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Psychological, Neuropsychological, and Medical Considerations in Assessment and Management of Pain

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Pain is a common yet challenging problem, particularly following traumatic injuries to the head or neck. It is a complex, multidimensional subjective experience with no clear or objective measures; yet it can have a significantly disabling effect across a wide range of functions. Persisting misconceptions owing to mind-body dualism have hampered advances in its understanding and treatment. In this article, a conceptualization of pain informed by recent research and derived from a more useful biopsychosocial model guides discussion of relevant medical, psychological, and neuropsychological considerations. This pain process model explains chronicity in terms of hyperresponsiveness and dysregulation of inhibitory or excitatory pain modulation mechanisms. Related neurocognitive effects of chronic pain are examined and recommendations for minimizing its confounding effects in neuropsychological evaluations are offered. A biopsychosocial assessment model is presented to guide understanding of the myriad of factors that contribute to chronicity. A brief survey of general classes and samples of the more useful pain assessment instruments is included. Finally, this model offers a rational means of organizing and planning individually tailored pain interventions, and some of the most useful pharmacologic, physical, and behavioral strategies are reviewed.
Key words: *assessment*, *biopsychosocial*, *brain injury*, *pain*, *treatment*

INTRODUCTION AND OVERVIEW: AN INFORMED CONCEPTUALIZATION OF PAIN

Pain, defined as an unpleasant sensory and emotional experience associated with real or potential tissue damage,[1] is a significant problem, particularly following traumatic injuries. Unfortunately, general medical and psychological thinking about pain is typically uninformed by recent specialty knowledge and research, and overly misinformed by recalcitrant myths, misconceptions, and false dichotomies.[2] The most pervasive and entrenched misconception from which most others emanate is mind-body dualism (see Nicholson and Martelli, in this issue, for discussion).

An important distinction, with associated differences in definition, pathophysiology, phenomenological experience, and also assessment and treatment focus, relates to chronicity. The subjective experience of pain, when acute, generally has more clearly identifiable triggers and neuroanatomic pathways,

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and communicates useful information that provokes adaptive responses. Resolution is expected with either natural healing or correction of underlying pathophysiology. As time passes without resolution, subjective components may become more pronounced, and responses more disproportionate to underlying pathophysiology and even obstructive to adaptation. Chronic pain can disrupt virtually all life areas and produce marked emotional and behavioral changes. As pain persists, it becomes more recalcitrant, typically resulting in multiple interventions and treatment goals that increasingly focus on improved coping with pain and its concomitants (eg, cyclic disability-enhancing patterns of overactivity-pain-underactivity).[3,4]

The fact that pain management remains a formidable challenge is due to not only complexities in assessment and management, but also persisting misconceptions due to mind-body dualism. Reductionistic definitions of pain in terms of peripheral triggers and pathology, as if it existed only in a peripheral body part, makes no more sense than defining vision as existing in the eye. There is now considerable evidence and general acceptance within the pain management field that chronic pain represents a central nervous dysregulation characterized by hyperexcitability, expansion of peripheral and central receptive fields with cerebral reorganization, often associated with hyperalgesia. That is, current evidence indicates that the psychoneurobiological substrate of chronic pain involves a sensitization or dysregulation of normal pain inhibitory mechanisms with hyperresponsiveness in the central (eg, medial pain system and limbic circuitry) and/or peripheral components of the nervous system[5–10] (see Nicholson and Martelli, and Walker, in this issue).

Nociceptive stimulation appears less responsible for production of pain than the widely distributed neural network underlying cognitive-evaluative, motivational-affective, and sensory-discriminative[11] as well as motor mnemonic, and social systems.[2,6] Moreover, increasing evidence implicating sensitization effects in the development and maintenance of many of the frequently comorbid conditions of posttraumatic head pain, such as posttraumatic stress syndrome and postconcussion disorder, is being offered.[12] The implications for pain management efforts seem clear. The implicit aim of assessment and treatment of acute pain must necessarily include the prevention of sensitization responses, while the aims for chronic pain must include the reversal of sensitization effects.

Pain experience is a multidimensional subjective experience mediated by beliefs, emotions, coping styles, and a variety of other perceptual influences. Responses to pain and pain treatments are typically variable and reflect complex biopsychosocial interactions between genetic, developmental, cultural, environmental, and psychological factors.[13,14] Adequately conceptualizing pain in this manner helps avoid the pitfalls of mind-body dualism and allows for consideration of the interaction of psychologic and organic factors in the presentation of any chronic pain patient.[2]

NEUROPSYCHOLOGICAL EFFECTS OF PAIN

As noted by Nicholson and Martelli in this issue, chronic pain and related problems can have a more disabling effect across a wider range of functions than brain or many other types of injuries. Impaired cognitive function is one of the frequently reported complaints of persons with chronic pain, especially head and neck pain. Recent reviews[4,15–19] have examined studies that objectively assess these complaints. The available evidence strongly supports the conclusion that chronic pain, especially head pain and neck pain, and pain-related symptomatology, independent of traumatic brain injury (TBI) or neurologic disorder, can and often do produce impairment of cognitive functioning. Multiple lines of evidence, including studies of acute and chronic pain, animal and human studies, experimental and clinical studies, and neurophysiologic studies support this conclusion. As assessed on neuropsychological tests, attentional capacity, processing speed, memory, and

executive functions are most likely to be affected.

