonly performed where pain is particularly distressing or has persisted for a period of time, and that similar evaluations are conducted in almost every pain center in the country. It may also be helpful to personalize the conversation—for the psychiatrist to tell the patient about his or her own experience in dealing with pain disorders. It is also necessary that the psychiatrist develop rapport with the patient, take an empathic approach, and refrain from projecting skepticism. Even if there are serious questions about the legitimacy of a patient's pain complaints, especially in a dramatic or hysterical presentation, the psychiatrist should never impugn the patient's credibility or direct challenges to the claim. Not only will doing so thwart the historical assessment but it will compromise the purpose of the evaluation. This is not to say that inconsistencies or contradictions in the patient's account and presentation be ignored; and they may need to be addressed in the evaluation report. However, usually it is self-defeating to try to do that with the patient directly, other than by making a very neutral inquiry about some of the more relevant noncorroborating data that may be contained in records or from other independent sources.

Psychiatrists should also be aware of their own unconscious reactions to a pain patient's personality and demeanor (i.e., *countertransference*). This can become a major problem when the psychiatrist faces a patient who is presenting extreme helplessness and dependency, or expressing dramatic negativity toward the psychiatrist. Accepting the patient's presentation as it is, without exhibiting irritability or skepticism, helps to move the evaluation process forward and allows for more complete information gathering. It also is the more ethical way to deal with a suffering individual, regardless of one's own opinions about the nature of the distress.

Finally, psychiatrists who perform evaluations on patients with pain disorders should be familiar with medical terminology, diagnostic descriptions, medical and surgical procedures, and the various diagnostic tests that are performed by medical treatment providers. Obtaining a history without background knowledge of this kind impairs the credibility of the psychiatrist. At the same time, psychiatrists should take care not to use this knowledge inappropriately—to go outside the bounds of their expertise to directly comment on medical or surgical care.

Pain History as Narrative

Obtaining a *pain history* that is both accurate (at least from the patient's perspective), and allows for a free-flowing exchange between the patient and the psychiatrist, is the foundation of a good historical assessment. Because patients with pain disorders typically are apprehensive about seeing a psychiatrist, the best starting point is to discuss the pain itself. The psychiatrist should ask about the onset of the pain and the setting in which it first occurred; for example:

What were the characteristics of the pain at the time of onset, and how did the pain progress over time?

What is its pattern?

Is this the first time the patient has experienced pain, or have there been repeated flare-ups?

Is the pain diffuse or focal?

A detailed inquiry such as this establishes the psychiatrist's interest in the pain and acknowledgment that it is the central problem. It also allows the psychiatrist to witness how the patient describes the pain experience, and the emotional effect that accompanies that description. Also, the more familiar the

psychiatrist is with relevant medical terminology, and medical and surgical treatment, the more credibility he or she will have as someone who can perform this type of evaluation.

An important concept that has gained renewed attention among pain practitioners is that of *narrative* (163). By this is meant the story that patients tell about their pain and suffering, and the meaning of pain in their lives. It requires that patients be given the opportunity to tell their story, and that they be comfortable with the psychiatrist to whom they are telling it. Every story involves a transaction between the teller and the listener; and the story changes over time, based on the circumstances of the transaction and the impact of life events on the story, both past and present. The story may be an expression of the disastrous consequences of pain in the life of the patient, or a metaphor for the patient's life.

The more closely a psychiatrist listens to the story, the more understanding he or she will gain about the pain and the patient. On the first telling, the story should not be examined overcritically, especially if it is not told logically or coherently. Also, the true meaning of story will never be understood if the psychiatrist interrupts too often by asking questions from checklists. A narrative approach to the history is much more useful, in that it allows for the various levels of the pain experience to become manifest and for the meaning of pain to be recognized. Therefore, regardless of the information that is necessary to be elicited in a historical assessment, the psychiatrist must take the time to listen to the patient's narrative.

Longitudinal History

The ultimate aim of a good historical assessment is to establish an accurate and detailed *longitudinal history* of the patient's life and the pain experience. This comes, of course, from the patient's own account and the narrative of his or her life, which he or she wants to present; but it also comes from the integration of corroborating information from many different sources. The importance of this cannot be overstated. A good longitudinal history is vital to a proper understanding of the framework in which pain has occurred, its antecedents, how it is received and withstood, and where it fits within the other independent struggles of a person's life. Is the pain a new experience or just one of a number of similar pain experiences at other times? Does it present now as the single focus of distress, or is it one of many diffuse pains and stressors? Does the patient's narrative really coincide with the time of onset of pain, attempts at rehabilitation, and earlier accounts of well-being? Obtaining an accurate longitudinal history and collating it from myriad sources of factual material often contained in records is a painstaking task. It is seldom done in the ordinary treatment situation by a clinical psychiatrist, much less a medical treatment provider. Yet, psychiatric and medical treatment providers often erroneously presume that the longitudinal history they have been given is accurate, hence may proceed to draw conclusions about causation and degree of impairment, which ultimately prove to be unreliable.

The timeline for a longitudinal history should extend back into childhood and early familial as well as developmental experiences, and proceed through the individual's life in all the traditional areas of psychiatric inquiry, weaving in medical and pain experiences as they arise. Where there are inconsistencies and contradictions, they may need to be reconciled or reexplained. The longitudinal history should include the impact of pain on the individual's life in personal, familial, and occupational functioning; the perception of the pain and its meaning through the course of medical and surgical treatment; and the response and effect of the pain in the patient's environment.

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Mental Status Examination

In addition to gathering historical data, all psychiatric evaluations include a mental status examination. This is no different in the evaluation of pain patients. Certainly, identifying features of a psychiatric disorder can be important since comorbid conditions with chronic pain affect the prognosis and treatment efforts. Moreover, there are particular aspects of a mental status examination, and observations of pain patients generally, that need discussion.

First, the general *appearance and attitude* of pain patients can vary tremendously. From the initial meeting, pain patients often present in a manner that reveals their level of psychic frustration. Some patients, of course, will attempt to appear normal and show that they are bearing their discomfort stoically, whereas others will make no effort to disguise the fact that they are visibly suffering. Patient posture and gait are also informative. Some will be hunched over and ambulate with labor; others will have a grotesque or distorted look to their physical stature. While a physical examination is not part of a typical psychiatric

evaluation, nonetheless visual inspection of a pain area, palpation for tenderness or exaggerated response, as well as other non-intrusive measures may at times be indicated and can provide relevant information. Likewise, it is important to note the "pain paraphernalia" that patients bring to the interview—canes, braces, cushions, crutches, wheelchairs, and so on. In terms of attitude, they may project their contempt for having to be at the evaluation in the first place, especially because of the implication that their pain is not real. These patients can often appear resistant or even combative. Their cooperativeness or hostility may be important factors in how easily treatment providers will be able to engage them in the rehabilitation process, and what obstacles they may be facing (22).

The *flow of thought* in a patient's speech is also a necessary subject of observation. To begin, the psychiatrist should note how spontaneous or reticent the patient's speech is. Some patients are extremely withdrawn, and great effort must be made to assess their history and feelings. This posture may reflect a resistance to the psychiatric interview, or may signal more troubling personality pathology. At times, the inability or unwillingness to communicate may also mean that an individual is unable to express his or her feelings, or can do so only in the form of describing bodily sensations. (Refer to the discussion on alexithymia, above, as it relates to psychosomatic illness.) In other cases, the patient's speech may be excessive, rambling, disorganized, or incoherent. Such a presentation could indicate that the person cannot order his or her thoughts in a meaningful way because of stress, personality disturbance, intellectual deficits, or more severe mental illness, thus creating another challenging obstacle to rehabilitation.

The patient's *mood and affect* are obviously of paramount importance in evaluating chronic pain. This includes the emotional state that is visibly projected, as well as that which is concealed. Careful observation of pain patients can usually lead to the identification of current feelings, which can then be correlated with their history. Patients may present with visible signs of worry, anxiety, and anger. Tension can be seen in their countenance, and autonomic arousal can be observed through flushing, sweating, and shakiness. Some patients reveal intense mood disturbance in the form of depression and despair; others are irritable, frightened, or even panicked. Still others will claim that they are emotionally unaffected, while at the same time they easily become tearful or even give way to sobbing. Some may cry while discussing the severity of their pain; but their sadness has become so pervasive that doing so obviously points to another level of emotional involvement. Others may display dramatic evidence of catastrophizing their discomfort, which precludes an objective approach to pain management and recovery. Inappropriate mood or emotional expression must also be noted, especially if it is markedly inconsistent with the patient's descriptions of symptoms and complaints.

The *content of thought* of pain patients can have unique characteristics, which need to be identified. As indicated above, the narrative history of symptoms is important because it leads to an understanding of what meaning the pain has in the patient's life, and how the pain is conceptualized. It is also important to query patients about their belief in what causes their pain. Often these descriptions are quite unusual and entirely inconsistent with medical data. Closely linked to this is whether patients believe in their physicians' explanations and/or have confidence in the treatment direction. Patients will frequently have visual depictions of bodily structural damage (e.g., tearing, grinding, or neural pressure) that are anatomically unfounded. Furthermore, they may be terrified that the pain represents increasing damage and, possibly, permanent injury or paralysis. Patients often have compelling irrational cognitive distortions about their pain. They may think categorically, so that the limitations the pain has caused in their lives may now present erroneously as total incapacity. This overgeneralization can be seen in their descriptions of what the pain has done to their lives, as well as what they believe they are still able to do. A discussion of the patient's general level of activity during the course of the day is helpful, and can reveal the presence or absence of self-efficacy and resilience. It may also demonstrate counterproductive behaviors and a preoccupation with pain. Patients will sometimes describe in exhaustive detail minute aspects of their pain and its course, so that it appears their life revolves entirely around those physical sensations. A telltale sign of obsessive preoccupation is when a patient produces a voluminous notebook or diary (without having been requested to do so) that tracks the hour-to-hour course of symptoms, sometimes illustrated with pictures and diagrams.

In addition, *cognitive screening* may be necessary as a general assessment of brain function. This can be done through a standard Mini Mental Status Examination, or other traditional means. At times brain impairment can signal an accompanying diagnostic problem which can complicate the prognosis of a chronic pain condition. It also can reflect on overmedication from narcotic analgesics and other sedating medications.

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Pain Behaviors

It has long been known that patients with chronic pain may exhibit behaviors of suffering that are not necessarily consistent with their own self-report of pain intensity or with the degree of objective medical pathology (164)(165). These behaviors help reveal signs of helplessness, symptom magnification, disability exaggeration, and invalidism. Their presence does not, however, mean that objective medical pathology cannot also exist. In some individuals, long-term suffering with pain that has been unresponsive to traditional treatment can bring on learned behaviors of this kind. In other cases, psychological factors are operative as an independent source of suffering, or simply as a reaction to pain (166). Recently, there has been an interest in distinguishing the functions of pain behaviors into two types, one being a *protective function*, which may be quite personal and linked to fear of injury, and the other a *communicative function*, which conveys information to observers about the internal state of the individual (167). A number of specific pain behaviors can, therefore, be grouped with regard to those differences in function, as follows (22)(167):

Protective Function

Guarding: Abnormally stiff, interrupted, or rigid movement while shifting from one position to another.

Bracing: A stationary position in which a fully extended limb supports and maintains an abnormal distribution of weight.

Rubbing: Touching, rubbing, or holding the affected area of pain for a minimum of three seconds.

Communicative Function

Grimacing: Obvious facial expression of pain, which may include furrowed brow, narrowed eyes, tightened lips, corners of mouth pulled back, and clenched teeth.

Sighing: Obvious exhalation of air, usually accompanied by shoulders first rising and then falling.

Moaning: Vocal expressions of suffering, usually with a difference in voice quality or pitch.

The precise reason for the development of pain behaviors is not clear. Behaviorists have argued that operant conditioning is the mechanism, with pain behaviors being reinforced by reward from the environment, including responses from significant others (168). By looking at protective and communicative functions, though, it may be possible to understand pain behaviors more in their adaptive context—that is, to escape from the cause of pain or mitigate its effect; command attention and promote vigilance; or, perhaps, in the longer term, encourage energy conservation during healing (169). In any case, pain behaviors do not need to be proportional to the extent of tissue damage, but may represent patients' perspective about or their need to communicate illness.

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Physical Inconsistencies

Typically, psychiatrists who conduct evaluations on individuals with chronic pain will not perform physical examinations. However, they will routinely review the physical examinations of nonpsychiatrists and medical treatment providers. Within those descriptions, they will often find references to physical inconsistencies, giving them some indication of the type of physical signs that are relevant and should be noted. Both from the standpoint of the patient's history and the physical examination, the most notable issue is the *distribution of pain*, in conjunction with the patient's own sensory experience that outlines that distribution. Commonly, physicians evaluating pain complaints will attempt to demarcate an area of numbness, tingling, or other related pain discomfort that is consistent with the dermatomal patterns that follow particular nerve root innervation. Diagnostically, the examination can then either confirm or refute the involvement of a specific nerve such as might be suggested by a herniated disc or noxious injury to a nerve root. When the distribution of pain is not consistent with dermatomal patterns, there may be a presumption that no nerve is involved. Moreover, even though other conditions exist that can at times be nondermatomal, it is not

uncommon to question the legitimacy of the patient's complaints generally when poorly explained nondermatomal symptoms are present (22). The physical examination can also include specific attention to nonorganic signs, which, as has been outlined for example with chronic back pain, are based on abnormal behavioral responses rather than purely physical pathology (166). These include assessing tenderness that is either superficial or nonanatomic (i.e., covers a wide area and crosses musculoskeletal boundaries); simulation tests in which it appears superficially that a pain-related movement is being required on examination, when that is not the case; distraction of the patient followed by observation to determine whether the same pain response is apparent; and demonstration of inconsistent regional weaknesses and sensory changes (166). It is important not to conclude that the presence of nonorganic signs on physical examination means that medical pathology is necessarily absent; that said, this observation does contribute to the overall clinical picture and can help to expose operative psychological factors. It is similarly important to understand that nonorganic signs may be seen with widespread neurological illness (more commonly in elderly patients) and can vary among different cultures (166).

Surveillance is a controversial area for obtaining corroboration of pain and pain impairment (170). Surveillance pictures or films within a discreet period of time may not accurately reflect an individual's overall functional ability. By necessity, many disabled individuals must exert themselves briefly beyond their actual capability, and then pay the physical consequence afterward. It is always difficult to assume that a discreet period of surveillance is representative of total functioning ability. Similarly, a surveillance tape may not be able to capture internal emotional states. However, if an individual suffering with chronic pain has represented that certain activities are impossible or never performed, then surveillance may be able to disprove this representation. Where this is observed in multiple settings or over a prolonged period of time, the inconsistencies may be relevant to actual versus claimed impairment.

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PSYCHOLOGICAL TESTING

In forensic psychiatry evaluations of individuals with pain disorders, psychological testing becomes a valuable supplement to historical assessment and observational data. Many of the tests used are straightforward questionnaires that have been developed to enhance an understanding of key psychological factors frequently seen in chronic pain patients. As such, they do not necessarily require a psychologist for interpretation; and manuals are provided with detailed instructions on scoring and interpretation of data. However, forensic psychiatrists who do not use a psychologist to administer such questionnaires should have some familiarity with psychological testing generally; statistical analysis, particularly with regard to issues of validity and reliability; and the limitations in drawing conclusions from psychological test data alone. Not infrequently, a psychological test may show results that are internally inconsistent or contradictory. This is often seen where multiple tests are used, and where it is sometimes difficult to reconcile variable findings. For those psychological tests that are more sophisticated than just a simple questionnaire, computer-programmed scoring of data is available, together with a narrative that provides interpretation and analysis of the data. Both psychiatrists and psychologists are cautioned to avoid relying too much on one or another description of the patient within the narrative, which could either be not representative of the test results as a whole or an erroneous description that is not consistent with the entire history and the total evaluation.

There are literally hundreds of psychological tests available today, a number of which may have relevance to pain disorders. No specific battery of tests should necessarily be used, and much depends on the availability of newer and more improved testing instruments or on more advanced knowledge within the research literature on pain disorders. Below is an outline of a number of categories of relevant data that takes into account factors identified in evidence-based studies, as described above, along with representative tests for those categories. Choosing which test or tests in each category to use is largely a matter of preference and/or the experience of the administrator. It is important to note that tests which do not have internally built validation scales, e.g. Beck Depression Inventory and others, may have limited usefulness in a forensic evaluation since they merely report the subjective account of the individual tested. Therefore, it is important to include in any battery of tests some instruments which include validity measures, exaggeration scales, and means to address inconsistencies. The following do not necessarily comprise the best or most used tests, but are representative of the areas of inquiry that are important to address in pain evaluations:

Mental Symptom Assessment

Beck Depression Inventory (171)

State Trait Anxiety Inventory (172)
Symptom Checklist-90R (173)
Comprehensive Mental and Personality Assessment
Personality Assessment Inventory (174)
NEO-Personality Inventory-Revised (175)
Minnesota Multiphasic Personality Inventory-2 (176)
Symptom Magnification, Catastrophizing, Fear-Avoidance
Pain Drawing Test (22)
Visual Analogue Scales (177)
McGill Pain Questionnaire (178)
Pain Catastrophizing Scale (179)
Tampa Scale for Kinesiophobia (180)
Emotional Awareness Measures
Toronto Alexithymia Scale (181)
Levels of Emotional Awareness Scale (182)
Sickness Impact
Oswestry Back Pain Disability Questionnaire (183)
Sickness Impact Profile (184)
Pain Locus of Control Scale (185)
Dallas Pain Questionnaire (22)
Pain Self-Efficacy Questionnaire (186)
Comprehensive Pain and Coping Assessment
Multidimensional Pain Inventory (187)
Chronic Pain Coping Inventory (188)
Profile of Chronic Pain: Extended Assessment Battery and Screening (189)
Coping Strategies Questionnaire (190)
Millon Behavioral Health Inventory (191)

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CHRONIC PAIN SYNDROMES

Pain disorders are a regular feature of civil litigation and workers' compensation claims. They can be subsumed under the more generic label of *pain and suffering*, or as one of a number of pain conditions often described as a *chronic pain syndrome*. The latter term is an offspring of concepts such as psychogenic pain or somatoform pain, in which psychological factors play a role in the onset, severity, exacerbation, or maintenance of the pain. As indicated above, many people with chronic pain in one location of the body have chronic pain in another as well. The reason for this is not clear, but may include a propensity for chronic pain, shared risk factors, or some connection in etiology between the two; for example, knee pain as a result of abnormal posture to accommodate back pain (192). The specific pain syndromes discussed in this section are those prevalent in litigation.

Low Back Pain

Low back pain is the primary cause of absenteeism and disability in every industrialized society (166). It often follows personal injury from an accident, or is a work-related injury. However, only 3% to 5% of all back-related disorders have a clear medical source (e.g., herniated lumbar disc) (193). Of these individuals, about 25% have a long-term reduced work capacity, and they contribute the most to the economic and social burden of low back pain (194). Many low back pain cases follow unsuccessful surgery, and are sometimes referred to as having a *failed back syndrome*. Studies have persistently shown the presence of psychological factors that contribute to chronic low back pain and disability (195)(196). Dissatisfaction with work is also a consistent predictor of poor outcomes in treatment and rehabilitation (197). Interestingly as well, in spite of advances in medicine and a greater understanding of pain disorders, chronic back pain and disability continue to increase (22). Claims of back pain disability are often seen in individuals whose work requires heavy lifting, carrying, or other physical labor. Those who have little education or other work skills may have difficulty finding new vocational directions. At the same time, because such a small percentage actually demonstrates clear objective medical pathology, low back pain litigants are often viewed skeptically and become the subject of adversarial legal proceedings.