Importantly, these reviews suggest that the concomitants of chronic pain may be the more important mediating variables. Specifically, cognitive impairment in chronic pain patients has been associated with mood change/emotional distress and with symptoms and clinical features such as increased somatic preoccupation, sleep disturbance, fatigue, and perceived interference with daily activities that are potential sources of chronic stress. These factors have been consistently found to be more strongly associated with both cognitive complaints and impairments than pain severity. Further, meta-analytic studies and increasing recent evidence have associated these individual factors with decrements in cognitive functioning, independent of chronic pain or combination effects.[15–19]

Hart, Wade, and Martelli[15] recently reviewed studies that examine cognitive functioning in patients with chronic pain, with an emphasis on the role of emotional distress and the mechanisms of stress-related effects. They found the association between psychological distress and cognitive impairment especially significant for pain-related negative emotions and variables that mediate suffering such as interference with activities and increased somatic vigilance. This relationship was independent of the effects of pain intensity. In examining possible underlying neurophysiologic substrates, they noted that the anterior cingulate cortex (ACC) plays an important role in pain processing and affective-motivational experience, and mediates the impact of pain-related emotional distress on cognitive functioning through allocation of attentional resources. In addition, maladaptive physiologic stress responses and dysregulation of the hypothalamic-pituitary-adrenocortical (HPA) axis can produce negative effects on hippocampal function and memory. They postulated that the underlying mechanism for cognitive impairment may be the anticipation of unpredictable pain symptoms, especially in individuals high in trait neuroticism, which presents a significant stressor that repeatedly activates both the HPA axis and ACC areas, thereby disrupting cognitive efficiency.

Importantly, there is a very high comorbidity of chronic pain problems with cranial trauma as well as TBI. Posttraumatic headache is the primary complaint in postconcussive disorders and in traumatic head and neck injury without brain injury. It has an estimated frequency as high as 90% in the first week and as high as 44% after 6 months.[4,16–19] In addition to headache, other posttraumatic pain problems can include back pain, complex regional pain syndrome, fractures, fibromyalgia, and others (see Ivanhoe in this issue). Further, pain problems are more frequently observed following milder TBI,[4,19–21] which represent 80% of all TBIs.

Head and neck injuries, with or without TBI, may result in increased vulnerability to delayed healing and peripheral and central sensitization effects in comparison with more severe TBI.[2,12] With more severe TBI, there may be promotion of more optimal posttraumatic physical healing (eg, musculature and ligamentous tissue) through longer periods of immobilization and/or early chemical paralysis[21] with associated anxiolytic effects and reduction of nociceptive cues and physiologic arousal. In addition, longer periods of loss of consciousness and posttraumatic amnesia likely reduce awareness and memory of pain early after injury, and may even result in reduced pain sensitivity resulting from acute or chronic lesions of the central nervous system (CNS) circuitry involved in processing pain, as is noted in some dementias.[5] With milder head and neck injuries, there is reduced opportunity for optimal physical healing, intact awareness and pain perception, and return to regular activity earlier and prior to physical healing versus severe injuries. Hence, there is likely increased opportunity for irritating unhealed tissue and increased vulnerability to effects of pain-related distress, anxiety and worry, and other psychological factors that can increase vigilance and physiological arousal and exacerbate pain experience.

The similarity in patterns of cognitive impairment observed with both chronic pain and TBI has important implications.[17,18] Chronic pain and associated problems can complicate the symptom picture in TBI, present a differential diagnostic challenge for especially milder TBI, and contribute to or maintain symptoms. Moreover, resolution of the postconcussive disorder and successful adaptation to residual sequelae frequently relies on successful coping with posttraumatic headache and/or other pain and associated symptomatology.[4,22] Careful consideration of pain in the differential diagnosis of brain injury is required. Some of the procedures recommended for neuropsychological assessment of persons with pain[17,18] have been included in Table 1.

ASSESSMENT OF PAIN

Pain is a subjective experience with no clear-cut objective measures. The complexity of this subjective and multidimensional experience is highlighted by independent replicated functional imaging studies showing that anticipation of pain produces similar activation of cortical networks as does pain itself[25] Not surprisingly, dualistic biomedical (ie, organic/biologic vs functional/psychologic) models that explain disease and health primarily in terms of measurable biological variables have not proven useful for measuring or treating pain. Biopsychosocial models better explain variability in healthcare outcomes and direct more effective interventions for challenging chronic healthcare situations, including chronic pain rehabilitation.[26] Notably, a biopsychosocial stress and coping formulation of postinjury adaptation conceptualizes adaptation to injury as a series of stressful demands, which require interactions between existing coping resources and injury-related stresses and demands. Identification of coping liabilities is an integral prerequisite to bolstering coping resources and improving long-term adaptation. A biopsychosocial assessment perspective affords the optimal understanding of the patient with pain and the myriad of factors that might contribute to this subjective experience and complicate, or otherwise affect, symptom presentation.[27] This understanding provides the framework for designing individually tailored treatment interventions and recommendations.

In addition, pain assessment and treatment strategies are best conceptualized in terms of suitability to a multidimensional process that evolves significantly as it persists over time. An illustrative model that distinguishes the sensory, affective, cognitive-evaluative, and behavioral dimensions of pain as it changes with increasing chronicity is the stages of pain processing model.[28–30] The first 2 stages involve sensory discrimination and associated affective responses. The former are commonly assessed by ratings of pain intensity, while the latter are assessed by ratings of pain unpleasantness. The third stage involves the meaning and implications of pain for the patient and associated emotional suffering, and is commonly assessed by measuring pain-related emotional states (eg, depression, anxiety, frustration) and beliefs (eg, perceived ability to control or endure pain). The fourth stage refers to illness behavior (eg, lifestyle and role disruption, avoidance) and can be assessed through self and collaborated ratings and observation (eg, pain behaviors manifested at home, work, clinical interview). As pain persists, there is a transition of the focus of assessment from the first 2 stages to the latter two.

Assessment of acute pain

The importance of pain assessment is demonstrated by references to pain as the fifth vital sign. In the case of pain occurring shortly after injury, there are often discrete triggers, identifiable noxious events, and possible tissue damage. The primary goal of assessment is identification of physical pain triggers and pain pathways. This guides treatment interventions aimed at resolving the causes of pain, either by promoting natural healing or correcting underlying pathophysiologic processes.

Because pain is subjective, the cornerstone of assessment is the patient's self-report.

Table 1. Recommendations for assessing and minimizing the confounding effects of pain during neurocognitive examinations

1. Chronic pain and associated symptoms represent a source of performance variance that must be considered as potential confounds in the interpretation of decrements in neuropsychological test performance.
2. Consideration should be given to postponing cognitive assessment in the too frequent cases where pain and related symptomatology have not yet received specific and appropriately aggressive treatment focus. Sleep may be an especially potent moderator variable for the effects of chronic pain on cognition,[23] and aggressive intervention (eg, sleep hygiene and pharmacologic and behavioral treatments) may be an appropriate prerequisite to evaluation.
3. Consideration should be given to alterations in test procedures to promote optimal performance and minimize discomfort and emotional distress (eg, comfortable seating/positioning, use of accustomed orthotics, cushions, heating/ice pads, optimized ergonomics, frequent breaks, frequent standing or position changes, modified lighting, etc).
4. Be prepared to assess (or refer for assessment to someone with specialty knowledge and experience) chronic pain and its concomitants when the complaint is salient, especially when limitations in everyday functioning and test performance seem atypical for the neurologic condition and/or given the suspicion that successful adaptation will depend upon coping with pain-related symptomatology.
5. The presence and severity (using Verbal or Visual Analogue or other scales) of pain should be assessed periodically throughout assessment when relevant. Other pain behaviors (eg, facial grimacing, moving, etc) and the intensity and frequency of these should be observed for possible relationship with variance in test performance, and when pain is the focus of attention versus when distracted by other discussion. The concordance of pain severity ratings and other pain behaviors should be assessed. Notably, it is probably not sufficient to simply measure the presence or intensity of pain at the time of evaluation as the cumulative effects of coping with chronic pain and associated symptoms appears to play a more important role.
6. Symptom checklists that assess the associated complaints of chronic pain (eg, fatigue, sleep disturbance) may be helpful. Collection of corroboratory data from relatives and others is advised, as is caution to avoid encouragement of symptom focus. The repeated administration of a sustained, attention-demanding, timed test at the end of a session may help identify or corroborate possible fatigue-related deficits.
7. Standard measures of mood and emotional-personality functioning, as well as pain-specific measures, should always be employed. Significant emotional distress and negative pain and illness-related beliefs, and lifestyle interference that seems inconsistent or disproportionate, should increase the level of caution in attributing performance decrements to brain dysfunction versus other causes.
8. Assessment of any response bias (pain as well as other complaints) should be conducted, not only to identify exaggeration, but to estimate effects of chronic pain on ability to sustain optimal or near optimal performance. Litigation effects should always be considered and pain inventories that address secondary gain and motivation should be employed, especially in compensation-seeking contexts.
9. Pain medications represent another possible moderator variable, and caution must be exercised in interpreting test results for persons who rely upon them for pain management. Simply instructing nonuse of medications during testing is not an adequate solution, because the effects of unmodulated chronic pain may be worse for some aspects of cognitive functioning than induced opiate analgesia.[24]