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Fibromyalgia and Myofascial Pain

Fibromyalgia is a chronic pain disorder characterized by widespread musculoskeletal aches, pains, stiffness, and tenderness (198). It may be associated with sleep disturbance, fatigue, and depression. There is considerable controversy about whether it represents an actual disease entity or simply a group of nonspecific symptoms (199)(200). Many researchers believe that the concept of fibromyalgia is so broad as to include many unrelated possibilities. Similarly, there is no consensus that fibromyalgia originates from trauma, meaning that it may not be a legitimate personal injury for litigation (198).

In contrast, *myofascial pain*, a widely used term that is short of the full spectrum of fibromyalgia, is often claimed to be the reason for chronic pain following trauma. This is not, however, well established in the scientific literature, especially because trigger points and/or tender points that are purportedly identified on physical examination have shown poor interrater reliability (201). No other diagnostic studies can confirm the diagnosis. Therefore, both fibromyalgia and myofascial pain should be scrutinized closely for psychological factors when they present as the basis for physical damage.

Complex Regional Pain Syndrome

Pain that spreads beyond the site of an original injury, usually to an entire extremity, and persists without evidence of structural damage is sometimes attributed to an abnormal sympathetic nervous system mediated response (202). Where there is direct damage to a peripheral nerve, which is the original trauma, this has been known as *causalgia*. Where there has been no direct nerve damage, it has been known as *reflex sympathetic dystrophy* (RSD). Typical sympathetically mediated symptoms include exquisite pain even to light touch, edema of the skin and subcutaneous tissues, temperature and color changes, mottled or shiny skin, increased hair growth, and disturbed hair growth. Where the typical symptoms have not been present, so that a sympathetically mediated response is not confirmed, persistent pain does not have a good medical explanation.

More recently, these conditions have been renamed as *complex regional pain syndrome* (CRPS-I and CRPS-II) (203). This new classification was purported to be an attempt to create a better definition; it has, however, subtly allowed conditions with no known medical explanation to receive a legitimate medical label. Specifically, CRPS can be subclassified into sympathetically and nonsympathetically mediated pain. To the extent that it is sympathetically mediated, CRPS-I would then include the earlier RSD classification, and CRPS-II would include *causalgia*. But when the pain is nonsympathetically mediated, it has no known medical explanation, in which case the diagnosis of CRPS is misleading. In such instances, CRPS may in fact be largely related to psychological factors. This becomes particularly significant because CRPS usually follows trauma, and simply making a diagnosis of CRPS implies that it is a medical condition as a result of that trauma, when in fact there may be no medical connection at all. In the *AMA Guides*, 6th Edition, this issue is specifically addressed as follows:

"Since a subjective complaint of pain is the hallmark of (CRPS), and many of the associated physical signs and radiological findings can be the result of disuse, the differential diagnosis is extensive; it includes somatoform pain disorder, somatoform conversion disorder, factitious disorder, and malingering. Consequently, the approach of the diagnosis of these syndromes should be conservative and based on objective findings."(16).

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Repetitive Motion Injury

The most common types of repetitive motion injury are *carpal tunnel syndrome* (wrist) and *cubital tunnel syndrome* (elbow). Typically, these conditions arise in the workplace where manual tasks involving the corresponding muscle groups are overused, leading to swelling and pressure on the median nerve or ulnar nerve, respectively. There also are a wide variety of tendon injuries or inflammations from similar overuse. At times, physical signs point conclusively to the underlying medical pathology, but often they are equivocal. Where pain persists even after repetitive motion tasks have been eliminated, psychological factors have been implicated along similar lines to other chronic pain syndromes (204). In addition, some studies have shown that along with localized pain a variety of generalized symptoms can be associated, and are seen in somatoform (psychosomatic) disorders-among them, fatigue, sleep disturbance, dizziness, nausea and gastrointestinal disturbance, difficulty in concentration, memory impairment, anxiety,

depression, and headache (204). There is also evidence that repetitive motion injury complaints are associated with the availability of disability payments. For example, at one time repetitive motion injuries of the upper extremities affected large numbers of Australian workers (up to 30% in some settings), until the diagnosis was no longer deemed legitimate and disability benefits were curtailed (205). There have also been concerns at times that surgical intervention in these injuries is complicated by a poor outcome due to psychological factors, and then by the additional damage from surgical invasion and scarring. Not infrequently, patients will have had multiple surgical procedures without significant improvement and with gradual entrenchment into chronic pain and invalidism.

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Whiplash

Whiplash injuries are the most common pain problem resulting from motor vehicle accidents (206). Although the vast majority of these patients recover within a few weeks, a substantial proportion do not (14% to 42%, depending on the study); and 10% report constant, severe long-term pain (207). Although a number of factors have been studied with regard to predicting which patients will develop chronic pain, psychological variables, higher levels of somatization, sleep difficulty, and helplessness appear to be associated with long-term symptoms and disability (208)(209)(210). There have also been, in recent years, a number of large population-based studies of potential prognostic factors. Two in particular are quite provocative. First, in Lithuania, a survey of all motor vehicle accidents during a particular time period showed a very low incidence of persistence whiplash syndrome; it was theorized that this was due in part to the fact that most drivers did not have personal injury insurance and, so, the likelihood of disability compensation was remote (211). The second study, conducted in Saskatchewan, Canada, showed a striking decline in whiplash injury, coupled with improved prognosis for the condition, following a change to the tort compensation system for traffic injuries in 1995, which disallowed future payments for pain and suffering (212). These psychological and socioeconomic variables should, again, point to the need for a comprehensive evaluation of such patients, one that includes attention to nonmedical/nonneurologic forces.

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Neuropathic Pain Disorder

As described above, pain is broadly characterized, based on its etiology, as either *nociceptive or neuropathic*. In nociceptive pain, tissue damage triggers a chemical or inflammatory reaction in tiny sensory nerve endings known as nociceptors. This reaction transmits the message of pain first to the peripheral and then to the central nervous system. As long as the noxious stimulus persists at the nociceptor site, the message continues to be transmitted (213). Neuropathic pain, on the other hand, may start with an initial nociceptive response to trauma but continue even after the tissue damage has healed; it appears to be generated from within the peripheral and central nervous systems, including the autonomic nervous system (214). This type of mechanism has been linked to commonly found conditions such as diabetic neuropathy, postherpetic neuralgia, trigeminal neuralgia, phantom limb pain, and others (215). It also appears to be a mechanism that can be responsible for more nonspecific and persistent pain syndromes that have some common features but otherwise cannot be explained by readily demonstrated medical pathology. While the concept of neuropathic pain is no longer in doubt, the diagnosis is made mainly on the basis of characteristic symptoms and signs apparent on physical examination. The symptoms include tingling pain, numbness, and increased pain with touch (16). The signs are allodynia (a painful response to light-pressure stroking), hyperalgesia (an exaggerated painful response to a painful stimulus- e.g., a safety pin), and decreased sensation to pin prick (16). Assessment tools have been used to address both symptoms and signs in a formal fashion (e.g., Self-Administered Leeds Assessment of Neuropathic Symptoms and Signs-S-LANSS), but the validity of this and, by inference, other screening tools has not clearly been demonstrated (216)(217). Yet, clinicians are often quick to assume that nonspecific and otherwise unexplainable pain syndrome must represent neuropathic pain, for which fairly aggressive anticonvulsant treatment is typically employed. It is likely that psychological factors play a role in at least some of these cases, either as secondary complications of chronic pain or as factors in its onset and maintenance. It is not uncommon to see false positives diagnosed as neuropathic pain, as well as other chronic pain syndromes, in which a recognized diagnosis is adopted clinically on spurious grounds.

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ASSESSING THE REASONABLENESS OF TREATMENT

Although the treatment of chronic pain is rarely in the hands of psychiatrists alone, and usually is not solely focused on psychological factors, psychiatrists may be in a position to assess the reasonableness of treatment because of the impact of psychological factors on the prognosis. Treatment of chronic pain is always challenging, and as months of pain accumulate, the prognosis becomes more guarded. In virtually every community there are professionals from various disciplines who undertake this treatment. Often, claims of success are not supported by validated research on the effectiveness of the treatment, or those successes are short lived. Even when there has been initial improvement, it may not be from the therapeutic methods employed but from the hope and promise communicated to the patient. It is also troubling to track how chronic pain patients move from one treatment resource to another, exhausting each in turn, at the same time incurring substantial costs for treatment. At times, the treatment proposed is no different from those the patient has already tried; all that is new is the treatment provider, who may implement what seems to be an innovative formulation for the pain problem. Along the way, patients frequently become disillusioned, deconditioned, and dependent (22).

Many of these treatment failures can be explained by the absence of two fundamental elements for successful treatment: the patient's *ownership* of the problem and *coordinated care*. Ownership means that the patient must assume an active and optimistic role in his or her own rehabilitation, even if cure is not possible. Rarely will restorative work succeed without this. The traditional medical model of diagnosing disease and supplying necessary treatment does not work well in chronic pain. Coordinated care means that the treatment plan and the various treatment providers who participate in it are unified in their approach. A multidisciplinary format is typically the best approach, and is needed both in the assessment of the individual as well as throughout the treatment process. Even with good communication between treatment providers, fragmentation can easily occur. One reason is that the treatment providers may have different perspectives on the approach, even when treatment goals have been clearly defined. Fragmentation can also occur when the treatment providers do not communicate with each other; and, inadvertently, some providers may emphasize or minimize aspects of the pain problem differently from other providers. This is especially likely when treatment is not going well. Here, a common tendency of the patient is to "split" treatment providers; the patient, consciously or unconsciously, plays them off against each other. A psychiatrist who is asked to assess the reasonableness of treatment may, therefore, focus in the first instance on whether the two fundamental elements for success, ownership and coordinated care, are present. In addition, psychiatrists may be able to address specific pain treatment methods that can be influenced directly or indirectly by psychological factors.

[Table C, Treatment of Chronic Pain](#), outlines typical treatment methods used in chronic pain conditions. The following sections provide brief descriptions of those treatment methods, together with how they may have relevance in a psychiatric assessment.

Pain treatment can be divided into three categories: physical treatment, pharmacotherapy, and psychological treatment. With regard to physical treatment and pharmacotherapy, two important considerations are necessary. The first is, again, whether or not the patient has accepted ownership of his or her pain problem and has taken an active role in the rehabilitation process. This is closely tied to the concept of self-efficacy described earlier. The more patients expect an external solution to their pain, rather than recognizing they will need internal resources as well, the less chance there is for success of any physical treatment. Therefore, two considerations come into play in understanding the various types of treatments described below. First, in assessing the potential effectiveness of any treatment process, it is paramount that psychiatrists keep in mind the overriding issue of the patient's perspective of the treatment—as an external versus internal solution. The second consideration, which is clearly within the province of a psychiatric assessment, is the presence of psychological factors that play an important and/or major role in the chronic pain condition. The greater the contribution of those psychological factors, the lower the chance that these treatments alone will be effective.

Table C. Treatment of Chronic Pain

Physical Treatment

- Physical Therapy
- Manipulative Medicine
- Anesthesiologic Pain Management
- Acupuncture and Acupressure

Electromedicine
Spinal Cord Stimulation

Pharmacotherapy

Anti-inflammatories
Opioid Analgesics
Muscle Relaxants
Anticonvulsants
Psychiatric Medications
Placebo

Psychological Treatment

Cognitive Behavioral Therapy
Relaxation and Mindfulness
Biofeedback
Guided Imagery
Hypnosis
Traditional Psychotherapy

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Physical Treatments

Physical Therapy

Physical therapy is probably the most common initial treatment method used with pain patients where more aggressive surgical treatment is not indicated. The general objectives of physical therapy were outlined years ago, although specific variations have evolved over time and the approach has diversified (218). Ultimately, the goals of physical therapy are to gain control of symptoms and improve functioning. Not uncommonly, chronic pain patients will have undergone numerous rounds of physical therapy with one or more therapists, and they will often complain that the therapy made their pain worse. Therefore, in a psychiatric assessment, where physical therapy has become protracted, indefinite, or repeatedly ineffective, there should be a greater level of scrutiny for the presence of psychological factors, or at least lack of ownership for the pain problem on the part of the patient.

More recently, there has been an interest in ordinary or light-resistance *exercise* for the treatment of chronic pain. For example, light-resistance exercise without aggressive physical therapy can have a statistically significant level of alleviation in the intensity of both headache and neck symptoms when combined with limited guidance and monitoring (219). Also, in chronic back pain patients, the combination of ordinary exercise and cognitive intervention has been shown to be as effective as a surgical fusion of the back (220). Since more intense physical therapy presumably accelerates the effects of exercise alone, these studies may show that the variable that leads to success may be cognitive outlook and active participation in rehabilitation, rather than formal physical therapy.

Manipulative Medicine

Closely akin to physical therapy are treatment procedures that can be labeled as *manipulative medicine*; these are represented by three main specialties: osteopathic medicine, chiropractic, and therapeutic massage (22). Although their origins share many common elements, their theoretical basis and the specific techniques employed can differ markedly, as follows:

In osteopathic medicine, the goal is to increase circulation so that the body can heal itself more effectively. This is accomplished by manual pressure on soft tissues, as well as spinal manipulation.

In chiropractic therapy, the principle is one in which a manipulatable lesion, known as subluxation, is identified within the spinal column and thought to cause impaired joint function and joint mobility. Chiropractors will apply a passive manual maneuver in which joints are carried beyond their normal physiological range for a brief sudden thrust, which is said to lead to greater joint mobility.

Therapeutic massage is another method of loosening soft tissue structures, increasing circulation, and soothing muscular spasm.

All of these manipulative medicine therapies can have a legitimate place in chronic pain treatment. Regardless of which one is chosen, the issue remains the same: that the core elements of patient ownership and coordination of care are maintained.

Anesthesiologic Pain Management

In the last 15 to 20 years, *anesthesiologic pain management* has taken a central role in chronic pain treatment. Many anesthesiologists have had advanced training in this field, and can add board certification in pain management. Much of their work involves therapeutic injections and blockades (22). In the hands of a skilled practitioner, dramatic results can be shown. Typically, injections are given into the epidural space covering the spinal cord and exiting spinal nerves, the nerve roots themselves, facet joints, and trigger points. Both steroids and anesthetics are used. Success depends on the method, the accuracy of the injection, and the underlying pathology of the patient. In epidural injections, nerve root blocks, and facet joint injections, the underlying pathology is thought to be deep in the nerve and connective tissue structures of the spinal segment. In trigger point injections, the pathology is thought to be in the myofascial (muscle and fascial covering) unit, and is more superficial. In trigger point injections, saline alone can be used. In principle, therapeutic injections and blocks are merely facilitating devices that help patients move sufficiently beyond their pain so that they can participate in the rest of the rehabilitation process.

Anesthesiologists also often assume the primary role in pharmacotherapy (see below), including narcotic and nonnarcotic medications. Here, too, if the patient harbors too great an expectation that these external solutions will solve his or her pain problem, results can be disappointing.

Acupuncture and Acupressure

Acupuncture and acupressure are treatments derived from oriental medicine, and based on an entirely different physiological orientation than that of Western medicine (221). Through the use of needles or manual pressure at specific points on hypothetical body meridians, a therapeutic benefit to various bodily systems is proposed. The mysterious nature of these techniques can provoke high expectations and a placebo response (see below), but more recent studies do show efficacy and safety in the use of acupuncture for certain chronic pain conditions (222)(223). Some studies have suggested that acupuncture may stimulate the production of the body's own opioid substances (endorphins). For example, where opiate antagonists (e.g., naloxone) are used, or where there are defective opiate receptors, the beneficial effects of acupuncture seem to be canceled (224). Other studies have compared acupuncture and acupressure to techniques that interface with gate control mechanisms in the peripheral and central nervous systems (224). Further research will be needed to determine whether these treatment methods have lasting value.

Electromedicine

Electromedicine has a long historical tradition in the treatment of pain disorders (22). The ion-contained fluids of the body can serve as a conductive medium through which therapeutic electrical stimulation has its effects. A number of electromedical methods continue to be popular, and have strong proponents; these methods include alpha stimulation, cranial electrotherapy stimulation, galvanic stimulation, and microcurrent electrical therapy (225). Another electromedical approach, transcutaneous electrical nerve stimulation (TENS), is through a device; when attached to the skin, in theory, it stimulates fast-conducting sensory fibers, which in turn inhibit slow-conducting fibers that carry pain impulses. This, too, is based on the hypothesis of gate control mechanisms that can increase or decrease the pain response (22).

One analogous electromedicine approach that has become widely used in pain centers is spinal cord *stimulation* (SCS) (226). This method is also based, in part, on the gate control theory of pain. Electrical stimulation is through implanted devices in the dorsal column of the spinal cord itself. Earlier studies indicated that pain in the extremities is more responsive to SCS, but more recently it has been used on various kinds of pain, including back pain (227). Frequently today, SCS is thought of as the last resort to treatment of chronic pain, where there has been little effect from other treatment methods. Its use has been controversial, however, largely due to its substantial cost, which can easily exceed \$50,000. Moreover, surgical complications with SCS are not uncommon, and the benefit may be not much more than 50% pain relief in 50% of subjects. As a result, SCS is usually done in stages: a trial implantation, if effective, can lead to the permanent implantation. In addition, most centers using SCS will require a psychiatric or psychological evaluation, either of which has the potential to be misleading. In the first instance, patients

are often so hopeful about SCS, and know that without a positive response in the trial implantation they will not have a permanent implant, so that the potential for early placebo effect is high. The psychological evaluation can be misleading, too, because patients will be reluctant to discuss psychological factors for fear that doing so would exclude them as a candidate for SCS. This results frequently in superficial psychiatric or psychological evaluations that are more perfunctory than probative. Ultimately, of course, the decision for SCS is a medical/surgical one; but where psychiatrists are involved in the assessment of its reasonableness, very thorough evaluations should be the rule.

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Pharmacotherapy

Contemporary pharmacotherapy of chronic pain is through the use of four groups of medications: analgesics, muscle relaxants, anticonvulsants, and psychotropics. The analgesics are of two types, anti-inflammatory drugs and opioid (narcotic) analgesics. Both groups of medications have been helpful to patients, in combination with other therapeutic approaches. At the same time, both groups have limitations in the maximum expected benefit, adverse side effects, and complications of prolonged use.

Anti-inflammatory Drugs

The *anti-inflammatory* drugs are divided into two categories: nonsteroidal and steroidal. As their name implies, they have the effect of reducing inflammation from chemical processes that are taking place at peripheral nerve sites; that is, they act where the tissue injury has taken place. They also may have a central nervous system effect, which is still poorly understood. By reducing inflammation, and thereby pain, patients are able to move more freely. This, then, hopefully, enables patients to participate more fully in their other therapies.