Assessment of important aspects of pain experience should include pain character, onset, location, duration, and factors that exacerbate or relieve it. Inquiry should also investigate pain frequency and intensity and interference with everyday activities, as well as pain-related distress associated with sensory disturbances, cognitive interpretations, affective distress, and behavioral avoidance responses. Useful adjunctive methods for assessing pain intensity in adults are the Visual Analogue Scale (VAS)[31] and the Verbal Analogue Scale. The Visual scale is a 10-cm line with anchors of "no pain" (or "no interference, etc) and "the most pain imaginable," while the Verbal Analogue Scale solicits rating of pain or associated features on a 0 to 10 scale with the same anchors. These scales are sensitive to variations in pain intensity, reactive distress, and treatment effects, and are widely used in clinical settings.

An important secondary goal of pain assessment is to identify potential complications or risk factors that can interfere with resolution of pain.[27,32] Because of the clearer association with injury and expectation for resolution, psychosocial factors are too often not considered until pain becomes subacute or chronic. However, the identification of emotional suffering, negative illness-related beliefs, and lifestyle interference that seems disproportionate to pain intensity, difficulty coping with pain and its associated symptoms, and resistance to benefit from treatment are critical areas in identifying risk factors for poor adjustment and risk of chronicity. That is, in addition to distress that is reactive to physical pathology, investigation of cognitive interpretations (eg, pervasiveness, sense of control over pain, expectations for treatment), level of affective distress (eg, anxiety, dysphoria), behavioral avoidance responses (eg, bracing or guarding behaviors in anticipation of pain and/or reduced activity levels leading to disuse atrophy), general coping repertoire (eg, physical, cognitive, and emotional coping skills as well as available social support systems), historical vulnerability factors (eg, childhood trauma) as well as environmental factors, such as secondary gain, represent potentially important factors that can mediate adaptation and outcome.

Some of the most commonly reported risk factors for poor postinjury adaptation that represent red flags that should alert professionals to vulnerabilities in coping were identified by review of the literature.[27,32,33] These factors that may trigger need for early and specific intervention aimed at bolstering coping skills and resources: high levels of anger, resentment, or perceived mistreatment; high levels of premorbid and/or postinjury anxiety; depression; catastrophizing or helpless/hopeless thoughts; childhood trauma/abuse; hysterical and hypochondriacal personality traits; premorbid perfectionistic tendencies, high trait social desirability, or other personality characteristics; extreme pain-suppressive behavior (to endure activities); extreme avoidance behavior; irrational fear of re-injury or pain with activity (kinesiophobia); external locus of control; reduced self-confidence and self-efficacy, and fear of failure or rejection associated with residual impairments and/or fear of losing disability status, benefits; discrepancies between personality/premorbid coping style and injury consequences (eg, highly physically active person with limited intellectual resources who has a back injury); low residual coping resources and skills; significant preinjury job dissatisfaction and/or stress; sociopathic personality traits; greater reinforcement for "illness" vs "wellness" behavior; perceptions of high compensability for injury; misdiagnosis and late diagnosis, and delayed treatment; insurance resistance or delays in authorizing treatment or paying bills; and previous treatment failures with increased duration of complaints. Behavioral observations are also an important component in pain assessment that provides additional information about consistency of report and response style and response biases.

Some of the most common behavioral and psychosocial assessment instruments that can

Table 2. A selected sample of general classes and common instruments for psychosocial assessment of adjustment and coping in chronic pain

Measures of Behavioral, Cognitive/Attitudinal, and Emotional Coping Strategies

- *The Cognitive Coping Strategies Inventory*[34] assesses degree of engagement in adaptive and maladaptive cognitive coping strategies.
- *The Coping Strategies Questionnaire*[35] rates frequency of engagement in 48 different behavioral and cognitive pain and physical symptom response coping strategies.
- *Vanderbilt Pain Management Inventory*[36] examines chronic pain coping strategies (eg, active, passive) and offers useful information for treatment planning and recommendations.