Nonsteroidal drugs are usually the first line of analgesic treatment in an acute pain problem. Prolonged use in chronic pain, however, has a host of potentially troublesome and dangerous side effects. These include gastrointestinal disturbance, bleeding ulcers, headache, dizziness or drowsiness, ringing in the ears, kidney damage, and various skin eruptions. Steroidal drugs, which also have a remarkable capability to inhibit the inflammatory process, tend to be a second, more aggressive, line of analgesic treatment. Most often they are used in a short trial. Extended use of steroids is fraught with many serious side effects, spanning multiple organ systems (22)(228).

Analgesics

Opioid analgesics have been known for centuries to have a beneficial effect on pain. These drugs primarily act in the central nervous system and have their effect by binding to naturally occurring opiate receptors. These receptors are normally the places where the body's own opiate substances, enkephalins and endorphins, bind. The use of opioids has gone through an evolution in pain management. Earlier, it was felt that their potential for dependence, tolerance, and rebound limited them to acute pain conditions or to chronic pain in a patient suffering with a terminal illness such as cancer. Physicians were duly apprehensive about ongoing use of opiate analgesics in benign pain conditions, and licensing boards would severely scrutinize such use as well (229)(230). In recent years, however, there has been a surge of pressure on physicians to prescribe opioids on a long-term basis even for benign chronic pain, concomitant to a strong movement of patient advocacy for the right to have one's pain relieved (231). The earliest excursion into this type of treatment included a number of guidelines to prevent inappropriate and dangerous use of opioids (232). Some of these, as well as other important principles, are outlined in [Table D, Guidelines for Long-Term Narcotic Treatment](#).

Unfortunately, the routine long-term use of opioids has now become the rule more than the exception at pain centers, with a primary focus on purported pain relief rather than functional improvement (233). This does not mean that opioid therapy is without benefit. In fact, there are many patients that can receive opioids in a controlled fashion indefinitely without serious problems and with much improved functioning. However, newer studies have raised concerns that the diminished effectiveness of opioid use may be related to underlying psychopathology (234)(235); that the long-term use of opioids can result in abnormal pain sensitivity and rebound pain (236); that genetic factors may be related to opioid analgesia, tolerance, and dependence (237); and, most importantly, that long-term opioid users report significantly more moderate/severe or very severe pain, poorer self-rated health, and lower quality of life scores (238). There is, additionally, a close association between opioid use and low levels of physical activity and employment,

and high levels of healthcare utilization (239). From a psychiatric assessment standpoint, therefore, where an individual is receiving long-term opioid treatment for benign pain, attention should be drawn to the level of functional improvement, if any, provided by the opioids; the level of apathy and dependency; secondary sedative and cognitive effects of the opioids; the presence of persistent psychopathology; the possibility of rebound effects; and addictive behavioral patterns in the patient's and the patient's family history. Because of these issues, the use of long term opioids should include a contract with the patient that delineates what would constitute a violation, abuse, or misrepresentation. Also, long term use of opioids should include some type of random drug testing for verification of contract compliance.

Muscle Relaxants

Muscle relaxants are also used in pain control, and are believed to work somewhat similarly to anti-inflammatory drugs; that is, by relieving pain they can promote greater mobility and, thereby, raise the potential for rehabilitation (240)(241). Muscular spasm, local pain and tenderness, and limitation of motion all have been shown to decrease with the use of these drugs. They are usually well tolerated in patients, but can cause side effects of sedation, dizziness, dry mouth, and gastrointestinal complaints. Particularly in combination with other pain-relieving drugs that have sedative effects, they can cause fatigue, apathy, and cognitive impairment; consequently, the impetus needed for investment in the rehabilitation process may not be there.

Anticonvulsants

Anticonvulsants are now used ubiquitously by physicians and pain specialists for the treatment of chronic pain. Hypothetically, their effect is for neuropathic pain (see above), but since neuropathic pain may present simply as nonspecific findings, they are inevitably prescribed, at least on a trial basis, for most patients who suffer with chronic pain. Examples of these agents include gabapentin, valproic acid, carbamazepine, and lamotrigine (241). From a psychiatric standpoint, it is known that the same medications are used in the treatment of mood disorders unrelated to pain (e.g., bipolar disorders, mood disorders, complicated depressive disorders). Although there seems to be evidence for at least some of these agents to be effective in chronic pain, the overlap with mental disorders may mean that there is a primary psychiatric effect operative, as well.

These drugs frequently are used in very high dosages, so that in patients who have signs of sedation, anergia, or cognitive sluggishness, a careful assessment of their benefit should include objective evidence of functional improvement and/or significant pain relief, along with consideration of any deleterious effects from sedation.

Psychiatric Medications

Psychiatric medications are also frequently used in the treatment of patients who have chronic pain. This is not only because of obvious comorbid mental disorders, but also because there has been a direct analgesic effect noted from some of these agents. In particular, antidepressant medications (e.g., amitriptyline, nortriptyline, desipramine, venlafaxine, duloxetine) are now frequently used to relieve pain, often in smaller doses than for depression, and can be quite effective (241). Also, anti-anxiety agents may be used (e.g., clonazepam, diazepam), which have a muscle relaxant effect, as well as a beneficial influence on autonomic arousal that helps maintain pain symptoms. However, both antidepressant medications and, more so, anti-anxiety agents may have their own sedative properties, which can lead to a serious additive effect with other pharmacological treatment. For example, when a pain patient is being prescribed large doses of opioid analgesics, muscle relaxants, anticonvulsants, and psychiatric medications, the additive effects can be dramatic. Thus, the assessment of their usefulness will be most revealing when attention is directed at the patient's functioning, rather than analgesia. When pain relief is the only variable measured, a vicious and destructive cycle of increasing medications that promote invalidism can easily be overlooked.

Placebo and Nocebo

A discussion about pharmacotherapy would not be complete without some attention to the phenomenon of *placebo*. Of course, placebo effects are not only related to medication but, as discussed above, can include any type of pain treatment. There is a great deal of controversy about whether placebo effects are real, and whether a placebo can cause a true biological change (242). Commonly, it is thought that where a placebo effect does occur, the underlying disorder must be either psychological or fabricated. These assumptions do not appear to be true. Mounting evidence shows, for example, that placebos do produce brain activation, such that there may be very real effects in the endorphin network of the nervous system (243). Other studies, however, indicate that the effects are more consistent with desire and expectation, rather than

endorphin mechanisms (244). It has also been shown on functional magnetic resonance imaging (fMRI) that placebo analgesia is accompanied by reductions in neural activity in pain-related areas of the brain during the time of stimulation and pain processing (245).

There may, in fact, be many types of placebo responses (246). Placebo effects may center on the surrounding psychosocial context in relationship to an individual's personal, physiological, and psychological makeup. This may have practical significance in ordinary treatment situations where manipulation of desire and expectation can produce results regardless of which treatment is used. The danger, in any case, is that the effects of placebo are typically short lasting. Therefore, treatment providers who are extremely enthusiastic about their methodology may initially have success in treatment, thereby seeming to validate the treatment method. But long-term value of the treatment is another matter, and personal, psychological, and psychosocial variables that significantly impact pain can be overlooked. It is no longer accepted that some individuals are placebo responders and others are not; but the effect of placebo can be robust, at least in the short term. The reciprocal issue, however, is even more provocative; namely, if desire and expectation can affect a therapeutic placebo response, and this is even measurable in brain functioning, do desire and expectation also affect the intensity and course of chronic pain?

In fact, there is such a counterpart, known as the *nocebo* effect. This is a negative equivalent in which there is a worsening of symptoms after treatment when an individual expects a negative outcome from that treatment (247). Interestingly, though, the mechanisms of action in nocebo are not just the reciprocal of that proposed in placebo. For example, it is possible to block the endorphin response in placebo with an opioid antagonist such as naloxone, but it is not shown that naloxone increases the nocebo effect. Instead, nocebo appears to be related to a separate network in the nervous system, endogenous cholecystikinin (CCK) (248). So, when a CCK antagonist is given, the nocebo response is blocked and the placebo effect is enhanced. This suggests that there may be a balance between the endorphin and CCK mechanisms that is responsible. In any event, research data shows that nocebo suggestions are capable of producing both hyperalgesia (i.e., an increase in pain sensitivity) and allodynia (i.e., the perception of pain in response to innocuous stimulation) (248). Therefore, in pain conditions where both hyperalgesia and allodynia are demonstrated, but without other objective medical findings, the role of expectation cannot be minimized. Similarly, after a period of treatment failure, the expectation of continued pain and misery may not be without its impact.

Table D. Guidelines for Long-Term Narcotic Treatment

1. Patients must have had a complete screening for correctable organic pathology and must be failing an intense and adequate conservative multidisciplinary treatment approach at functional improvement.
2. Patients must have had a thorough psychological evaluation to identify contra- indications to opioid use such as psychiatric disease, markedly unstable social conditions, sociopathy or addiction potential, lack of motivation, or the absence of any functional direction.
3. Patients should be prescribed opioids in a strictly controlled and monitored fashion that is directly linked to continued functional improvement toward a specified objectively measured level.
4. Treatment should include informed consent, a single practitioner taking primary responsibility, achievement of at least partial analgesia at low initial doses, regular physician contact, and elimination of the program when there are signs of drug hoarding or other inappropriate drug seeking behavior.

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Psychological Treatment

Psychological treatment for chronic pain is, correctly, a regular adjunct to a multidisciplinary approach. Often, standard psychiatric/psychological treatment methods appear to be appropriate because of the emotional distress and comorbid mental disorders that accompany chronic pain. However, it is difficult to

measure the true effectiveness of psychological treatment in chronic pain and to design credible randomized control trials to show its success. That said, several types of psychological treatment are known, and have shown effectiveness outside of psychiatric medications alone.

Cognitive Behavioral Therapy

Cognitive behavioral therapy has been studied the most, and includes those methods that aim to decrease maladaptive cognitions and increase adaptive coping to the pain experience (249)(250). As part of the treatment, and consistent with the types of psychological factors discussed earlier, the goal is to decrease catastrophizing, increase perceived control over pain, and improve functional activity (251). Both brief and longer-term cognitive behavioral therapy has shown effectiveness in a variety of chronic pain conditions (252). A word of caution is in order here, however, because focusing on individual parameters of pain response, such as catastrophizing or self-efficacy, may sometimes lead to devaluing the more complex psychosocial context of a chronic pain patient, and in turn to a superficial formulation of the psychological problem (253). Notably, even though cognitive behavioral therapy has produced significant results, substantial numbers of chronic pain patients who have undergone this type of treatment remain distressed and nonfunctional. This should make clear the complexity of these problems. An interesting recent study, for example, showed that another less examined variable, the role of values, can be an important predictor of success with cognitive behavioral therapy. Here, the investigators found that higher success at living according to one's values, and fewer discrepancies between one's sense of importance and success, were correlated with less physical, psychosocial, and "other" disability, and with less depression, depression-related interference with functioning, and pain-related anxiety (254). The authors indicate that these results are consistent with the interpretation that when values function as guides for action, patients with chronic pain experience have relatively higher levels of daily activity and better emotional functioning. Such findings are consistent with studies that show better results in treatment when chronic pain sufferers struggle less for control over pain, continue to engage in life activities in the presence of pain, and have found ways to accept their pain. This may now be enhanced by an additional focus on value-based treatment (254).

Relaxation and Mindfulness

Another frequently used psychological treatment incorporates techniques of *relaxation and mindfulness*. This comes from the understanding that chronic pain sufferers become overly focused on their pain, think in negative terms about their situation, and become fixed in recurrent patterns of unsuccessful struggling with pain. Relaxation and mindfulness are intended to reduce the contribution of emotional distress on chronic pain through a practice of present-focused and behaviorally neutral awareness (255). The goal of relaxation and mindfulness is not to alter the content of what is experienced but to change how it is experienced and the influences it exerts on behavior. This treatment method works on the principle that, in chronic pain, changing what is felt is much more difficult than changing one's behavior in relationship to what is felt (256). Recent studies have demonstrated that the use of these techniques has a significantly positive effect on general health perceptions, emotional well-being, cognitive functioning, sleep, pain, and family and role limitations (257). In general, there appears to be far better functioning when such techniques were followed. Practically, the introduction of relaxation and mindfulness techniques is quite common, but their effectiveness relies greatly on the patient continuing to practice those techniques regularly. Therefore, identifying that a patient has been taught those techniques is not enough to conclude that they were or were not effective, without knowledge of how faithfully they were actually practiced.

Biofeedback

Closely akin to relaxation and mindfulness is *biofeedback*, which increases awareness and voluntary control over a patient's arousal states by a direct measure of physiological functioning, which is communicated back to the individual (22). The most common forms of feedback are electromyography (EMG), which measures muscle activity; electroencephalography (EEG), which measures brain waves; thermal, which measures temperature and blood supply; and galvanic skin response (GSR), which measures sweat gland activity (258). Feedback, which can be auditory or visual, creates awareness at a much lower threshold than would otherwise be possible. With time and practice, the degree of change becomes apparent on a subjective level, too. Without this microfeedback, many patients would give up trying to alter their physiological functions, since they would not know whether their efforts were making a difference. Thus, micros successes create incentive for further practice and skill development.

The use of biofeedback in pain conditions is a natural development because of the overarousal states that so frequently are present. These states are closely linked with emotions of anger, fear, frustration, and agitation. It has been shown that biofeedback does have a significant effect on lowering muscular tension, a

reduction in need for medication, and an increase in levels of activity (259) (260). One of the pitfalls of this treatment, however, is that it may reinforce the perception of an external solution to the pain patient, since reliance on an instrument can lead a person away from the necessary mental attitude of acceptance and self-mastery. Biofeedback also lacks the deeper philosophical stance of relaxation and mindfulness training, which includes a greater awareness of one's self and one's body as a whole, and the capabilities that one retains in spite of chronic pain.

Guided Imagery

Guided imagery is a strategy for pain treatment that is based on the hypothesis that mental representations have a profound influence on physical processes, emotions, and behavior (257)(261). Visualization is a necessary component of planning for future projects or entering a new situation, even beginning some new physical activity. Visualization and imagery are thought to have some effect on disease processes, perhaps through a psychobiological mechanism, and therefore could be utilized to help deal with pain more effectively. They operate by helping patients to relax muscles by visualizing them relaxing, diverting attention from pain to something more pleasant and distracting, attenuating the negative reactions the patient has to the painful sensory experience, and replacing negative meanings that are attributed to pain with positive ones. For example, visualizing a tight band that is loosening or falling away, or muscles that are becoming loose and limp, may actually diminish the intensity of the painful sensory experience that is attached to the visualization. Patients can often find their own images that work best for them. In its simplest model, seeing pain going away can actually help it go away, or at least to become less prominent in the person's life.

Hypnosis

For over one hundred years, *hypnosis* has been used as a means of pain relief (262). The specific mechanism by which hypnosis works in that regard is not clear, but psychological as well as neurophysiological mechanisms are now proposed. Hypnotic induction methods are very similar to relaxation and mindfulness, but in hypnosis there appears to be another level that is operative. Recent studies have also shown that highly hypnotizable subjects show greater response to analgesic suggestions following a formal hypnotic induction than in the waking condition (263). Long-term beneficial effects of hypnosis have not been shown, but in situations where an individual is taught to practice autohypnosis, it may help reinforce other treatment methods. Because hypnosis carries an impression of being a mysterious phenomenon, it is easy for patients to believe in this external solution, which, in the end, may have little gain.

Traditional Psychotherapy

Finally, *traditional psychotherapy* is often used in patients who have chronic pain, because pain treatment specialists will refer an individual who is emotionally distressed to someone for counseling and/or psychiatric/psychological intervention. While this may appear to be appropriate-and where there are significant comorbid mental conditions, it probably is helpful-psychotherapy by itself has not been shown to have a substantial beneficial effect in pain disorders. In fact, in most cases, psychotherapists may be unfamiliar with psychological treatment methods that are helpful in chronic pain, hence may not realize how their treatment can inadvertently reinforce pain and invalidism. It is not unusual to see a pain patient continuing psychotherapy at an intense level for years with absolutely no significant change in the pain status. This does not mean that psychotherapy has no value, and it may be subjectively helpful to the patient, but it is not well suited to the type of therapeutic approach that is needed in these complex cases.

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IMPAIRMENT AND DISABILITY

Psychiatrists are frequently called upon to provide opinions on impairment and disability, but pain disorders present unique issues. In the first place, impairment is defined in the *AMA Guides* as a significant deviation, loss, or loss of use of any body structure or body function in an individual with a health condition, disorder, or disease (16). It appears, therefore, that rating impairment addresses bodily or physical limitations that are more in the province of medical, rather than psychiatric, evaluators. Disability is defined as activity limitations and/or participation restrictions in an individual with a health condition, disorder, or disease. Here, the language does not appear as restricted to bodily limitations; but since disability is a product of impairment, physical limitations may be implied. Indeed, in earlier editions of the *AMA Guides*, rating mental impairment was restricted to qualitative, not quantitative, ratings because, it was said: "There are

no precise measures of impairment in mental disorders. The use of percentages implies a certainty that does not exist." (15). The sixth and most recent edition of the *AMA Guides* modifies that position, but allows that only impairments for selected well-validated major mental illnesses are considered (i.e., mood disorders, anxiety disorders, and psychotic disorders) (16). It specifically indicates which mental disorders are not ratable, and these include "psychiatric reaction to pain" and "somatoform disorders" (16). Instead, the impairment rating for a physical condition, which presumably is provided by a medical evaluator, already should take into account both the pain and the psychological distress associated with the physical impairment. It would seem, then, that psychiatrists should not be involved at all in impairment ratings of pain disorders. For example, if a patient has two herniated lumbar discs at multiple levels, and on the basis of physical criteria is rated as Class 3, 15% to 24% whole-person impairment, a severe pain impairment could be included as a modifier within a net adjustment formula, to result in a final rating of 19%. The pain-related impairment is determined by using the Pain Disability Questionnaire, which is made up of a functional status component and a psychosocial component (264). It is important to note that in this example the pain disorder was accompanied by objective findings of injury; therefore, the pain-related impairment does not by itself add to the percentage of whole-person impairment, but only indirectly within the net adjustment formula. Here, clearly, a medical evaluator would be providing the impairment rating, and modifying it according to the formula that takes into account pain-related impairment.

At times, however, psychiatrists may become involved in the rating of pain disorders when specific circumstances present. The first of these is when a pain disorder is not accompanied by objective findings. For example, if a patient suffers a cervical sprain (i.e., whiplash), but radiological evidence shows no abnormalities, the physical examination is normal, and the symptoms are only subjective. From a purely physical standpoint, this would lead to a Class 0, or 0% whole-person impairment. No pain-related impairment as a modifier would be included. This condition, psychiatrically, might correspond to a Somatoform Disorder—that is, Pain Disorder associated with psychological factors, in which the psychological factors are playing the major role. Here, the *AMA Guides* would allow an impairment rating based directly on the pain-related impairment (16). Again, this would be calculated using the Pain Disability Questionnaire, but only within a range of 0% to 3% whole-person impairment. So, if the whiplash patient would score a moderate degree of pain-related impairment on the Pain Disability Questionnaire, using the table provided in the *AMA Guides*, 6th Edition (16), the resulting whole-person impairment would be only 1%. The reason behind the low cap of 3% is articulated in the *AMA Guides*, 6th Edition, and is based largely on the problems of reliability and validity of any pain-related impairment assessment. Some opponents of pain-related impairment have even argued that it should be given no percentage rating because it is so dependent on subjective factors (16). In situations where pain is not accompanied by objective findings, it is again arguable that psychiatrists could provide an impairment rating, especially where psychological factors appear to play the major role.

The second instance where a psychiatric opinion may be sought is where pain disorders are accompanied by a comorbid mental disorder such as a Mood Disorder or an Anxiety Disorder. Taking the above example of a patient with two herniated lumbar discs who may warrant a 19% whole-person impairment medically, if the patient has developed a secondary Major Depressive Disorder, he or she could warrant an additional impairment rating as defined in the "Mental and Behavioral Disorders" chapter in the *AMA Guides*. The difficulty presented, however, is that symptoms comprising the psychosocial component in pain-related impairments can overlap with symptoms of the mental disorder. So, it is not unusual for patients with a pain disorder to have difficulty with sleep, concentration, depression, tension, and anxiety, symptoms so closely tied to the pain itself that without it those symptoms would disappear. Most patients, in fact, will acknowledge that the only reason they have such symptoms is because of their pain. Should this, then, really be classified as a separate mental disorder, or simply as part of pain-related impairment? Often, in attempting to rate impairment for the mental disorder, psychiatrists will use the Global Assessment of Functioning Axis (GAF) in *DSM-IV-TR* (14). However, two issues arise with the use of the GAF. The first is that it specifically does not include impairment in functioning due to physical limitations. So, for example, when a patient appears to have serious impairment in social or occupational functioning that could lead to a rating of 50 out of 100, is it possible to accurately separate the degree of impairment that is due to the mental disorder alone, where, typically, the patient's predominant focus is on the pain and is the overwhelming reason that the patient sees himself or herself as impaired? The second problem using the GAF is that it provides only a means of reporting impairment, not one to actually assess it (265). For example, a GAF score in the range of 41 to 50 could note serious symptoms or serious impairment in functioning, but the scale does not help in making the assessment as to whether the person was in fact impaired and unable to function. Only limited objective information can be obtained through a psychiatric history and mental status examination, without a great deal of corroborating information about the patient's actual day-to-day function. Furthermore, to the extent that a psychiatrist may believe that a separate

impairment for a mental disorder is warranted, should that percentage be greater than that of the pain-related impairment, which is, typically, the main focus of the patient?

In summary, psychiatrists should be cautious when venturing into determinations of impairment and disability in pain disorders. In most cases, the determination is really a medical one. Where psychological factors or an independent mental disorder may warrant an impairment, care should be taken that the lines between physical and mental impairment do not become blurred.

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CAUSATION

The cause of pain disorders is an important consideration in litigation that involves these conditions, and is typically an element that the plaintiff or claimant has to prove in order to receive recovery damages. These claims are primarily of two kinds: personal injury and workers' compensation. This section first addresses general legal principles in both of these types of claims, related medical legal issues, and the role of psychiatrists in providing causation opinions.

Personal Injury Claims

Personal injury claims for pain disorders are usually brought on the basis of an intentional tort or negligence. In both, the threshold question is whether the defendant's conduct was a *cause in fact* of the pain (150(266)). A cause in fact is established by a simple but-for determination-that is, *but for* the defendant's conduct, the plaintiff would not have been harmed. Because intentional torts involve the willful acts of a defendant, little more is required in most cases. An alternative analysis is whether the defendant's conduct was a substantial factor in causing the harm (i.e., a necessary element); but even with that, causation may not be that difficult to prove. In negligence claims, on the other hand, there is an additional requirement to show *proximate cause*. In many ways this is merely a means of limiting the scope of a defendant's liability. For example, while there may be some causal connection between the defendant's conduct and the pain, the harm is too insignificant, remote, logically unrelated, or just beyond what a defendant should be held liable for. Traditionally, proximate cause centers on the question of whether or not the harm was foreseeable.

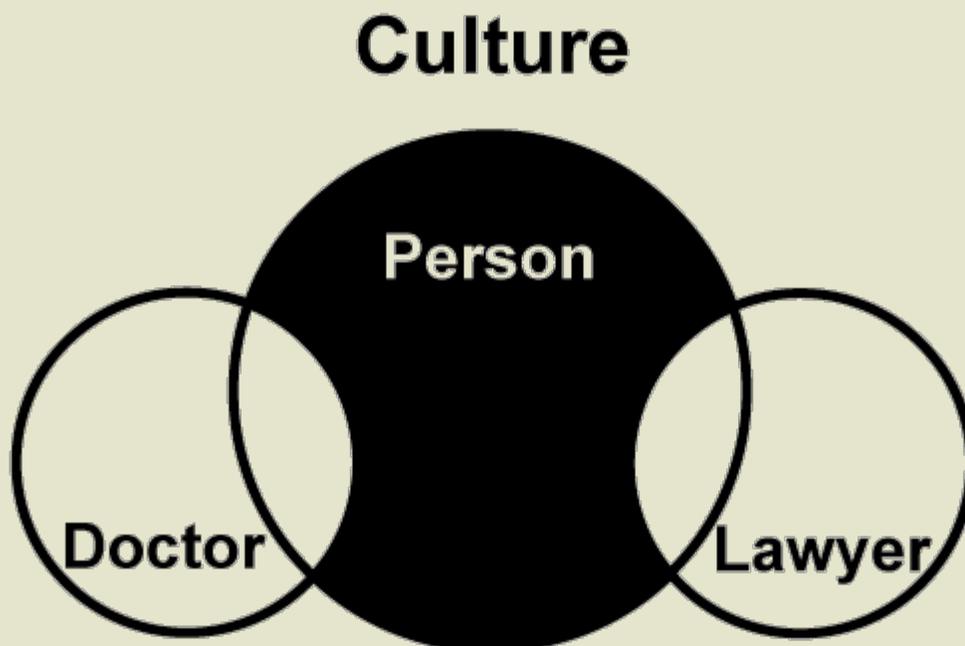
The chain of causation in personal injury claims can be broken by an *intervening cause* (266). This is considered a new force that can intervene to trigger the injury after the defendant's action and their consequences have come to conclusion; in which case, the responsibility now falls on the new intervening cause. However, there is a limitation by which the intervening cause must be unforeseeable and superseding. For example, if a plaintiff is injured and then unsuccessfully treated by a physician, the original defendant might still be liable because treatment for the condition is not guaranteed. Where pain disorders are so often subjective, there is a potential for physicians to use biased methods of diagnosis and treatment, which could reinforce or suggest an erroneous diagnosis. Lawyers for the plaintiff may also have a theoretical bias due to specializing in personal injury law, hence can, perhaps inadvertently, shape symptoms or reinforce invalidism that is more likely to lead to a higher damage award. In addition, cultural acceptance of certain pain conditions, as well as plaintiffs' own investment in legitimizing their condition, can have a powerful influence in suggesting a greater harm than the injury itself would have caused (150) (see [Figure B, Sources of Suggestibility](#)). In such instances, the question is, did the defendant's conduct only create an opportunity for suggestibility to become an intervening, superceding cause of the continuing pain disorder. Similarly, did the plaintiff's own actions or needs create avoidable consequences that should have been mitigated?

Courts also make a distinction between the nature of the harm and its extent. In general, a defendant is liable if he or she could reasonably foresee the nature of the harm done, even if the total amount of harm turns out to be quite unforeseeably large. This is closely tied to the concept of the *thin skull or eggshell skull rule*. Here, the defendant may have no reason to know of a particular susceptibility to pain that the plaintiff may have, but must take the plaintiff as he or she finds him or her (266). The difficulty, again, for someone who is demonstrating only subjective pain symptoms is that there could be unlimited possibilities of recovery when this rule is applied liberally. Unlike in pure mental damage cases in which a psychological stressor can be further scrutinized as to whether it would have severely distressed a reasonable person at the time of alleged injury, in pain disorders there typically has been a physical impact, which purportedly makes the claim more objective. Yet, if psychological factors are playing the major role, should the pain disorder be treated differently from other types of mental disorders where both are mostly subjective?

(267)(268).

Complicating the matter further is the ongoing controversy about how to classify psychiatric disorders. This has particular relevance when psychological factors in a pain disorder are playing the major role. In this instance, the pain condition is substantially a psychiatric disorder, and may not fit the causation models used for medical disorders. The history of medicine shows that a "true disorder" was initially established by the discovery of a discreet and unique cause (269). This type of thinking may have been rooted in infectious diseases where a clear etiological agent was identified. It may not have a counterpart in many medical conditions where the etiology is not yet known or is based on more than one factor, and may have little analogy to psychiatric disorders that are too complex to assign an etiology/cause. Even seeking a substantial cause varies with current scientific thinking and theoretical differences that can exist simultaneously. Therefore, to reiterate, causation in pain disorders may not follow a linear analysis.

Figure B Sources of Suggestibility



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Workers' Compensation

Many of the same issues described above with regard to causation for personal injury claims apply as well to workers' compensation; at the same time, there are some unique differences. In the first place, workers' compensation is a no-fault system so, in theory at least, the claimant does not have to show that the defendant inflicted injury either intentionally or negligently. Instead, it must be shown that the injury *arose out of and in the course of employment* (270). This is usually not as difficult to prove as a personal injury claim, but it also results in limited damages set by statute (i.e., a percentage of the worker's pay and the cost of related medical treatment). All U.S. states have workers' compensation statutes, and most federal employees are similarly covered. To reiterate, since a pain disorder typically arises from a physical impact and/or may be considered a physical manifestation, these conditions are purportedly more objective than a pure mental injury claim. Although the states vary as to how the general law of workers' compensation is applied, in general the injury must be shown to have a sufficient relationship with employment in order for benefits to be available (270). There are also variations with regard to the level of certainty required for an opinion on causation, as well as the requisite terminology used to communicate such opinions. It is important, therefore, to not assume that the ordinary meaning of *work-related* will apply or be sufficient.

In evaluating whether a work injury caused a pain disorder, it is also important to explore the potential impact of *personnel issues* (271). This becomes particularly critical where the pain disorder may present with symptoms that are greatly out of proportion to objective medical findings or to the nature of the injury itself. Personnel issues refers to administrative or personnel actions by the employer that surround the time

of the claimed injury and that can provide an independent reason for psychological factors that may affect a pain disorder or an incentive for a work injury claim. In most states, good-faith actions by an employer, even if stressful, are not sufficient grounds for a workers' compensation stress claim. But in pain disorders, the stress may be legitimized because there appears to be a physical injury. These personnel issues may be covert and come to light only as a consequence of a more in-depth evaluation of what has actually been occurring at the workplace. They include such areas as:

- Performance problems (e.g., poor productivity or quality of work)
- Personality disturbance (e.g., maladaptive or disruptive interpersonal behavior)
- Motivational issues (e.g., job dissatisfaction or malcontent)
- Employee misbehavior (e.g., harassment, pranks, or misappropriation)
- Employment insecurity (e.g., fear of layoff or termination)

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Related Medical-Legal Issues

A number of major medical-legal issues are related and need attention in any causation analysis of pain disorders.

First, in determining whether a medical condition exists, and its medical etiology, increased attention has been paid to the reliability of the scientific foundation for medical and, by extension, legal opinion. Known as *evidence-based medicine* (EBM), this looks to the conscientious, explicit, and judicious use of current best evidence in arriving at medical decisions (272). In spite of the great numbers of research studies published every year, good, controlled, and randomized clinical studies are not always easy to design; and some of the issues that arise with both mental and pain disorders may not lend themselves readily to those types of studies. Nonetheless, an effort should be made to rely as much as possible on the best scientific evidence available. In 1993, in *Daubert v. Merrell Dow Pharmaceuticals*, this principle led the U.S. Supreme Court to outline criteria that must be met for expert opinion to be admissible (273). Now known popularly as the "Daubert issue," which may require a separate hearing by the court, four specific criteria must be met:

- The methods on which the testimony is based are centered on a testable hypothesis.
- There is a known or potential rate of error associated with the method.
- The method has been subject to peer review.
- The method is generally accepted in the relevant scientific community.

In regard to pain disorders, there is extensive research by practitioners in various disciplines, but this often leads to polarization and to opposing schools of thought. This may result in:

Questions about whether a pain condition is satisfactorily recognized through objective scientific criteria (e.g., complex regional pain syndrome, or fibromyalgia)

Proposed methods of measuring a pain response in order to objectify the pain (e.g., thermogram, or fMRI)

Psychiatric and psychological methods of analysis (e.g., psychological tests that claim to show evidence of somatization)

Therefore, it is essential that the methodology used to derive opinions about pain disorders be scrutinized as to its accuracy and its limitations.

Another consideration in the analysis of causation is whether a particular injury is regarded as an aggravation of a preexisting injury even if not the original cause. Some jurisdictions distinguish between an aggravation that is temporary and does not actually change the underlying condition—that is, only exacerbation, which may not be compensable—and a permanent worsening of the underlying condition, which is compensable. A closely tied concept is one of *apportionment*, in which more than one cause to a pain disorder may be implicated, and the damage or compensation that is claimed must weigh the relative contribution of each (274). In medicine, multiple factors in the etiology of any disorder can almost always be implicated, yet the legal question is more narrow and requires a different though related analysis. It is necessary, therefore, to know the legal standard in the respective jurisdiction that needs to be addressed

when forming such opinions.

Mental as well as physical symptoms can be maintained by a process known as *secondary gain* (275). Unlike the unconscious conflicts that directly lead to mental conditions such as Conversion Disorder, which are known as *primary gain*, secondary gain refers to those perhaps unexpected environmental responses to symptoms or impairment that sustain the disorder by reinforcing it. Pain disorders are no different in being susceptible to this influence. Examples include financial reimbursement from a potential damage award or disability compensation (sometimes both), attention from family members that was heretofore lacking, or avoidance of less than satisfactory work conditions. Secondary gain not only influences the symptoms themselves but the reporting of symptoms by the patient. The history can, therefore, easily have elements of exaggeration and distortion. This is not necessarily a conscious process, but can, nevertheless, be a powerful one. Secondary gain is a significant potential factor to be considered in pain disorders, and what can cause and maintain them. Arguably, every pain disorder for which there is litigation could be assumed to involve secondary gain; but scrutiny as to the relative weight of psychological factors, the apparent motivation toward recovery, and the financial and personal circumstances of the individual may be helpful in assessing the actual degree of secondary gain.

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The Role of a Psychiatrist

For the most part, causation of pain disorders is a medical, not a psychiatric determination, at least initially. In the diagnosis of Pain Disorder associated with both psychological factors and a general medical condition, where the psychological factors are playing an important but not major role, then the psychiatric opinion is relatively limited. This, of course, is the diagnosis in most chronic pain disorders or chronic pain syndromes. The psychiatrist may address the varied sources of psychological factors, both preexisting and subsequent, as obstacles to recovery, but would not have the chief responsibility to address causation, degree of impairment, and medical treatment. On the other hand, if the diagnosis is Pain Disorder associated with psychological factors, then those factors have the major role, and this becomes mostly a mental disorder. The diagnosis at times may also be Undifferentiated Somatoform Disorder, which, along with pain symptoms, may include a variety of sensory and motor complaints that cannot be adequately explained by objective medical pathology. Here, too, the psychiatrist can have a primary responsibility. However, in both instances, objective medical pathology that would be consistent with a general medical condition must be ruled out or discounted as to its importance by a medical determination. Frequently, of course, there are differences of opinion among medical practitioners who evaluate pain disorders. Where substantive differences exist, the psychiatrist's opinions can only be contingent on resolution of the initial dispute about the presence of objective medical pathology. Two caveats to this analysis must be mentioned. The first is that the absence of objective medical pathology does not automatically put the condition in the diagnosis of Pain Disorder associated with (mostly) psychological factors, since the psychological factors in that instance need to be clearly demonstrated as well. Second, even if the diagnosis is a Pain Disorder associated with psychological factors and/or an Undifferentiated Somatoform Disorder, this does not equate with the earlier concept of psychogenic pain, which seemed to imply that the pain was imaginary. Today, the pain experience itself is viewed phenomenologically as a true physical sensation of pain, which is based on a complex of interrelated mechanisms that drive and maintain it. Those mechanisms can include very prominent psychological factors, which provide a greater understanding about the degree of suffering and the resultant claims of impairment and disability. In fact, psychological factors affecting impairment and disability may be even more relevant than the analysis of the pain experience itself, since there are tremendous differences in individuals personally, socially, and cross-culturally, as to how much impairment pain causes. Practically, though, the challenge is to determine to what extent psychological factors are involved, if at all, and what they consist of.

Unfortunately, the difference in schools of thought with regard to pain disorders has generated major controversy, which invariably enters the arena of litigation. Opposing experts from the same and from different disciplines are frequently at odds with one another, and may knowingly or unknowingly reflect strongly divergent interests. Psychoanalytic thinking that formed the theoretical base for earlier beliefs regarding psychogenic pain is not currently regarded seriously; thus, explanations of pain as merely the expression of some internal conflict, guilt, aggression, or a need for love are generally rejected in favor of behavioral and physiological explanations. This has been fueled by a large pain treatment industry that, for the most part, must-at least where litigation is concerned-accept the pain as due to accidental or work-related injury for financial reimbursement (150). This, in part, has led to a backlash from critics who use

claims of somatization, secondary gain, and disproportionate disability to argue against the legitimacy of many pain conditions and their connection to an actual injury (276). Historically, somatization and the psychosomatic components of pain disorders have had support as well, since it has been shown how more primitive psychosomatic conditions in previous centuries (e.g., hysteria, psychogenic paralysis) have been replaced in more recent times by growing numbers of individuals claiming fatigue and pain (277). On the other hand, there are equally strong proponents who say that pain disorders have a neurophysiological base, in which psychological factors play a relatively limited role, and who claim that those who dismiss the legitimacy of these conditions are using somatization pejoratively to imply that the pain does not exist or that the sufferer is malingering (276). However, as has been outlined earlier, there is abundant newer research that confirms the strong role of psychological and psychosocial factors in chronic pain disorders, as well as their interrelationship with neurophysiological, hormonal, and other mechanisms. Neither position is completely accurate. Most pain disorders are probably initiated by a physical injury, and except for malingerers, most individuals with pain disorders are experiencing real pain symptoms. Likewise, most pain disorders have at least some psychological factors associated; and those factors are not playing the major role in perpetuating the condition. However, where psychological factors play an important role, there can be major obstacles to recovery, which may be independent of the physical injury itself. Even if modern concepts of neurobiopsychosocial interactions apply, this should not mean that the psychological factors can be dismissed. Just because most individuals with pain disorders experience legitimate pain, due in many cases to physical injury, it does not mean that all individuals with pain disorders have physical injury as their cause or that neurophysiological mechanisms adequately explain the poor recovery. In most cases, impairment and disability from pain disorders are strongly related to psychological and motivational factors, regardless of the type of pain disorder.