Measures of general health behavior

- *The Illness Behavior Questionnaire (IBQ)*,[37,38] a general health behavior inventory that provides useful information about attitudes, perceived reactions of others and psychosocial variables. Seven delineated factors include (1) general hypochondriasis, (2) disease conviction, (3) psychological vs somatic focusing, (4) affective disturbance, (5) affective inhibition, (6) denial, and (7) irritability. It can help identify reliance on illness behavior as a coping style for need procurement.
- *The Millon Behavioral Health Inventory (MBHI)*,[39] one of the most frequently used health inventories in the United States. It provides information across 4 broad categories: (1) basic coping styles, (2) psychogenic attitudes, (3) specific disease syndromes, and (4) prognostic indices. It has good psychometric properties, a large normative database of representative medical patients, and specific disease scales developed for specific patient groups. It is the precursor of the Millon Behavioral Medicine Diagnostic test (MBMD). The MBMD assists with identification of significant psychiatric problems, making specific recommendations, pinpointing personal and social assets to facilitate adjustment, identifying medical regimen compliance problems, and structuring posttreatment plans and self-care responsibilities in the patient's social network.
- *The Sickness Impact Profile (SIP)*,[40] a behaviorally based measure of health status designed to assess both psychosocial and physical dysfunction. It is characterized by sound psychometric properties and wide use with chronic pain patient's provision of relevant information regarding degree of functional limitation in daily activities.

Specific pain domain inventories

- *The Multiaxial Pain Inventory (MPI)*,[41] a biopsychosocial measure that assesses relevant psychosocial, cognitive, and behavioral aspects of pain responses. It includes specific norms for different statistically derived chronic pain subtypes: interpersonally distressed with inadequate social support; globally dysfunctional coping; and adaptive coping. The psychometrically derived multiaxial classification provides an objective method of evaluating chronic pain patients through integrating useful psychological information with data from multiple other sources, and offers benefit for matching patients to types of pain management interventions. An inexpensive software scoring program is available.[35]
- *The Hendler Chronic Pain Screening Test*[42] estimates contribution of physical versus psychological variables to pain behavior expressions. It employs a composite predictor approach where increasingly higher rating scores reflect increasing psychological influence or motivation on pain behavior. Higher scores suggest recommendations for conservative treatments with multimodality treatment programs. Very high scores typically require psychological or psychiatric referral and intervention.
- *Kinesiophobia and Cogniphobia scales*,[43,44] quick screening measures of unreasonable or irrational fear of movement or headache or painful reinjury upon physical or cognitive effort or exertion, respectively. These scales are designed to assess anxiety-based avoidant behavior with regard to physical or cognitive activity. These instruments offer information about need for combination therapies that include such anxiety reduction procedures as graduated exposure, cognitive reinterpretation, and systematic desensitization.

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Table 2. (Continued)

- *Headache Disability Rating*,[45] a scale procedure of Packard and Ham that estimates impairment from headache from rated frequency, severity, and duration of attacks and how activities impact on functional skills and activities of daily living. Its noteworthy feature is inclusion of a modifier variable for rating motivation (ie, treatment motivation, exaggeration/overconcern, and legal interest) and is used to adjust the total impairment rating.
- *Pain Assessment Battery (PAB), Research Edition*,[46] collection of 4 measures instruments that provide information regarding patient stress and pain reports with qualitative pain analysis and pain-coping strategies, including nonorganic pain complaints. Format allows serial computerized administrations and tracking.

Psychological screening measures: mood, anger, and anxiety

- *The Beck Anxiety Inventory (BAI)*,[47] measures severity of anxiety symptoms. Designed to assess anxiety symptoms independent of depression symptoms, it assesses physiological and cognitive components of anxiety in 21 items describing subjective, somatic, or panic-related symptoms. It shows good differentiation between anxious and nonanxious groups in a variety of clinical settings.
- *The Beck Depression Inventory – 2 (BDI-2)*,[48] common self-report measure of depressive symptomatology. It possesses some utility to differentiate chronic pain patients with and without major depression (optimal cutoff score = 21)[49] and has well-documented predictive validity.
- *The Perceived Stress Scale (PSS)*[50] is a widely used instrument for measuring the degree to which situations in one's life are appraised as stressful. Items measure how unpredictable, uncontrollable, and overloaded respondents find their lives and directly query current levels of experienced stress. Higher PSS scores have been associated with greater vulnerability to physical and psychological symptoms following stressful life events.
- *The State-Trait Anger Expression Inventory – 2 (STAXI-2)*,[51] a reliable, well-normed measure of the experience, expression, and control of both current state and trait anger. Anger Expression and Anger Control scales assess 4 relatively independent anger-related traits: (*a*) expression of anger outward, (*b*) holding anger in, (*c*) controlling outward expression, and (*d*) controlling internal angry feelings. It provides information regarding how experience, expression, and control of anger may contribute to psychophysiologic arousal and symptoms and increase risk for developing somatic symptoms and medical problems. It offers implied suggestions for directing appropriate remediations.
- *The Zung Self-rating Depression Scale (SDS)*,[52] a well-suited instrument for medical settings with several advantages over other measures: (1) shorter, simpler to administer and score, (2) requires a lower reading level, (3) fits well with medical and injury situations, (4) can be easily administered in an interview format, and (5) has subscales with some potential utility. Self ratings items on a 4-point scale are scored in the direction of increased depressive symptomatology.

Comprehensive measures of personality assessment

- *The Minnesota Multiphasic Personality Inventory*[53] *(MMPI)* and MMPI-2,[54] the most widely used psychological assessment instrument in the United States. A 567-item (true-false) objective (ie, 10 clinical and 3 [7 in revised version] validity scales derived through empirical discrimination) measure of personality function and emotional status. It has over 50 years of actuarial data collection and analysis supporting its predictive abilities. It is a very sensitive measure of psychological states, traits and styles (eg, excessive anxiety, tension, hostility, somatization tendencies, sociopathy), as well as other traits (eg, substance abuse, deviant thinking and experience, social withdrawal, problematic anger, and suicidal, homicidal, or other violent tendencies). Configural interpretation of the relative scale elevations allow tentative hypotheses regarding personality and coping style and relative degree of particular types of psychological disturbance. Although frequently misused and misinterpreted in the chronic pain population,[55] it remains one of the most useful adjuncts to personality assessment and treatment

(*continues*)

Table 2. (Continued)

planning. Efforts to distinguish organic versus psychological causes for chronic pain using this technique represent mostly failed applications,[37,56] while cookbook interpretations based on psychiatric patient normative data remain generally problematic. However, other significant information regarding emotional distress and coping styles can be derived.

- *The Personality Assessment Inventory (PAI)*,[57] a good measure of general psychopathology that can help with identification of a wide variety of risk factors that could adversely affect adjustment. It has good psychometric properties and contains 340 items, with 22 scales, including 4 validity scales. As with most other general psychological assessment measures, it has no norms for chronic pain and tends to overpathologize this group.
- *Millon Clinical Multiaxial Inventory, 3rd edition (MCMI-III)*,[58] includes scales assessing DSM-IV based psychiatric disorders, including affective, personality and psychotic disorders, and somatization and others. Useful for the differential diagnosis of personality disorders and psychological vulnerabilities for adaptation to pain. Like other psychiatric measures, it has no chronic pain group norms and may be prone to overpathologizing patients.

Qualitative and physical measures of response bias[59,60]

Waddell's nonorganic signs
>3 of 6 suggests that psychological factors appear to be influencing patient's responses and behavior

Mankopf's maneuver discrepancy
Heart rate not increasing commensurate with nociceptive stimulation during exam (controversy whether this always occurs)

Toe test for simulated low back pain
Flexion of hip and knee with movement only of toes should not produce an increase in low back pain

Magnuson's test
Inconsistencies on instruction to point to pain area several times over period of exam suggest increased potential for nonorganicity

Delayed response sign
Pain reaction temporally delayed relative to application of perceived nociceptive stimulus

Pinch test for low back pain
Pinching lumbar fat pad should not reproduce pain due to axial structure involvement; positive test suggests possible functional overlay

Exaggerated self reported complaints
High frequency, severity with higher frequency, and severity versus collaborative report

Poor response to typically helpful pain interventions
Failure to show any pain relief to at least 1 of the following: biofeedback, hypnosis, mild analgesics, psychotherapy, relaxation exercises, heat and ice, mild exercise Failure to show any pain relief in response to transcutaneous electrical nerve stimulation (TENS)

Exaggerated incapacity[61]
Failure to comply with reasonable treatment; report of severe pain with no associated psychological effects; marked inconsistencies in effects of pain on general activities; poor work record and history of compensation claims; previous litigation

assist in the identification of coping liabilities are included in Table 2. Notably, these measures are similar whether pain is acute or more chronic, although they become more important as pain persists. When administered to persons with acute pain, they can assist with planning for treatment interventions designed to prevent chronicity and

maximize adaptation and resolution of pain. See Martelli and Zasler[56] for a more detailed review.

Assessment of chronic pain

With the exception of ensuring against the too frequent occurrence that identifiable pathophysiology has been previously undiagnosed, assessment in chronic pain moves from focusing on identifying pain triggers and pathways to examining the individual's reactions to enduring or coping with pain. A comprehensive, biopsychosocial assessment becomes especially critical as pain persists beyond the early acute stage. The most useful assessments usually include measures of cognitive interpretations and beliefs about pain and pain-related disability; screening measures of stress, psychological adjustment, and distress; coping style, strategies, and behavioral avoidance responses; and quality of life.[62] Additionally, physical, emotional, and cognitive coping responses are examined, including factors affecting perceived personal control of pain and pain-related disruption of activities, roles, and usual and preferred means of deriving reinforcement and self-esteem. Such inquiry assists with identifying personal coping strengths and vulnerabilities that help with formulating individually tailored treatment interventions and recommendations aimed at improving adaptation.

Although psychological symptoms are fairly common in patients with chronic pain, emotional distress is usually reactive. Factors such as previous psychiatric history, drug use, lifestyle variables, etc., should be considered, but a comprehensive, traditional psychological is usually only indicated when screening suggests significant psychopathology, treatment response is poor, and/or when significant inconsistencies in pain responses and behavior are noted.