Even more complex is whether the psychological factors in chronic pain disorders are subject to any conscious control, or are merely inherent, unconscious variables within the individual and his or her social setting. Assuming that they are conscious has often led to poorly founded conclusions about malingering in these patients, simply because the symptom presentation is exaggerated (278). Observations of suboptimal effort, overreporting of symptoms, and even secondary gain do not necessarily mean that someone is malingering; but, overstatement of pathology and understatement of premorbid health are nevertheless tied to the utility of such representations, whether conscious or unconscious (279). More likely than not malingering, exaggeration, and even genuine symptoms are on a continuum with no bright light separating them, and elements of conscious and unconscious control in the symptom presentation are more a matter of gradation. Where a diagnosis of Somatoform Disorder (the category in which psychologically impacted Pain Disorders are found) is made, by definition, the symptoms are not intentionally produced or feigned, and are not malingered. But this should not imply that the individual is helpless, either. Assuming a passive invalid role, not fully investing in the rehabilitation process, and focusing only on external pain relief, can suggest a lack of healing intention. Therefore, even if the precipitant of the pain appears to be an injury, with clear physical findings, the cause of the pain condition may be self-generated, to a lesser or greater degree, and may include variable levels of exaggeration and symptom magnification. Understanding that rigid conceptual divisions on these issues are artificial, opinions about causation, where appropriate for a psychiatrist, should rely on a balancing test that considers the relative weight of physical versus psychological factors, injury versus noninjury components, and investment versus noninvestment in recovery.

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RELEVANT QUESTIONS FOR A PSYCHIATRIC EVALUATION

Is there a diagnosable pain disorder?

Are all the symptoms consistent with the disorder, or might they represent some other condition?

Was the nature of the injury proportional to the symptoms claimed? Is the injury verifiable?

Are there reliable medical evaluations that demonstrate objective physical pathology?

Are there diagnostic studies that help objectify physical pathology?

Is the patient's presentation one of significant symptom exaggeration, pain behavior, or inconsistency?

Does the patient demonstrate fear-avoidance, catastrophizing, lack of self-efficacy, anxiety, or hypervigilance to pain?

What was the individual's life adjustment prior to the injury? Job adjustment? Family and personal adjustment?

Is there a history of other pain disorders, previously or currently?

Is this a typical course of illness and/or response to treatment? If not, why? Is there motivation to heal?

Do psychological tests show somatization potential, poor coping style, pain-enhancing factors, or clinically relevant mental symptoms?

Is there overreliance on narcotic medications? Heavily sedating medications?

What objective measures of impairment and disability are there?

Is the individual adopting a passive invalid role? Does the individual see himself or herself as helpless? Is he or she waiting for external solutions?

Is there a history of mental disorder? Personality disorder?

What predisposing personality factors are there? Ergomania? Alexithymia? A history of excessive responsibility for others?

What is the longitudinal history of the pain disorder? Is it corroborated?

Does the patient's personal history show evidence of abuse, lack of early emotional attachment, repeated life traumas?

Did the injury or onset of pain occur at a time when the patient was already worn out from personal struggles or conflicts?

How does the patient's family and others interact with the individual? With anger? Solicitousness? Support?

What are the reinforcing elements for pain? Disability compensation? A potential damage award? Other secondary gain?

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FINAL OPINIONS

Because chronic pain disorders regularly involve an interweaving of physical and psychological factors, and because a full understanding of these conditions may involve more than one discipline, final conclusions are often contingent on the validity of opinions from another discipline. For example, one orthopedic surgeon may conclude that a patient does not have evidence of objective medical pathology, whereas another orthopedic surgeon or perhaps a physiatrist may conclude that there is. Since a psychiatric evaluation of any Somatoform Disorder first must address the threshold question of whether the symptoms are not fully explained by a general medical condition, the psychiatric opinions that follow typically do not stand alone but refer to prior medical evaluations. Of course, psychiatrists, by virtue of their medical background, may be in a position to independently assess observations from physical examinations and diagnostic testing; but, typically, they do not perform physical examinations, other than in a limited capacity. Therefore, once a psychiatrist conducting an evaluation has gathered sufficient information from personal interviews, psychological testing, and record review, and has addressed relevant questions such as those posed in the previous section with regard to the information gathered, it is then necessary to form final opinions that incorporate opinions from outside of his or her own psychiatric discipline, and to demonstrate if and when the psychiatric opinion is contingent on the resolution of any dispute with regard to the medical opinions. It is also important to recognize the limitations of psychiatric opinion where significant medical pathology is demonstrated, even though psychological factors are also prominent. To that end, an algorithm is presented here to help frame psychiatric conclusions within the context of multidisciplinary opinions (see [Figure C, Algorithm: Psychiatric Opinions for Pain Disorders](#)). The following guide to the algorithm expounds in greater detail the steps outlined:

(A) *Are there clear physical findings?* Here the attention is to medical records, physical examinations, and diagnostic studies that corroborate a specific pain disorder or condition. Any dispute among evaluators should be noted, as well as any inconsistencies.

(B) If there are clear physical findings, *do they fully explain the pain symptoms?* Here the attention is again on medical evaluations and whether there has been concern raised about the extent of the pain disorder compared to objective findings. Usually, a psychiatric evaluation will not be requested unless there is some concern. At the same time, medical evaluators may also be concerned only a secondary psychiatric problem, even if the medical pathology is well documented.

(C) To the extent that medical evaluations do not express concern about the proportionality of symptoms to objective findings, and a secondary psychiatric complication is not being raised, no further psychiatric opinion may be needed.

(D) If the pain disorder is not fully explained by medical pathology, and medical evaluators do express concern about inconsistencies, the extent and duration of the symptoms, the lack of progress in the natural course of the disorder, or symptom exaggeration, the question then is, *are psychological factors evident?* Some would say that symptom exaggeration, pain behavior, or inconsistency by itself is evidence of psychological factors; but care must be taken to understand that reactions to pain are variable, both within the individual and within the cultural background of the individual. Therefore, issues such as fear-avoidance, catastrophizing, lack of self-efficacy, a strong emotional overlay, hypervigilance, or similar findings may need to be demonstrated.

(E) To the extent that psychological factors are not evident, more than in just variability in pain response, further psychiatric opinion may not be needed. It is important, though, to be sure that a comprehensive history is available, which addresses premorbid psychological adjustment, the presence of other pain disorders, and psychiatric disturbance generally, before excluding psychological factors as having no significant bearing.

(F) If psychological factors are evident in more than an incidental way, it is then important to answer, *do they play an important role?* This becomes a balancing test that weighs the strength of the physical findings and any substantial dispute as to the diagnosis against the degree of psychological factors and psychiatric disturbance.

(G) If the psychological factors do not appear to play an important role, and if no more relevant history points to psychiatric disturbance, further psychiatric opinions may not be necessary, except for pain management issues. These, presumably, could help any patient with a pain disorder deal with his or her condition more effectively. An actual diagnosis of Pain Disorder associated with both psychological factors and a general medical condition may not necessarily be warranted.

(H) If psychological factors are playing an important role, then more likely than not the diagnosis is Pain Disorder associated with both psychological factors and a general medical condition. In this case, there is typically no primary psychiatric opinion regarding either causation or impairment. There are, however, possible ancillary opinions that deal with pain management issues, motivation and mitigation, comorbid psychiatric disorders, and degree of nonphysical psychiatric impairment.

(I) The second major branch in the algorithm comes into play when there are no clear physical findings, as per (A); now it becomes even more important to determine, *are psychological factors evident?* The same issues with regard to symptom exaggeration and inconsistency, fear-avoidance, catastrophizing, lack of self-efficacy, psychiatric disturbance, and hypervigilance come into play, see (D). However, in this instance, even more detailed history and background information becomes necessary.

(J) If psychological factors are not evident, and it is possible that for varied reasons physical findings are not clear, at least as of yet, then psychiatric input may be minimal. Here, too, it is very important to ensure that a thorough psychiatric and personal history has been taken, in the process of which covert psychological factors should be suspected. One of the difficulties in this situation is that some individuals with chronic pain disorders are very defensive about insinuations that psychological factors may play a role. They also may tend to deny psychiatric disturbance because they are lacking in psychological insight or are concerned that they will not be believed as to their pain. Sometimes it is difficult to gain access to information when the individual without clear physical findings refuses to submit to a more detailed psychiatric

evaluation, arguing that it is irrelevant.

(K) If psychological factors are evident, as with (F), the next question is, *do they play an important role?* The same balancing test described earlier applies here, but with the caveat that the starting point may be a questionable medical condition.

(L) To the extent that the psychological factors do not appear to play an important role, this presents a conundrum for both medical and psychiatric evaluators. How then can the condition be explained? At times, redundant evaluations and diagnostic testing follow, with little return. In this case, there is an even greater responsibility to perform a thorough psychiatric evaluation and obtain a comprehensive personal history. The psychological impact of even limited psychological factors should be suspect, as well as the possibility that a full understanding of those psychological factors is not yet available. Although not necessarily the case, the diagnosis of Pain Disorder associated with both psychological factors and a general medical condition is more likely here than meets the eye.

(M) Once it has been determined that psychological factors do play an important role, the next question is, *do they play the major role?* Appreciating once again that previous divisions between psychogenic pain and physical pain are artificial, there are, nonetheless, a minority of pain conditions in which psychological factors overwhelmingly predominate and trigger important causation and treatment decisions.

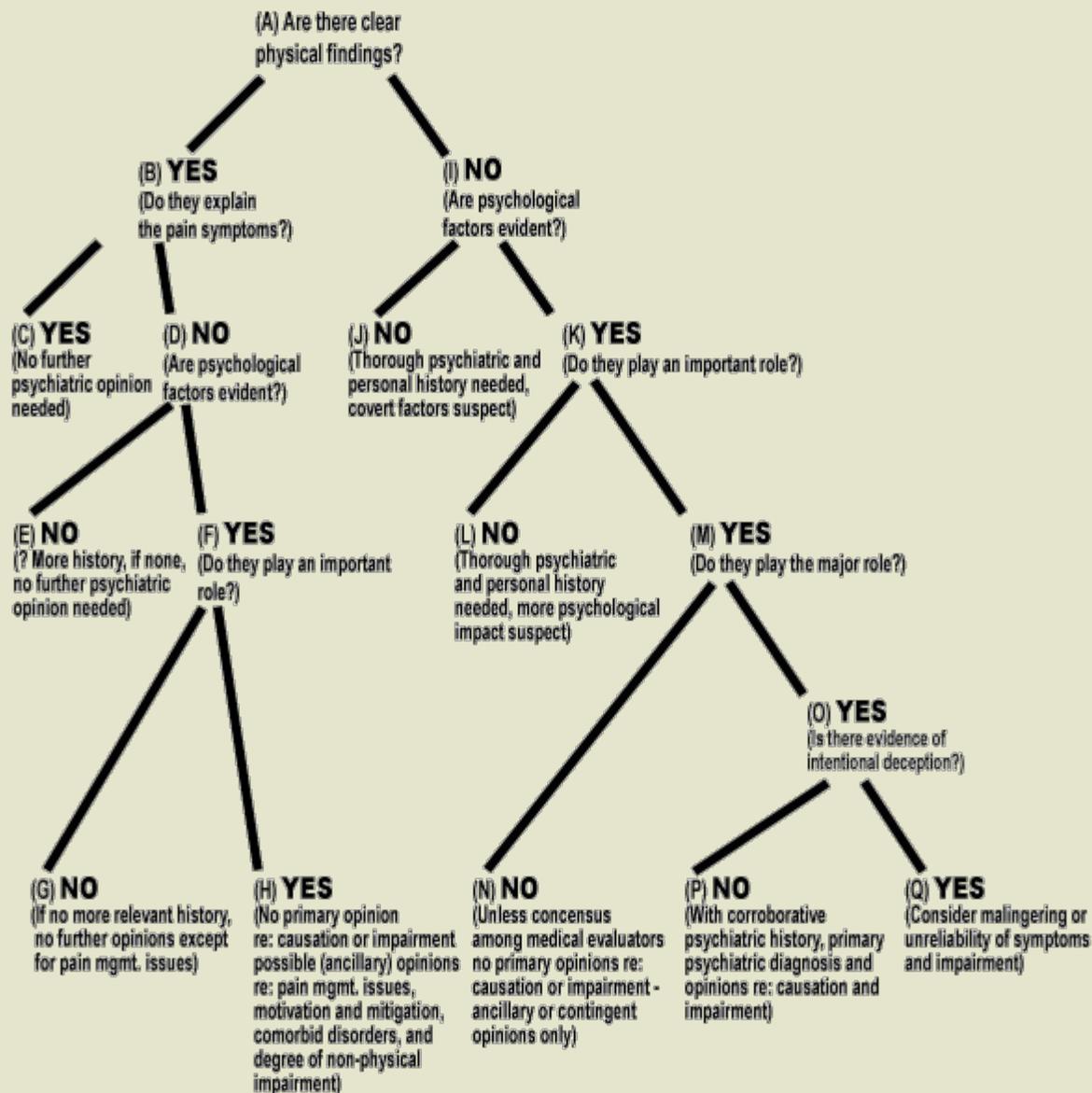
(N) Unless there is a consensus among medical evaluators that no physical findings are present, or at least that they make an insignificant contribution, the opinion remains that this is a Pain Disorder associated with both psychological factors and a general medical condition. No primary psychiatric opinion regarding causation or impairment is given except, as in (H), for ancillary opinions. In some instances, there are wide disputes among medical evaluators, with some convinced that there are no significant physical findings, and others possibly relying on questionable or nonspecific diagnoses. Here, psychiatric opinion can be contingent, to the extent that there are insignificant physical findings to explain the extent and duration of the pain condition; then psychiatric opinion can follow as per (O), below, but the resolution of the medical dispute must precede this contingency.

(O) If psychiatric factors are playing the major role, then the diagnosis is at least a Pain Disorder associated with psychological factors. Once this has been established, the question remains, *is there evidence of intentional deception?* This is rarely something that can be concluded through a personal interview or psychological testing. It is typically shown through records that dramatically point to inconsistencies in the individual's behavior. Surveillance may or may not be a useful tool, but the films generated by this method speak for themselves to the fact finder, although the psychiatrist may also comment about the inconsistency between what is viewed and what the individual has represented.

(P) If there is no evidence of intentional deception, then this is substantially a psychiatric condition, in which case primary psychiatric opinions about causation and impairment are appropriate. It is important to emphasize that there should be corroborative information within the psychiatric history of adjustment problems, psychiatric disturbance, and the role the pain disorder may play as a pathological resolution to intolerable personal circumstances.

(Q) If there is evidence of intentional deception, then the possibilities of a Factitious Disorder or Malingering may be present; or at least a conclusion about the unreliability of symptoms and impairment can be made. Even when Malingering is strongly suspected, it may be better to leave it as a rule-out diagnosis, because, ultimately, it is based on a factual determination. Possible diagnoses then could be: Pain Disorder associated with psychological factors; Rule-out Malingering.

Figure C Algorithm: Psychiatric Opinions for Pain Disorders



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CASE EXAMPLES

The following case examples are adapted from actual histories of patients who presented with complex chronic pain problems in conjunction with suspected psychological factors:

Jane R.

Jane R. is a 41-year-old woman who was employed at an electronics assembly plant for about two years, until she slipped on a patch of grease, fell, and landed on her left elbow. Although her elbow was mildly sore, she was able to finish her shift without apparent difficulty. When she woke up the next day, however, the pain in her elbow had increased substantially and she felt unable to go to work. She was seen at a local emergency department where X-rays of her elbow revealed no fracture. There was mild swelling of the elbow, and the diagnosis was, simply, contusion. Jane was instructed to follow up with her primary care physician, who then referred her for physical therapy (PT). She said that PT only made her pain worse, so she stopped going. Over the past three years, Jane has seen numerous physicians, including orthopedic

surgeons and psychiatrists, who have evaluated her and treated her with anti-inflammatory and narcotic analgesics, anticonvulsants used for neuropathic pain, and steroid injections, all without marked relief. Jane has had numerous diagnostic studies that have not demonstrated objective pathology. Her diagnoses typically have been tendonitis, ulnar neuralgia, contusion, and reflex sympathetic dystrophy (RSD)/complex regional pain syndrome (CRPS). Sympathetic blocks and a bone scan failed to corroborate RSD/CRPS, although on physical examination her left upper extremity has often appeared cooler than the right, and sensitive to even light touch. She has had additional rounds of physical therapy, as well as occupational therapy. She has not returned to work for the last three years.

Not only has Jane's elbow pain continued, it has worsened and, over time, spread down the entire left upper extremity to her hand and up to her shoulder and neck. She keeps her left arm in a flexed position against her chest most of the time, and says she cannot extend it completely or lift her arm beyond shoulder height. On presentation, she shows marked pain behaviors and symptom exaggeration; and attempts to assess her range of motion are accompanied by grimacing, wincing, and guarding against any manipulation of the arm. Jane is concerned that the pain is starting to spread into her right upper extremity. Although Jane has a history of chronic migraine headaches, she says that she now has a new type of headache, which is more tension in nature and spreads from her neck up and over her scalp. She also indicates that she continues to have problems with longstanding low back pain, irritable bowel symptoms, and fatigue.

Jane was referred to a psychologist for pain management, at which time she was diagnosed as having Pain Disorder associated with both psychological factors and a general medical condition, and Adjustment Disorder with depressed mood. At the recommendation of the psychologist, her primary care physician started her on an antidepressant medication. Jane says she feels comfortable speaking with the psychologist, but does not feel that the pain management techniques are helpful. Jane does admit that she has been very frustrated and depressed because she cannot work or participate in recreational activities as she did before. She claims that she has difficulty with sleep because of her pain, and so spends most of the day in bed trying to get some rest. Her mother, who lives nearby, comes daily to help with cooking and cleaning, and takes her to medical appointments. Jane receives two-thirds of her pay from the electronics assembly plant as a workers' compensation benefit, and has now been approved for Social Security disability benefits. The combination of these benefits totals almost as much as she previously earned from her job at the plant.

Jane's background history, which was gleaned mainly from earlier physician and mental health records, shows that she was raised in a very chaotic family and that her father was a severe alcoholic. Reportedly, he physically and sexually abused Jane, as well as two of her sisters. The mother appears to have been helpless, and while acknowledging the physical abuse, denied that any sexual abuse was occurring. When Jane was 16 years old, she became pregnant as a result of a date rape. She did not report the incident, but went on to have the baby. The father of the baby offered no support. In the early years following the birth of her child, she survived by holding down a couple of jobs. She subsequently married a man who was physically and verbally abusive, and became disabled himself about four years into the marriage, following a workers' compensation back injury. Jane had two more children by her husband, and became the main breadwinner in the family. Finally, when she learned that her husband was sexually abusing one of her daughters, she left him. Their divorce was acrimonious, and she was afraid to report her husband's abuse of the daughter, for fear that her children might be taken away from her. Jane raised her children alone after that; they are now grown and have left home. However, the daughter who had been abused became pregnant and moved back in with Jane, where she currently lives and raises her own child. She does not work, so Jane became the primary support for her daughter and her grandchild. Prior to her injury, in addition to working at the electronics assembly plant, she also would bartend at night.

Jane R. presents with a minor upper extremity injury that showed no fracture and was initially diagnosed only as a contusion. In spite of conservative treatment, she did not improve, and instead has had persistent and worsening pain over a three-year period. This immediately raises the question of why her pain did not subside in the natural course of a minor injury, and why she has become totally disabled. The diagnoses that have been given are, for the most part, nonspecific and difficult to validate. The fact that she had some temperature difference in the injured extremity, and sensitivity to light touch, led evaluators to suspect RSD/CRPS. However, as previously indicated, this diagnosis is frequently used when nothing else from a medical standpoint fits. It is based primarily on subjective symptoms and/or physical examination, which does not always have interrater reliability. While a sympathetically mediated type of phenomenon may be responsible for the CRPS, and could represent the earlier diagnosis of RSD, in Jane's case, neither sympathetic blocks nor bone scan were able to confirm this. Therefore, in fact, CRPS is very nonspecific in her case. Furthermore, the spreading pain, while described in the literature as possible with RSD, is not

sufficient explanation here, again because there is no validated evidence of a sympathetic mechanism. It is also noted that Jane keeps her left arm in disuse, so that some of her symptoms that resemble RSD/CRPS can arise from that. Therefore, she does appear to have a pain condition that is out of proportion to her injury and without objective medical basis. Of course, one or more of her treatment providers may claim that an objective medical basis exists; and while a psychiatrist may need to defer medical questions, it is likely here that even those evaluators who support medical pathology will acknowledge that it does not alone explain her chronic pain. To the extent that psychological factors are acknowledged by everyone, it is likely that they will be attributed to a secondary emotional reaction to the unrelieved pain, which purportedly further aggravates the pain condition but is still causally related as a foreseeable complication.

The psychologist to whom Jane R. was sent for pain management has diagnosed her as having Pain Disorder associated with both psychological factors and a general medical condition, and Adjustment Disorder with depressed mood. Unfortunately, pain management techniques taught by the psychologist have not been helpful; and, paradoxically, Jane persists in explaining her emotional reaction as due to frustration because she cannot work or function otherwise as she did before. This type of explanation may not be unreasonable in some individuals, but it does not appear that Jane is the type of patient who has tried to be as functional as possible in spite of her pain. In fact, she has taken on a prominent invalid role; she does very little and depends on others for help and support. More importantly, the question of whether the psychological factors in her Pain Disorder are secondary to the injury or preexisting remains to be answered by examining her life as a whole in conjunction with the timing of the onset of her pain condition. Here, Jane demonstrates marked pain behaviors and symptom exaggeration, as well as visible emotional distress. For example, her left arm is in a flexed, immovable position against her chest most of the time, and she has little if any use of it. The helplessness that she shows by staying in bed most of the day reflects her lack of self-efficacy and her acceptance of the invalid role. The case example also does not discuss any psychological testing, which may or may not have been done when Jane was referred to pain management. Chances are that testing would have shown symptom exaggeration and somatization potential, as well. This, together with the out-of-proportion symptoms that she describes, supports the prominent role of the psychological factors.

To the extent that it is determined that Jane's Pain Disorder has little if any objective medical pathology, then the diagnosis could be Pain Disorder associated with psychological factors (primarily or playing a major role). In this situation, it is difficult to justify the conclusion that the psychological factors represent only an emotional reaction to her pain, when there is insufficient basis for the pain in the first place. Of course, it should always be understood that this is not a simple *psyche/soma* distinction, but only an acknowledgment of the relative impact of psychological versus physical factors.

Having determined that psychological factors are playing at least an important if not major role, what does Jane's background and personal history show with regard to her life adjustment prior to the injury? Here, the historical evidence is overwhelming for a background of chaos, abuse, and personal trauma. It also shows that Jane was someone forced to assume inordinate responsibility for herself and others, without much support. She appears to have been overextended and, most likely, worn out. She also may be someone who does not deal well with emotional issues arising out of conflict and crisis, and displays concrete and psychologically unskillful defense mechanisms. Psychological testing may also show characteristics of *alexithymia*, which together with her traumatic history predispose her to physical symptoms as a manifestation of stress, since she does not verbalize emotions well. Jane's background and personal history are not just coincidental with this out-of-proportion and persistent pain condition. So, to the extent that other evaluators have not known about this history, any conclusions by them about psychological factors being secondary to the injury are not reliable.

Finally, the potential for secondary gain is significant in this case for two reasons: first, Jane can avoid the previously overwhelming responsibilities in her life; and, second, she is receiving substantial disability benefits. This is not to say that Jane is necessarily malingering but that her motivation to heal is probably lacking, given that she would then have to resume the overwhelming responsibility she previously had. Depending on the jurisdiction, the cause of Jane's Pain Disorder may or may not still be based on the work injury. Nonetheless, the substantial reasons for her persistent pain and psychological distress are not simply due to a minor contusion at work.

Gary O.

Gary O. is a 30-year-old man who reported a low back injury he suffered by lifting bags of cement mix at a construction site where he had been working. His foreman took him to a local emergency department, at which time Gary described severe pain in his low back that radiated down his right leg. He had difficulty

straightening up and appeared to be in intense agony. Although X-rays of his back were negative, a herniated lumbar disc was suspected. Gary was prescribed a muscle relaxant and a narcotic analgesic, and released with a referral to an orthopedic surgeon. The orthopedist subsequently obtained magnetic resonance imaging (MRI) studies, which confirmed a herniated lumbar disc. Initially, the doctor chose to treat Gary conservatively, with physical therapy; but after several weeks with no improvement, a lumbar discectomy was scheduled.

Gary said that the surgery alleviated his right leg pain, but made the low back pain worse. A subsequent MRI showed that the surgery had been successful and that there was no further evidence of a disc impinging upon neural structures. The orthopedist sent Gary for another round of physical therapy, which again proved unsuccessful. Notes from the orthopedist and the physical therapist, however, stated that Gary presented with significant symptom exaggeration and inconsistencies on physical examination, and that he walked hunched over and leaning to the left side. A neurosurgical consultant was then asked to see Gary; he reported that the physical examination was essentially normal but with a number of Waddell's findings and significant symptom exaggeration.

Gary has not returned to work. He continues to take high doses of narcotic analgesics, which he says are the only thing that makes a difference. Most of his days are spent in a reclining or a sitting position. He does few chores. Light-duty work was offered to him, but Gary indicates that due to the narcotics he cannot concentrate well; and, besides, he has never wanted to work at a desk job. Gary denies that he is depressed, only frustrated that his pain continues. He receives workers' compensation benefits, but the insurance carrier has been challenging this on the basis that Gary has not been willing to try light-duty work.

Gary's background shows that his father and two brothers all had difficulties with alcohol abuse; and an older sister has for years taken narcotic analgesics for her chronic neck pain. Gary dropped out of high school in part because of disciplinary problems and drug abuse, including marijuana, methamphetamine, and hallucinogens. He has at least one conviction for driving under the influence. His work history indicates sporadic employment, usually in heavy-labor positions. Gary is married and has a son who is 8 years old. Gary's wife works as a licensed nurse's assistant at a nursing home. Since his injury, she has been working extra shifts to earn overtime pay because their income has dropped since Gary stopped working.

Gary's orthopedist has recommended that he see a psychiatrist or psychologist for pain management, but he became angry at this suggestion, and refused to go, believing that the orthopedist must think his pain is all in his head. Gary's workers' compensation insurance carrier is recommending an independent psychiatric evaluation, but Gary's attorney is challenging this.

Gary O. presents with a somewhat different case from that of Jane R., even though there are similarities. Here, there was objective evidence of medical pathology in the form of a herniated lumbar disc. Conservative treatment, followed by a surgical discectomy, did not result in any significant improvement, though there was no further diagnostic evidence of a disc impinging upon neural structures. It is difficult, however, to ignore the fact that there was medical pathology at one time and that intrusive surgery was required, which potentially has its own complications (i.e., scarring, etc). In this regard, Gary's presentation shows notable symptom exaggeration, inconsistencies, and pain behaviors. Like Jane, Gary, too, has taken on an invalid role. Thus, it is likely that he would be diagnosed as having Pain Disorder associated with both psychological factors and a general medical condition, on the basis of his dramatic presentation alone. But he is refusing to see a psychiatrist or a psychologist for pain management, and will probably resist even having an evaluation by a psychiatrist or a psychologist. Clearly, this becomes a difficult problem for his medical treatment providers, and raises the question whether he can be forced to undergo such an examination, and subject himself to the detailed questioning and intrusion on his personal life it would entail. To the extent that medical treatment providers, or independent medical examiners, emphasize the psychological and emotional presentation that appears to exist, the argument can be made that Gary will have to submit to a psychiatric or psychological evaluation. If he does, it is unlikely that he will be cooperative in relaying his entire history, meaning that an adequate understanding of his background may still not be possible.

In Gary's case, there is some very important history that does need to be addressed, however; namely, his apparent dependence on narcotic analgesics. In the first place, he indicates that he cannot concentrate because of the narcotics, and this is one reason he is not able to work at a desk job. It is difficult, then, to justify continuing narcotics if they are creating impairment. Furthermore, Gary's family and personal background, which indicate a predisposition to dependence and addiction, are very problematic, as well, and should send up a red flag to proceed with caution in considering him for long-term narcotic analgesic

treatment.

Assuming that no other background history that predisposes him to chronic pain is identified, Gary's diagnosis of Pain Disorder associated with both psychological factors and a general medical condition becomes mostly a medical determination as to causation. Since psychological factors are not playing the major role, psychiatric opinion is limited, though not without its importance. In particular, psychiatric opinion can address whether secondary gain factors are sufficient to make his claims of impairment less than reliable. Also, psychiatric opinion can address that he has not sufficiently mitigated his condition-specifically, he has refused to attempt work he could be doing; and he has refused to participate in recommended psychological pain management, if that were made available. Finally, and most importantly, psychiatric opinion can point out the deleterious role of the narcotic analgesics in Gary's rehabilitation process. There is little justification in continuing narcotic analgesics that do not increase functioning, especially in an individual with a predisposition to substance abuse.

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AFTERWORD

The complexities of chronic pain disorders present a major challenge to medical and psychiatric evaluators alike. Earlier psychoanalytic concepts about psychosomatic illness have been replaced by a greater appreciation of the interrelationship between pain symptoms and neurochemical processes within the peripheral and central nervous system. At the same time, psychological factors and psychiatric disturbance continue to regularly accompany chronic pain disorders. This psychiatric update and evaluation guide has tried to show how recent research with regard to those psychological factors suggests not only their prominent role but also their connection to central nervous system mechanisms. It also has highlighted the greater awareness that exists today of the personal, familial, and circumstantial context in which pain disorders occur, and described why that cannot be excluded from questions of causation, extent and duration of illness, and prognosis for rehabilitation. All of these components are more layered than linear, indicating that legal and administrative standards for addressing these issues in litigation may need to be reconsidered, to the extent that they rely on simple before-and-after concepts and unidimensional stress analysis. Moreover, the importance of the patient narrative cannot be overstated-simply, a patient's pain can never be understood without understanding the patient. Unfortunately, there is rarely enough time in contemporary pain treatment centers to accomplish this. This, of course, limits the reliability of any opinions about a patient's pain; at the same time, it may help explain the often-limited benefit of long-term pain treatment, in spite of enormous resources expended in the process.

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REFERENCES

1. Verhaak PF, Kerssens JJ, Dekker J, Sorbi MJ, Bensing JM: Prevalence of chronic benign pain disorder among adults: a review of the literature. *Pain* 1998; 77:231-239
2. Harstall C, Ospina M: How prevalent is chronic pain? *Pain Clin Updates* 2003; 11:1-4
3. Merikangas KR, Ames M, Cuil, Stang PE et al: The impact of comorbidity of mental and physical conditions on role disability in the U.S. adult household population. *Arch Gen Psychiatry*, 2007; 64:10:1180-1188
4. Hestbaek L, Leboeuf-Ydec, Engberg M, Lauritzen T et al: The course of low back pain in the general population. Results from a 5-year prospective study. *J Manipul Physiol Therap* 2003; 26:213-219
5. Pengel LH, Herbert RD, Maher CG, Refshauge KM: Acute low back pain: systematic review of its prognosis. *Br Med J* 2003; 327:323-327
6. Smith BH, Elliott AM, Hannaford PC: Is chronic pain a distinct diagnosis in primary care? Evidence from the Royal College of General Practitioners' Oral Contraception Study. *Fam Pract* 2004; 21:66-74
7. France RD, Krishnan KRR: *Chronic Pain*. Washington, DC, American Psychiatric Press, 1988
8. Bierman AE: The medico-legal enigma of fibromyalgia: social security disability determinations and

- subjective complaints of pain. *Wayne L Rev* 1998; 44:259
9. McCaffery EJ et al: Framing the jury: cognitive perspectives on pain and suffering awards. *Va L Rev* 1995; 81:1341
 10. Finch M: Law and the problem of pain. *U Cin L Rev* 2005; 74:285
 11. Niemeyer PV: Awards for pain and suffering: the irrational centerpiece of our tort system. *Va L Rev* 2004; 90:1401
 12. American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised*. Washington, DC, American Psychiatric Association, 1987
 13. American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*. Washington, DC, American Psychiatric Association, 1994
 14. American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision*. Washington, DC, American Psychiatric Association, 2000
 15. American Medical Association: *Guides to the Evaluation of Permanent Impairment, Fifth Edition*. Chicago, IL, American Medical Association Press, 2000
 16. American Medical Association: *Guides to the Evaluation of Permanent Impairment, Sixth Edition*. Chicago, IL, American Medical Association Press, 2008
 17. Merskey H: *History of Psychoanalytic Ideas Concerning Pain*, in *Personality Characteristics of Patients with Pain*. Edited by Gatchel RJ, Weisberg JN. Washington, DC, American Psychological Association, 2000
 18. Engel GL: Psychogenic pain and the pain-prone patient. *Am J Medicine* 1959; 26:899-918
 19. Blumer D, Heilbron M: *Dysthymic Pain Disorder*, in *Handbook of Chronic Pain Management*. Edited by Tollison CD. Baltimore, William and Wilkins, 1989
 20. Binswanger O: *Die Hysterie*. Vienna, Holder, 1904
 21. Melzack R, Wall PD: Pain mechanisms: a new theory. *Science* 1965; 150:971-979
 22. Drukteinis AM: *The Psychology of Back Pain*. Springfield, IL, Charles C. Thomas, 1996.
 23. Namaka M, Gramlich CR, Ruhlen D, Melanson M et al: A treatment algorithm for neuropathic pain. *Clin Ther* 2004; 26:7:951-979
 24. Harden RN: A clinical approach to complex regional pain syndrome. *Clin J Pain* 2000; 162:2 suppl:S26-S32
 25. Apkarian AB, Bushnell MC, Treede RD, Zubieta JK: Human brain mechanisms of pain perception and regulation in health and disease. *Eur J Pain* 2005; 9:463-484
 26. Gracely RH, Petzke F, Wolf JM, Clauw DJ: Functional magnetic resonance imaging evidence of augmented pain processing in fibromyalgia. *Arthritis Rheum* 2002; 46:1333-1343
 27. Banks WA, Watkins, LR: Mediation of chronic pain: not by neurons alone. *Pain* 2006; 124:1-2
 28. Pedersen LH, Scheel-Kruger J, Blackburn-Munro G: Amygdala GABA-A receptor involvement in mediating sensory-discriminative and affective-motivational pain responses in a rat model of peripheral nerve injury. *Pain* 2007; 127:17-26
 29. Neugebauer V: The amygdala: different pains, different mechanisms. *Pain* 2007; 127:1-2
 30. Phelps EA, Ledoux JE: Contributions of the amygdala to emotion processing; from animal models to human behavior. *Neuron* 2005; 48:175-187

31. Balleine BW, Killcross S: Parallel incentive processing: an integrated view of amygdala function. *J Neurosci* 2006; 29:5:272-279
32. Smith BH, MacFarlane GJ, Torrance N: Epidemiology of chronic pain, from the laboratory to the bus stop: time to add understanding of biological mechanisms to the study of risk factors in population-based research? *Pain* 2007; 127:5-10
33. Haddad JJ, Saade NE, Safieh-Garabedian B: Cytokines and neuroimmune-endocrine interactions: a role for the hypothalamic-pituitary-adrenal revolving axis. *J Neuroimmunol* 2002; 133:1-19
34. McBeth J, Chiu YH, Silman AJ, Ray D et al: Hypothalamic-pituitary-adrenal stress axis function and the relationship with chronic widespread pain and its antecedents. *Arthritis Res Ther* 2005; 7:R992-R1000
35. Gaab J., Baumann S, Budnoik A, Gmunder H et al: Reduced reactivity and enhanced negative feedback sensitivity of the hypothalamus-pituitary-adrenal axis in chronic whiplash-associated disorder. *Pain* 2005; 119:219-224
36. Jerjes WK, Cleare AJ, Wessely S, Wood PJ, Taylor NF: Diurnal patterns of salivary cortisol and cortisone output in chronic fatigue syndrome. *J Affect Disord* 2005; 87:299-304
37. MacGregor AJ, Andrew T, Sambrook PN, Spector TD: Structural, psychological, and genetic influences on low back and neck pain: a study of adult female twins. *Arthritis Rheum* 2004; 51:160-167
38. Manek NJ, MacGregor AJ: Epidemiology of back disorders: prevalence, risk factors, and prognosis. *Curr Opin Rheumatol* 2005; 17:134-140
39. Matsui H, Maeda A, Tsuji H, Naruse Y: Risk indicators of low back pain among workers in Japan association of familial and physical factors with low back pain. *Spine* 1997; 22:1242-1247
40. Borge AI, Nordhagen R: Recurrent pain symptoms in children and parents. *Acta Paediatr* 2000; 89:1479-1483
41. Solovieva S, Kouhia S, Leino-Arjas P, Ala-Kokko L et al: Interleukin 1 polymorphisms and intervertebral disk degeneration. *Epidemiology* 2004; 15:626-633
42. Mogil JS, Wilson SG, Chesler EJ, Rankin AL et al: The melanocortin-1 receptor gene mediates female-specific mechanisms of analgesia in mice and humans. *Proc Natl Acad Sci USA* 2003; 100:4867-4872
43. Yu L: Pharmacogenetics: The OPRM1 (mu-opioid-receptor) Gene, in *The Genetics of Pain (Progress in Pain Research and Management)*. Edited by Mogil JS. Seattle, IASP Press, 2004
44. Wolf CR, Smith G, Smith RL: Science, medicine, and the future: pharmacogenetics. *BMJ* 2007, 320:987-990
45. Fishbain DA, Fishbain D, Lewis J, Cutler RB et al: Genetic testing for enzymes of drug metabolism: does it have clinical utility for pain medicine at the present time? A structured review. *Pain Med* 2004; 5:81-93
46. Vlaeyen JW, Linton SJ: Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. *Pain* 2000; 85:317-332
47. Picavet HS, Vlaeyen JW, Shouten JS: Pain catastrophizing and kinesiophobia: predictors of chronic low back pain. *Am J Epidemiol* 2002; 256:1028-1034
48. Boersma K, Linton SJ: Expectancy, fear and pain, in the prediction of chronic pain and disability: a prospective analysis. *Eur J Pain* 2006; 10:551-557
49. Ward LC, Thorn BE: The fear-avoidance model of chronic pain: further thoughts on the process of validating a causal model. *Pain* 2006; 121:173-174
50. Cook, AJ, Braweer PA, Vowles KE: The fear-avoidance model of chronic pain: validation and age analysis using structural equation modeling. *Pain* 2006; 121:195-206