Pain is ultimately a subjective complaint that is difficult to verify or refute. Diagnosis and treatment is the presumed reason for assessment. Empirical reviews estimate that the incidence of frank malingering is small.[63] Notwithstanding the fact that there are no reliable methodologies for detecting malingering versus exaggeration in chronic pain patients,[33,63] pain evaluation referrals frequently involve contexts where significant functional or financial incentives for emitting symptoms do exist. Hence, response bias is an important variable to attempt to assess, and more detailed review of methodologies and considerations on this topic is advised.[33]

Instruments that are useful in the assessment of persons with chronic pain generally extend and expand upon information gained in patient description and interview report. A detailed review of the many available instruments, and their use in performing competent assessment in persons with chronic pain, is beyond the scope of this article. These instruments, as a class, have psychometric limitations, and generally demonstrate underdevelopment of behavioral, psychophysiologic, and nonquestionnaire self-report measures.[64] Nonetheless, a brief survey of general classes and samples of the more useful pain assessment instruments, based on previous work,[21,56] is included in Table 2. For additional Internet-accessible reviews and resources, see Martelli[65] or Bruns.[66]

Notably, several useful assessment instruments have arisen from biopsychosocial conceptualizations of chronic pain assessment and treatment. One of the most widely used instruments is the Multiaxial Pain Inventory (MPI).[41] The MPI assesses relevant dimensions of the chronic pain experience, has specific norms for different chronic pain subtypes,[67] and offers an inexpensive MPI scoring program to empirically classify patient's responses into prototypic patient response subtypes that help match patients to types of pain management interventions and identify those likely to need more intensive services.

Other instruments derived from more general stress and coping models of behavior have been adapted to more specifically assess

chronic pain related cognitive coping strategies.[68] These instruments assess adaptive and maladaptive beliefs and pain-related coping strategy use (eg, feelings of helplessness and external locus of control regarding pain, use of catastrophization, avoidance, self-blame) and have been shown to have utility for predicting response and adaptation to pain and for directing appropriate treatment interventions (eg, relaxation procedures, disputation of catastrophic beliefs, adaptive cognitive reinterpretation to emphasize internal control, goal setting, and activity quotas). Other measures have been designed to assess maladaptive avoidance behaviors. For example, the Kinesiophobia scale[43] measures maladaptive pain-phobic behavior and indicates need for combination therapies that should emphasize reeducation, countering maladaptive phobic responses, and promoting adaptive attitudes and treatment participation/cooperation (eg, reeducation, graduated exposure, and cognitive reinterpretation).

Behavioral observation is a very important assessment tool that should not be overlooked. Observations should focus on vocalizations (eg, groaning, grunting, sighing), verbalizations, facial expressions, muscle tension and rigidity, ability to be reassured, guarding of body parts, demeanor, appearance, and behavior in different activities. Observations should be made regarding factors that seem to reduce or increase observed and reported distress, as well as consistencies versus inconsistencies between and within reports and situations (eg, does a 10/10 pain rating look like it when observed, or when distracted?), whether there is seemingly overly impaired performance versus reasonable comparisons, or signs of exaggeration or feigning, or of pain suppression or minimization or amplification. Although adequate reliability and validity documentation is lacking for behavioral observations, these nonetheless add important information that must be integrated with all other available data in a biopsychosocial assessment.

MANAGEMENT OF PAIN

The focus, methods, and goals of pain management parallel those for pain assessment. Management strategies follow the same continuum, with a transition from more biomedically based treatments early postinjury to more psychosocially based interventions as pain becomes more chronic. The primary goal in acute pain management is analgesia and resolving the causes of pain by promoting natural healing and/or correcting underlying pathophysiologic processes, while modulating associated physical and psychological symptoms. The important secondary goal is to prevent chronicity and reduce functional disability. This goal necessarily includes the minimization of physical, cognitive, and emotional distress reactions to pain in order to prevent development of sensitization effects in the peripheral and central nervous system.

As pain becomes chronic, it becomes more intractable, and associated goals address reducing psychoemotional distress and functional disability, the factors that maintain and perpetuate them, and impoving adaptation and coping with pain. With chronic pain management, combination treatments and interdisciplinary team interventions are usually necessary to optimize success. The primary goal of treatment becomes the reduction of emotional, behavioral, and cognitive sequelae of chronic pain and associated sensitization effect.