51. Swinkels-Meewisse IEJ, Roelofs J, Oosterndorp RAB, Verbeek ALM, Blaeyen JWS: Acute low back pain: pain-related fear and pain catastrophizing influence physical performance and perceived disability. *Pain* 2006; 120:36-43
52. Meredith PJ, Strong J, Feeney JA: The relationship of adult attachment to emotion catastrophizing, control, threshold intolerance, in experimentally-induced pain. *Pain* 2006; 120:44-52
53. Goubert L, Crombez G, Van DS: The role of neuroticism pain catastrophizing and pain-related fear in vigilance to pain: a structural equations approach. *Pain* 2004; 107:234-241
54. Edwards RR, Smith MT, Kudel I, Haythornthwaite J: Pain-related catastrophizing as a risk factor for suicidal ideation in chronic pain. *Pain* 2006; 126:272-279
55. Vlieger PD, Crombez G, Eccleston C: Worrying about chronic pain. An examination of worry and problem solving in adults who identify as chronic pain sufferers. *Pain* 2006; 120:138-144
56. Davey GCL: Pathological Worrying as Exacerbated Problem-Solving, in *Worrying: Perspectives on Theory Assessment and Treatment*. Edited by Davey GCL, Tallis F. Chichester, Wiley, 1994
57. Aldrich S, Eccleston C, Crombez G: Worrying about chronic pain: vigilance to threat and misdirected problem solving. *Behav Res Ther* 2000; 38:457-470
58. Meredith P, Strong J, Feeney JA: Adult attachment, anxiety, and pain self-efficacy as predictors of pain intensity and disability. *Pain* 2006; 126:146-154
59. Turk DC, Okifuji A: Psychological factors in chronic pain: evolution and revolution. *J Consult Clin Psychol* 2002; 70:3:678-690
60. Taylor WJ, Dean SG, Seigert RJ: Differential association of general and health self-efficacy with disability, health-related quality of life and psychological distress from musculoskeletal pain in a cross-sectional general adult population survey. *Pain* 2006; 125:225-232
61. Karoly P, Ruhlman LS: Psychological "resilience" and its correlates in chronic pain: findings from a national community sample. *Pain* 2006; 123:90-97
62. Olson CA, Bond L, Burns JM, Vella-Broderick DA, Sawyer SM: Adolescent resilience: a conceptual analysis. *J Adoles* 2003; 26:1-11
63. Curtis WJ, Cicchetti D: Moving research on resilience into the 21st century: theoretical and methodological considerations in examining the biological contributors to resilience. *Dev Psychopathol* 2003; 15:773-810
64. Mercado AC, Carroll LJ, Cassidy JD, Cote P: Passive coping is a risk factor for disabling neck or low back pain. *Pain* 2005; 117:51-57
65. Brown GK, Nicassio PM: Development of a questionnaire for the assessment for active and passive coping strategies in chronic pain patients. *Pain* 1987; 31:53-64
66. McCracken LM, Eccleston C: A prospective study of acceptance of pain and patient functioning with chronic pain. *Pain* 2005; 118:164-169
67. Heapy A, Otis J, Marcus KS, Frantsve LM et al: Intercession coping skill practice mediates the relationship between readiness for self-management treatment and goal accomplishment. *Pain* 2005; 118: 360-368
68. Kut E, Schaffner N, Wittwer A, Candia V et al: Changes in self-perceived role identity modulate pain perception. *Pain* 2007; 131:191-201
69. Ochsner KN, Ludlow DH, Knierim K, Hanelin J et al: Neural correlates of individual differences in pain-related fear and anxiety. *Pain* 2006; 120:69-77
70. McCracken LM, Faber SD, Janek AS: Pain-related anxiety predicts non specific physical complaints in

- persons with chronic pain. *Behav Res Ther* 1998; 36:621-630
71. Peyron R, Laurent B, Garcia-Larrea L: Functional imaging of brain responses to pain: a review and meta-analysis. *Neurophysiol Clin* 2000; 30:263-288
 72. Apkarian AV, Chialvo DR: The shadows of pain. *Pain* 2006; 123:221-222
 73. Ohara S, Crone NE, Weiss N, Lenz FA: Analysis of synchrony demonstrates "pain networks" defined by rapidly switching, task-specific, functional connectivity between pain-related cortical structures. *Pain* 2006; 123:244-253
 74. Crombez G, VanDamme S, Eccleston C: Hypervigilance to pain: an experimental and clinical analysis. *Pain* 2005; 116:4-7
 75. McCracken LM, Carson JW, Eccleston C, Keefe FJ: Acceptance and change in the context of chronic pain. *Pain* 2004; 109: 4-7
 76. Polatin PB, Kinney RK, Gatchel RJ, Lillo E, Mayer TG: Psychiatric illness and chronic low back pain: the mind and the spine-which goes first? *Spine* 1993;18:66-71
 77. Bair MJ, Robinson RL, Katon W, Kroenke K: Depression and pain comorbidity: a literature review. *Arch Int Med* 2003; 163:2433-2445
 78. Twillman RK: Mental disorders in chronic pain patients. *J Pain Palliat Care Pharmacother* 2007; 21:4:13-19
 79. Demyttenaere K, Bruffaerts R, Lee S, Posada-Villa J et al: Mental disorders among persons with chronic back or neck pain: results from the world mental health surveys. *Pain* 2007; 129:332-342
 80. Lanteri-Minet M, Radad F, Chautard MH, Lucas C: Anxiety and depression associated with migraine: influence on migraine subjects' disability and quality of life, and acute migraine management. *Pain* 2005; 118:319-326
 81. Kivioja J, Sjaljan M, Lindgren U: Psychiatric morbidity in patients with chronic whiplash-associated disorder. *Spine* 2004; 29:11:1235-1239
 82. Wood PB: Treating comorbidities in fibromyalgia. *The Pain Practitioner* 2008; 18:1:42-48
 83. Arnow BA, Hunkeler EM, Blasey CM, Lee J: Comorbid depression, chronic pain, and disability in primary care. *Psychosom Med* 2006; 68:2:262-268
 84. Nicholas MK: Mental disorders in people with chronic pain: an international perspective. *Pain* 2007; 129:231-232
 85. Karp JF, Scott J, Houck P, Reynolds CF et al: Pain predicts longer time to remission during treatment of recurrent depression. *J Clin Psychiat* 2005; 66:591-597
 86. Haythorn-Thwaite JA: Clinical trials studying pharmacotherapy and psychological treatments alone and together. *Neurology* 2005; 65:20-31
 87. Blumer D, Heilbronn M: Chronic pain as a variant of depressive disease: the pain-prone disorder. *J Nerv Mental and Dis* 1982; 170:381-406
 88. VonKorff M, Crane P, Lane M, Miglioretti DL: Chronic spinal pain and physical- mental comorbidity in the United States: results from the national comorbidity survey replication. *Pain* 2005; 115:331-339
 89. Geuze E, Westenberg H, Jochim SA, de Kloet CS et al: Altered pain processing in veterans with post traumatic stress disorder. *Arch Gen Psychiatry* 2007; 64:76-85
 90. Palyo SA, Beck JG: Post traumatic stress disorder symptoms, pain, and perceived life control: associations with psychosocial and physical functioning. *Pain* 2005; 117:121-127
 91. Sharp TJ, Harvey AG: Chronic pain and post traumatic stress disorder: mutual maintenance? *Clin*

Psychol Rev 2001; 21:857-877

92. Currie SR, Wang J: More data on major depression as an antecedent risk factor for first onset of chronic back pain. *Psychol Med* 2005; 35:9:1275-1282
93. Weisberg J: Personality and personality disorders in chronic pain. *Curr Rev Pain* 2000; 4:60-70
94. Fishbain DA, Goldberg M, Meagher BR, Steele R, Rosomoff H: Male and female chronic pain patients categorized by the DSM-III psychiatric diagnostic criteria. *Pain* 1986; 26:181-197
95. Riech J, Tupin JP, Abramowitz SI: Psychiatric diagnosis of chronic pain patients. *Am J Psych* 1983; 140:11:1495-1498
96. Riech J, Thompson D: DSM-III personality disorder clusters in three populations. *British Journal of Psychiatry* 1987; 150:471-475
97. Weisberg JN: Studies Investigating the Prevalence of Personality Disorders in Patients with Chronic Pain, in *Personality Characteristics of Patients with Pain*. Edited by Gatchel RJ, Weisberg JN. Washington, DC, American Psychological Association, 2000
98. Polatin PB, Kinney RK, Gatchel RJ, Lillo E, Mayer TG: Psychiatric illness and chronic low back pain. The mind and the spine-which goes first? *Spine* 1993; 18:1:66-71
99. Gatchel RJ, Garofalo JP, Ellis E, Holt C: Major psychological disorders in acute and chronic TMD: an initial examination. *Journal of the American Dental Association* 1996; 127:1365-1374
100. Monti D, Herring C, Schwartzman R, Marchese M: Personality assessment of patients with complex regional pain syndrome type I. *Clinical Journal of Pain* 1998; 14:4:295-302
101. Gamsa A: The role of psychological factors in chronic pain I: a half century of study. *Pain* 1994; 57:5-15
102. Gamsa A: The role of psychological factors in chronic pain II: a critical appraisal. *Pain* 1994; 57:17-29
103. Nemiah JC, Sifneos PE: Affect and Fantasy in Psychosomatic Disorders, in *Modern Trends in Psychosomatic Medicine*. Edited by Hill O. London, Butterworth, 1970
104. Sifneos PE: The prevalence of alexithymic characteristics in psychosomatic patients. *Psychotherapy and Psychosomatics* 1973; 22:255-262
105. Phillips JM, Gatchel RJ: Extraversion-Introversion and Chronic Pain, in *Personality Characteristics of patients with pain*. Edited by Gatchel RJ, Weisberg JN. Washington, DC, American Psychological Association, 2000.
106. Garofalo JP: Perceived Optimism and Chronic Pain, in *Personality Characteristics of Patients with Pain*. Edited by Gatchel RJ, Weisber JN. Washington, DC, American Psychological Association, 2000
107. Weisberg JN, Keefe FJ: Personality disorders in the chronic pain population: basic concepts, empirical findings, and clinical implications. *Pain Forum* 1997; 6:1:1-9
108. Conrad R, Schilling G, Bausch C, Nadstawekj et al: Temperament and character personality profiles and personality disorders in chronic pain patients. *Pain* 2007; 133:197-209
109. Weisberg JN, Boatwright BA: Mood, anxiety and personality traits and states in chronic pain. *Pain* 2007; 133:1-2
110. Kellner R: *Somatization and Hypochondriasis*. New York, Praeger Publishers, 1986
111. Binswanger O: *Die Hysterie*. Vienna, Holder, 1904
112. Singer BH, Ryff CD: *New Horizons in Health: an Integrative Approach*. Washington, DC, National Academies Press, 2001

113. denBoer JJ, Oostendorp RAB, Beems T, Munneke M, Evers AWM: Continued disability and pain after lumbar disc surgery: the role of cognitive-behavioral factors. *Pain* 2006; 123:45-52
114. Pincus T, Burton AK, Vogel S, Field AP: A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. *Spine* 2002; 27:109-120
115. Klein T, Magerl W, Treede RD: Forget about your chronic pain. *Pain* 2007; 132:16-17
116. Choi DS, Choi DY, Whittington RA, Nedeljkovic SS: Sudden amnesia resulting in pain relief: the relationship between memory and pain. *Pain* 2007; 132:206-210
117. McWilliams LA, Asmundson GJG: The relationship of adult attachment dimensions to pain-related fear, hypervigilance, and catastrophizing. *Pain* 2007; 127:27-34
118. Meredith P, Strong J, Feeney JA: Adult attachment, anxiety, and pain self-efficacy as predictors of pain intensity and disability. *Pain* 2006; 123:146-154
119. Meredith PJ, Strong J, Feeney JA: Evidence of a relationship between adult attachment variables and appraisals of chronic pain. *Pain Res Manag* 2005; 10:191-200
120. Ciechanowski P, Sullivan M, Jensen M, Romano J, Summer H: The relationship of attachment style to depression, catastrophizing and health care utilization in patients with chronic pain. *Pain* 2003; 104:627-637
121. Porter LS, Davis D, Keefe FJ: Attachment and pain: recent findings in future directions (Topical Review). *Pain* 2007; 128:195-198
122. Gundel H, Tolle TR: How Physical Pain May Interact with Psychological Pain: Evidence for a Mutual Neurobiological basis of Emotions and Pain, in *Narrative, Pain, and Suffering*. Edited by Carr DB, Loeser JD, Morris DB. Seattle, IASP Press, 2005
123. Damasio AR, Grabowski TJ, Bechara A, et al: Subcortical and cortical brain activity during the feeling of self-generated emotions. *Journal of Nat Neurosci* 2000; 3:1049-1156
124. Greco M: Inconspicuous anomalies: alexithimia and ethical relations to the self. *Health* 2001; 5:4:471-492
125. Simon GE, VonKorff M, Piccinelli M, Fullerton NC, Ormel J: An international study of the relation between somatic symptoms and depression. *N Engl J Med* 1999; 341:18:1329-1335
126. Sachs-Ericsson N, Kendall-Tackett K, Hernandez A: Childhood abuse, chronic pain, and depression in the National Comorbidity Survey. *Child Abuse Negl* 2007; 31:5:531-547
127. Heim C, Wagner D, Maloney E, Papanicolaou et al: Early adverse experience and risk for chronic fatigue syndrome: results from a population-based study. *Arch Gen Psychiatry* 2006; 63:1258-1266
128. Dube SR, Felitti VJ, Dong M, Giles WH, Anda RF: The impact of adverse child experiences on health problems: evidence from four birth cohorts dating back to 1900. *J Prev Med* 2003; 37:268-277
129. Chapin DP, Whitfield CL, Felitti VJ, Dube SR et al: Adverse childhood experiences and the risk of depressive disorders in adulthood. *J Affect Disord* 2004; 82:217-225
130. Ladd CO, Huot RL, Thivikraman KV, Nemeroff CB et al: Long-term behavioral and neuroendocrin adaptations to adverse early experience. *Prog Brain Res* 2000; 122:81-103
131. Meaney MJ: Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annu Rev Neurosci* 2000; 24:1161-1192
132. Gracely RH, Petzke F, Wolf JM, Clauw DJ: Functional magnetic resonance imaging evidence of augmented pain processing in fibromyalgia. *Arthritis Rheum* 2002; 46:5:133-134
133. Giesecke T, Gracely RH, Grant MA et al: Evidence of augmented central pain processing in idiopathic chronic low back pain. *Arthritis Rheum* 2004; 50:2:613-623

134. Vogt BA, Berger GR, Derbyshire SW: Structural and functional dichotomy of human midcingulate cortex. *Eur J Neurosci* 2003; 18:11:3134-3144
135. Lane RD: Neural Correlates of Conscious Emotional Experience, in *Cognitive Neuroscience of Emotion*. Edited by Lane RD, Nadel L. New York, Oxford University Press, 2000
136. Lane RD, Schwartz GE: Levels of emotional awareness: a cognitive-developmental theory and its application to psychopathology. *Am J P* 1987; 14:133-143
137. Craig AD: A new view of pain as a homeostatic emotion. *Trends in Neurosciences* 2003; 26:6:303-307
138. Craig AD: How do you feel? Interoception: the sense of the physiological condition of the body. *Nat Rev Neurosci* 2002; 3:655-666
139. Lane RD, Quinlan DM, Schwartz GE, Walker PA, Zeitlin SB: The Levels of Emotional Awareness Scale: a cognitive developmental measure of emotion. *Journal of Personality Assessment* 1990; 55:1&2:124-134
140. Blyth FM, MacFarlane GJ, Nicholas MK: The contribution of psychosocial factors to the development of chronic pain: the key to better outcomes for patients? *Pain* 2007; 129:8-11
141. Bongers PM, Kremer AM, terLaak J: Are psychosocial factors risk factors for symptoms and signs of the shoulder, elbow, or hand/wrist?: a review of epidemiological literature. *Am J Ind Med* 2002; 41:315-342
142. Newton-John TR, Williams ACC: Chronic pain couples: perceived marital interactions and pain behaviours. *Pain* 2006; 123:53-63
143. Leonard MT, Cano A: Pain affects spouses too: personal experience with pain and catastrophizing as correlates of spouse distress. *Pain* 2006; 126:139-146
144. Goubert L, Craig KD, Vervoort T, Morley S et al: Facing others in pain: the effects of empathy. *Pain* 2005; 118:285-288
145. Jackson PL, Rainville P, Decety J: To what extent do we share the pain of others? insight from the neural bases of pain empathy. *Pain* 2006; 125:5-9
146. Coste J, Lefrancois G, Guillemin F, Pouchot J: French Study Group for Quality of Life in Rheumatology. Prognosis and quality of life in patients with acute low back pain: insights from a comprehensive inception cohort study. *Arthritis Rheum* 2004; 51:168-176
147. Shibnall JT, Tait RC, Andresen EM, Hadler NM: Race and socioeconomic differences in post-settlement outcomes for African American and Caucasian workers' compensation claimants with low back injuries. *Pain* 2005; 114:462-472
148. Crauford DIO, Creed F, Jayson MIV: Life events and psychological disturbance in patients with low-back pain. *Spine* 1990; 15:490-494
149. Bigos SJ, Battie MC, Spengler DM et al: A prospective study of work perceptions and psychosocial factors affecting the report of back injury. *Spine* 1991; 16:1-6
150. Drukteinis AM: The role of suggestibility in mental damage claims. *American Journal of Forensic Psychiatry* 2005; 26:1:15-35
151. Drukteinis AM: Overlapping somatoform syndromes in personal injury litigation. *American Journal of Forensic Psychiatry* 200; 21:4:37-66
152. Bishop A, Thomas E, Foster NE: Healthcare practitioners' attitudes and beliefs about low back pain: a systematic search and critical review of available measurement tools. *Pain* 2007; 132:91-101
153. Coes BW, vanTulder MW, Ostelo R, Kim BA, Waddell G: Clinical guidelines for the management of low

- back pain in primary care: an international comparison. *Spine* 2001; 26:2504-2513
154. Zborowski M: Cultural components in responses to pain. *Journal of Social Issues* 1952; 18:16-30.
 155. Edwards RR, Doleys DM, Fillingim RB, Lowery D: Ethnic differences in pain tolerance: clinical implications in chronic pain population. *Psychosom Med* 2001; 63:316-323
 156. Rahim-Williams FB, Riley JL, Herrera D, Campbell CM et al: Ethnic identity predicts experimental pain sensitivity in African Americans and Hispanics. *Pain* 2007; 129:177-184
 157. Bates MS, Edwards WT, Anderson KO: Ethnocultural influences on variation in chronic pain perception. *Pain* 1193; 52:101-112
 158. Shorter E: *From Paralysis to Fatigue: A History of Psychosomatic Illness in the Modern Era*. New York, The Free Press, 1992
 159. Morris DB: *The Culture of Pain*. Berkeley, University of California Press, 1991
 160. Saul LJ: *Psychodynamically-Based Psychotherapy*. New York, Science House, 1972.
 161. Greenson RR: *The Technique and Practice of Psychoanalysis*. New York, International Universities Press, 1967
 162. Alexander F: *Psychoanalysis and Psychotherapy*. New York, W.W. Norton, 1956.
 163. Charon R, Montello M: *Stories Matter: The Role of Narrative in Medical Ethics*. New York, Routledge, 2002.
 164. Labus JS, Keefe FJ, Jensen MP: Self-reports of pain intensity and direct observations of pain behavior: when are they correlated? *Pain* 2003; 102:1-2:109-124
 165. Pilowsky I: A general classification of abnormal illness behaviours. *British Journal of Medical Psychology* 1978; 51:131-137
 166. Waddell G: *The Back Pain Revolution*. Edinburgh, Churchill Livingstone, 1998
 167. Sullivan MJL, Thibault P, Savard A, Catchlove R et al: The influence of communication goals and physical demands on different dimensions of pain behavior. *Pain* 2006; 125:270-277
 168. Jolliffe C, Nicholas M: Verbally reinforcing pain reports: a experimental test of the operant model of chronic pain. *Pain* 2004; 107:167-175
 169. Williams AC: Facial expression of pain: an evolutionary account. *Behav Brain Sci* 2002; 25:439-488
 170. Disease, disability and injury prevention and control Special Emphasis Panel (SEP): Genomic Applications in Practice and Prevention: Translation programs in education, surveillance, and policy; Program Announcement (PA) #GD08-801. Centers for Disease Control and Prevention Documents and Publications, July 14, 2008, Centers for Disease Control and Prevention "Regulatory Documents
 171. Beck A: *Depression: Clinical, Experimental, and Theoretical Aspects*. New York, Harper and Rowe, 1967
 172. Spielberger CD: *State-Trait Anxiety Inventory: Professional Testing Resources*. Lutz FL, Psychological Assessment Resources 2007.
 173. Derogatis LR: *Symptoms Checklist-90R*. Clinical Psychometric Research, Towson, MD, 1983
 174. Morey LC: *Personality Assessment Inventory*. Western Psychological Services. Los Angeles CA, WPS, 2007
 175. Costa PT, McCray RR: *Revised NEO Personality Inventory (NEO PI-R) and NEO Five-Factor Inventory (NEO-FFI): Professional Manual*. Odessa, FL, Psychological Assessment Resources, 2007

176. Greene RL, Brown RC: Psychological Assessment Resources. Lutz FL, PAR, 2007
177. Turk DC, Melzak R (eds.): Handbook of Pain Assessment, Second Edition. New York, Guilford Press, 2001
178. Melzack R: The McGill Pain Questionnaire, in Pain Measurement and Assessment. Edited by Melzack R. New York, Raven Press, 1983
179. Sullivan MJ, Bishop SR, Pivik J: The Pain Catastrophizing Scale: development and validation. Psychol Assess 1995; 7:524-532
180. Clark ME, Kori SH, Brockel J: Kinesiophobia and chronic pain: psychometric characteristics and factor analysis of the Tampa Scale. Am Pain Soc Abstracts 1996
181. Taylor GJ, Ryan D, Bagby RM: Toward the development of a new self-report alexithimia scale. Psychotherapy and Psychosomatics 1986; 44:4:191-199
182. Lane RD, Quinlan DM, Schwartz GE, Walker PA, Zeitlin SB. The Levels of Emotional Awareness Scale: A cognitive-developmental measure of emotion. J Pers Assess 1990; 55:1-2:124-134
183. Fairbank JCT, Couper J, Davies JB, O'Brien JP: The Oswestry low-back pain disability questionnaire. Physiotherapy 1980; 66:271-273
184. Bergner M, Bobbitt RA, Carter WB, Gilson BS: The Sickness Impact Profile: development and final revision of a health status measure. Med Care 1981; 19:787-805
185. TerKuile M, Linssen A, Spinhoven P: The development of the multidimensional locus of pain control questionnaire (MLPC): factor structure, reliability, and validity. Journal of Psychopathology and Behavioral Assessment 2003; 15:3: 387-404
186. Nicholas MK: Pain Self-Efficacy Questionnaire (PSEQ): Preliminary Report. University of Sydney Pain Management and Research Centre, St. Leonards, 1994
187. Turk D, Rudy T: Toward an empirically derived taxonomy of chronic pain patients: integration of psychological assessment data. Journal of Consulting and Clinical Psychology 1988; 56:233-238
188. Jensen MP, Turner JA, Romano JM, Strom SE: Chronic Pain Coping Inventory: development and preliminary validation. Pain 1995; 60:203-216
189. Ruelhman LS, Karoly P, Newton C, Aiken LS: The development and preliminary validation of the Profile of Chronic Pain: Extended Assessment Battery. Pain 2005; 118:380-389
190. 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
191. Millon T, Green C, Meagher R: The MBHI: a new inventory for the psychodiagnostician in medical settings. Professional Psychology 1979; 10:529-539
192. Croft P, Dunn KM, VonKorff M: Chronic pain syndromes: you can't have one without another. Pain 2007; 131:237-238
193. Koes BW, VanTulder MW, Ostello R, Waddell G: Clinical guidelines for the management of low back pain in primary care: an international comparison. Spine 2001; 26:2504-2513
194. denBoer JJ, Oostendorp RAB, Beems T, Munneke M et al: A systematic review of bio-psychosocial risk factors for an unfavorable outcome after lumbar disk surgery. Eur Spine J 2006; 15:527-536
195. Linton SJ: A review of psychological risk factors in back and neck pain. Spine 2000; 25:9:1148-1156
196. Grotle M, Brox JI, Veierod MB, Vollestad NK: Clinical course and prognostic factors in acute low back: patients seeking help for their first time. Spine 2005; 30:8:976-982
197. Linton SJ: Occupational psychological factors increase the risk for back pain: a systematic review. J

Occup Rehabil 2001; 11:1:53-66

198. Wolfe F: Fibromyalgia. *Rheumatological Disease Clinics of North America* 1990; 16:3:681-698
199. Cohen ML, Quintner JL: Fibromyalgia syndrome, a problem of tautology. *Lancet* 1993; 342:906-909
200. Vierck Jr. CJ: Mechanisms underlying development of spatially distributed chronic pain (fibromyalgia). *Pain* 2006; 124:242-262
201. Bohr TW: Fibromyalgia syndrome and myofascial pain syndrome, do they exist? *Neurologic Clinics* 1995; 13:2:365-384
202. Janig W, Stanton-Hicks M (eds.): *Reflex Sympathetic Dystrophy: A Reappraisal*. Seattle, IASP Press, 1996
203. Allen G, Galer BS, Schwartz L: Epidemiology of complex regional pain syndrome: a retrospective chart review of 134 patients. *Pain* 1999; 80:539-544
204. Helfenstein M, Feldman D: The pervasiveness of the illness suffered by workers seeking compensation for disabling arm pain. *Journal of Occupational and Environmental Medicine* 2000; 42:2:171-175
205. Cleland LG: RSI: A model of social iatrogenesis. *Medical Journal of Australia* 1987; 147:236-239
206. Quinlan KP, Annest JL, Myers B, Ryan G, Hill H: Neck strains and sprains among motor vehicle occupants-United States, 2000. *Accid Anal Prev* 2004; 36:21-27
207. Barnsley L, Lord S, Bogduk N: Clinical review, whiplash injury. *Pain* 1994; 58:283-307
208. Berglund A, Bodin L, Jensen I, Wiklund A, Alfredsson L: The influence of prognostic factors on neck pain intensity, disability, anxiety and depression over a 2-year period in subjects with acute whiplash injury. *Pain* 2006; 125:244-256
209. Hendricks EJM, Scholten-Peeters GGM, van der Windt DAWM, Neeleman-van der Steen CWM et al: Prognostic factors for poor recovery in acute whiplash patients. *Pain* 2005; 114:408-416
210. Sterling M, Jull G, Kenardy J: Physical and psychological factors maintain long-term predictive capacity post-whiplash injury. *Pain* 2006; 122:102-108
211. Schrader H, Obeliene D, Bovin G, Surkiene D et al: Natural evolution of last whiplash syndrome outside the medical legal context. *Lancet* 1996; 347:1207-1211
212. Cassidy JD, Carroll LJ, Cote P, Lemstra M et al: Effect of eliminating compensation for pain and suffering on the outcome of insurance claims for whiplash injury. *New England Journal of Medicine* 2000; 342:16:1179-1186
213. Chapman CR: Pain, Perception, and Illusion, in *The Psychology of Pain*, Second Edition. Edited by Sternbach RA. New York, Raven Press, 1986
214. Bennett MI, Attal N, Backonja MM, Baron R et al: Using screening tools to identify neuropathic pain (Topical Review). *Pain* 2007; 127:199-203
215. Hall GC, Carroll D, Parry D, McQuay HJ: Epidemiology and treatment of neuropathic pain: the UK primary care perspective. *Pain* 2006; 122:156-162
216. Weingarten TN, Watson JC, Hooten WM, Woolan PC et al: Validation of the S-LANSS in community setting. *Pain* 2007; 132:189-194
217. Bennett MI, Bouhassira D: Epidemiology of neuropathic pain: can we use the screening tools? *Pain* 2007; 132:12-13
218. Saal JA, Saal JS: Physical Medicine and Rehabilitation as it Relates to Pain Management, in *Innovations in Pain Management*. Edited by Weiner RS. Winter-Park FL, Paul M. Deutsch Press, 1992

219. Sjogren T, Nissinen KJ, Jarvenpaa SK, Ojanen MT et al: Effects of a workplace physical exercise intervention on the intensity of headache and neck and shoulder symptoms and upper extremity muscular strength of office workers: a cluster randomized controlled cross-over trial. *Pain* 2005; 116:119-128
220. Brox JI, Reikera SO, Nygaard O, Sorensen R et al: Lumbar instrumented fusion compared with cognitive intervention and exercises in patients with chronic back pain after previous surgery for disk herniation: a prospective randomized controlled study. *Pain* 2006; 122:145-155
221. Vas J, Perea-Milla E, Mendez C, Navarro CS et al: Efficacy and safety for acupuncture for chronic uncomplicated neck pain: a randomized controlled study. *Pain* 2006; 126:245-255
222. Witt CM, Jena S, Brinkhaus B, Liecker B et al: Acupuncture for patients with chronic neck pain. *Pain* 2006; 125:98-106
223. Willich SN, Reinhold T, Selim D, Jena S et al: Cost-effectiveness of acupuncture treatment in patients with chronic neck pain (no other info dictated)
224. Berman BM: Seminal studies in acupuncture research. *J Altern Complement in Med* 2001; 7:Suppl 1:S129-137
225. Kirsch DL: Electromedicine: the Other Side of Physiology, in *Pain Management: A Practical Guide for Clinicians*. Edited by Weiner RS. Boca Raton, FL, CRC Press, 2002.
226. Barolat G: Spinal cord stimulation for chronic pain management. *Arch Med Res* 2000; 31:3:258-262
227. Taylor RS, Van Buyten JP, Buchser E: Spinal cord stimulation for chronic back and leg pain and failed back syndrome: a systematic review of prognostic factors. *Spine* 2005; 30:1:152-160
228. Kovac SH, Saag KG, Curtis JR, Allison J: Association of Health-related Quality of Life with Dual Use of Prescription and Over-the-Counter Nonsteroidal Anti-inflammatory Drugs. *Arthritis Rheum* 2008; 59:2:227-233
229. Caudill-Slosberg M, Schwartz L, Woloshin S: Office visits and analgesic prescriptions for musculoskeletal pain in US: 1980 versus 2000. *Pain* 2004; 109:514-519
230. Ballantyne JC, Laforge KS: Opioid dependence and addiction during opioid treatment of chronic pain. *Pain* 2007; 129:235-255
231. Reddy BS: The epidemic of unrelieved chronic pain: the ethical, societal, and regulatory barriers facing opioid prescribing physicians. *Journal of Legal Medicine* 2006; 27:427-442
232. Portenoy RK: Chronic opioid therapy in nonmalignant pain. *Journal of Pain and Symptom Management* 1990; 5:546-562
233. de C Williams AC: Psychological distress and opioid efficacy: more questions than answers. *Pain* 2005; 117:245-246
234. Wasan AD, Devar G, Jamison R: The association between negative affect and opioid analgesia in patients with discogenic low back pain. *Pain* 2005; 117:450-461
235. Edlund MJ, Steffick D, Hudson T, Harris KM, Sullivan M: Risk factors for clinically recognized opioid abuse and dependence among veterans using opioids for chronic non-cancer pain. *Pain* 2007; 129:355-362
236. Mao J: Opioid-induced abnormal pain sensitivity: implications in clinical opioid therapy (Topical Review). *Pain* 2002; 100:213-217
237. Liang DY, Guo TZ, Liao G, Kingery WS et al: Chronic pain and genetic background interact and influence opioid analgesia, tolerance, and physical dependence. *Pain* 2006; 121:232-240
238. Ballantyne JC: Opioids for chronic pain: taking stock. *Pain* 2006; 125:3-4

239. Eriksen J, Sjogren P, Bruera E, Ekholm O, Rasmussen NK: Critical issues on opioids in chronic non-cancer pain: an epidemiological study. *Pain* 2006; 125:172-179
240. Cedarbaum JM, Schleifer LS: *Drugs for Parkinson's Disease, Spasticity, and Acute Muscle Spasms*, in Goodman and Gilman's *The Pharmacological Basis of Therapeutics*, Eighth Edition. Edited by Gilman AG. New York, Pergamon Press, 1990
241. Dworkin RH, O'Connor AB, Backonja M, Farrar JT et al: Pharmacologic management of neuropathic pain: evidence-based recommendations. *Pain* 2007; 132:237-251
242. Watson A, El-Dereby W, Bentley DE, Vogt BA, Jones AKB: Categories of placebo response in the absence of site-specific expectation of analgesia. *Pain* 2006; 126:115-122
243. Ploghaus A, Becerra L, Borras C, Borsook D: Neural circuitry underlying pain modulation: expectation, hypnosis, placebo. *Cogn Sci* 2003; 17:197-200
244. Vase L, Robinson ME, Verne GN, Price DD: Increased placebo analgesia over time in irritable bowel syndrome (IBS) patients is associated with desire and expectation but not endogenous opioid mechanisms. *Pain* 2006; 126:115-122
245. Price DD, Cragg SJ, Verne GN, Perlstein WM, Robinson ME: Placebo analgesia is accompanied by large reductions in pain-related brain activity in irritable bowel syndrome patients. *Pain* 2007; 127:63-72
246. Finniss DG, Benedetti F: Placebo analgesia, nocebo hyperalgesia. *Pain: Clinical Updates (IASP)* 2007; 15:1:1-4
247. Petrovic P: Placebo analgesia and nocebo hyperalgesia: two sides of the same coin? *Pain* 2008; 136:5-6
248. Colloca L, Sigauco M, Benedetti F: The role of learning in nocebo and placebo effects. *Pain* 2008; 136:211-218
249. Chen E, Cole SW, Kato PM: A review of empirically supported psychosocial interventions for pain and adherence outcomes in sickle cell disease. *J Pediatr Psychol* 2004; 29:197-209
250. Morley S, Eccleston C, Williams A: Systematic review in meta-analysis of randomized controlled trials of cognitive behaviour therapy and behaviour therapy for chronic pain in adults, excluding headache. *Pain* 1999; 80:1-13
251. Turner JA, Mancl L, Aaron LA: Brief cognitive-behavioral therapy for temporomandibular joint disorder pain: effects on daily electronic outcome and process measures. *Pain* 2005; 117:377-387
252. Turner JA, Mancl L, Aaron LA: Short-and long-term efficacy of brief cognitive-behavioral therapy for patients with chronic temporomandibular joint disorder pain: a randomized, controlled trial. *Pain* 2006; 121:181-194
253. McCracken LM, Yang SY: The role of values in a contextual cognitive-behavioral approach to chronic pain. *Pain* 2006; 123:137-145
254. McCracken LM, Vowles KE, Eccleston C: Acceptance-based treatment for persons with complex, longstanding chronic pain: a preliminary analysis of treatment outcome in comparison to a waiting phase. *Behav Res Ther* 2005; 43:1335-1346
255. Baer RA, Krietemeyer J: *Overview of Mindfulness-and Acceptance-Based Treatment Approaches*, in *Mindfulness-Based Treatment Approaches: Clinicians Guide to Evidence Base and Applications*. Edited by Baer RA. London, Academic Press, 2006
256. McCracken LM, Gauntlett-Gilbert J, Vowles KE: The role of mindfulness in a contextual cognitive-behavioral analysis of chronic pain-related suffering and disability. *Pain* 2007; 131:63-69
257. Fernros L, Furhoff AK, Wandell PE: Improving quality of life using compound mind-body therapies: evaluation of a course intervention with body movement and breath therapy, guided imagery, chakra

- experiencing and mindfulness medication. *Quality of Life Research* 2008; 17:3:367-376
258. Schwartz MS (ed.): *Biofeedback: A Practitioner's Guide*. New York, The Guilford Press, 1987
 259. Keefe FJ, Block AR, Williams RB, Surwit RS: Behavioral treatment of chronic low back pain: clinical outcome and individual differences in pain relief. *Pain* 1981; 11:221-231
 260. Hassett AL, Radvanski DC, Vaschillo EG, Vaschillo B et al: A pilot study of the efficacy of heart rate variability (HRV) biofeedback in patients with fibromyalgia. *Applied Psychophysiology and Biofeedback* 2007; 32:1:1-10
 261. Achterberg J: *Imagery in Healing*. Boston, New Science Library, 1985
 262. Hilgard ER, Hilgard JR: *Hypnosis in the Relief of Pain, Revised Edition*. Los Altos, CA, William Kaufmann, 1983
 263. DePascalis V, Cacace I, Massicotte F: Focus analgesia in waking and hypnosis: effects on pain, memory, and somatosensory event-related potentials. *Pain* 2008; 134:197-208
 264. Anagnostis C, Gatchel RJ, Mayer TG: The Pain Disability Questionnaire: a new psychometrically sound measure for chronic musculoskeletal disorders. *Spine* 2004; 29:2290-2302
 265. Drukteinis AM: Disability. *Textbook of Forensic Psychiatry* (Simon R, Gold L Eds). Washington DC, APA Press, 2004
 266. Dobbs BD: *The Law of Torts*. St. Paul, MN, West Group, 2001
 267. *Colbert v. Samsons Supermarkets Inc.*, 44 A2d 433, Nevada, 1982
 268. *Miley v. Landry*, 582 So.2d 833, Louisiana, 1991
 269. Zachar P, Kendler KS: Psychiatric disorders: a conceptual taxonomy. *Am J Psychiatry* 2007; 164:557-565
 270. Larson A: *The Law of Workers' Compensation*. New York, Matthew Bender Press, 1972 (1993 supp.)
 271. Drukteinis AM: Personnel issues in workers' compensation stress claims. *American Journal of Forensic Psychiatry* 197; 18:3:3-23
 272. Sackett DL, Rosenberg WM, Gray JA, Haynes RB, Richardson WS: Evidenced based medicine: what it is and what it isn't. *British Medical Journal* 1996; 312:71-72
 273. *Daubert v. Merrell Dow Pharmaceuticals*, 509 US 579 (1993)
 274. Melhorn JM, Ackerman WE (eds.): *Guides to the Evaluation of Disease and Injury Causation*. Chicago, IL, American Medical Association, 2008
 275. Fishbain DA, Rosomoff HL, Cutler RB, Rosomoff RS: Secondary gain concept: a review of the scientific evidence. *Clinical Journal of Pain* 1995; 11:6-21
 276. Finch M: *Law and the Problem of Pain*. University of Cincinnati L REV 2005; 15:18:34
 277. Shorter E: *From Paralysis to Fatigue: A History of Psychosomatic Illness in the Modern Era*. New York, The Free Press, 1992
 278. Fishbain D, Cutler R, Rosomoff HL, Rosomoff RS: Chronic pain disability exaggeration/malingering and submaximal effort research. *Clin Journal of Pain* 1999; 15:4:244-274
 279. Rogers R (ed.): *Clinical Assessment of Malingering and Deception: Third Edition*. New York, The Guilford Press, 2008

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