On the basis of considerable evidence and growing consensus that most forms of chronic pain involve central and/or peripheral sensitization,[5–10] the authors have proposed a preliminary classification model for conceptualizing chronic pain management interventions. This model, summarized in Table 3, enables classifying currently available and potentially useful chronic pain treatment approaches according to specific area and manner of desensitization targeted. This model offers an intuitively appealing classification system for conceptually organizing the wide variety of available treatment

Table 3. A desensitization model for chronic pain treatment interventions

Desensitizing peripheral CNS procedures
Electromyographic (EMG) and temperature biofeedback; various relaxation and imagery procedures; TENS
Desensitizing CNS medications
Anti-epileptic drugs, tizanidine HCL, Amytal, neuroimmunomodulators, SSRIs, etc
Desensitizing behavioral activity procedures
Operant behavioral activity programs; graduated exposure/graduated activity programs; relaxation, imagery, refocusing; cognitive behavioral reinterpretive strategies
Desensitizing psychotherapeutic procedures
Emotional desensitization of catastrophic reaction to injury and pain and other fears and trauma; splinting of emotional reactions and calming of catastrophic reactions and hypervigilance to pain; specific formal pain and fear desensitization procedures; pain exposure/desensitization procedures; cognitive behavioral reinterpretive strategies
Desensitizing neurophysiologic procedures
Cranioelectrotherapy stimulation (CES). Consider electroencephalographic (EEG) biofeedback or other potentially helpful adjunctive relaxation procedures such as sound and light (AudioVisualStimulation [AVS]) and transcranial magnetic stimulation, and brain electrical stimulation.

interventions and in planning combination treatments.

The goals of both acute and chronic pain are to reduce pain levels and restore functioning in areas negatively affected by pain. There should be elucidation of the patient's expectations for treatment and identification of anticipated treatment interventions and realistic endpoints, which might include increased functioning and adaptation to the pain condition. The simplest and least invasive pain management approach should be used whenever possible, and the choice of pharmacologic agents should aim to minimize adverse effects and inconvenience to the patient, both of which will optimize compliance.

Importantly, multicomponent treatment packages are the preferred treatment choice in chronic pain, especially when comorbid with brain injury.[4,12,21,69] Others[70] recommend the development of pain management facilities specifically designed for persons with brain injuries. The emotional disturbances associated with pain are also frequently comorbid with TBI, highlighting the importance of a biopsychosocial perspective. Such a perspective allows for a holistic conceptualization of the patient, incorporating multimethod, multimodal assessments that facilitate individualized treatment planning. Treatment goals include not only the reduction/relief from pain, but also increased self-control, increased adaptation to life changes secondary to pain and brain injury, and improved functioning and quality of life.

Pharmacologic management

A comprehensive review of the options for pharmacologic management of pain is beyond the scope of this article and interested readers are referred to Zasler et al.[21] The choice of medication typically depends on the severity of pain, with aspirin, mixed narcotic analgesics with aspirin or acetaminophen, and anticonvulsants as examples of medications used for mild, moderate, and severe pain, respectively. There are a variety of medications for use of pain at different severity levels, each with different side effect profiles and requirements for monitoring of adverse side effects (eg, blood counts for some anticonvulsants). Medications for neuropathic pain should generally be initiated at a low dosage and titrated

up slowly on the basis of pain relief and patient response. Additional neuropathic pain treatments are Capsaicin, which is used topically, and topical agents such as "speed gels."

There are a number of medications that do not serve as firstline or primary pain interventions, but that work as adjuvant agents in pharmocologic management of pain, eg, corticosteroids and tizanidine (Zanaflex). In addition to providing assistance with pain relief, these agents are beneficial to combat common symptoms associated with pain or other medications being used (eg, nausea, dysphoric mood, reduced appetite, reduced sense of well being). Mailis et al[71] published an excellent review of the use of sodium amytal infusion in the assessment and treatment of chronic pain (and functional disorders). Stimulants such as methylphenidate are used with opioid analgesics as an adjuvant analgesic and to help manage opioid-induced sedation and cognitive impairment. Optimizing compliance and minimizing side effects should be the physician's goal for medication choice. Ideally, treatment is based on the individualized needs of patients (eg, choosing medications requiring less frequent dosing in cognitively impaired patients) and includes a discussion about goals of treatment, concerns about side effects (eg, sexual side effects, constipation), and fears of addiction and dependence. When appropriate, limiting polypharmacy is ideal, and as part of this effort it is important to obtain a complete listing of over-the-counter medication taken and determine whether patients are taking their medications as prescribed. While "opiophobia" may prevent some patients, particularly those with substance abuse histories, from receiving opioid medications, careful screening for usage trends and/or abuse as well as standard implementation of a "narcotics agreement" can help assuage concerns of treating physicians and facilitate the appropriate use of such medications. Although most often indicated for severe acute pain, opiods may also be used for severe chronic pain when other treatment methods have failed.

In addition to medication interventions, a variety of psychological (eg, biofeedback), behavioral (eg, adaptive equipment), physical (eg, physiotherapy, exercise, chiropractic, massage, etc), or other medical interventions for chronic pain exist. For reviews of such interventions, readers are referred to the works of McQuay and Moore,[72] the College of Physicians and Surgeons of Ontario report,[73] and Martelli et al.[4] These treatment modalities, in addition to focusing on pain relief, address goals of improved functioning and quality of life and should be given adequate consideration as part of a comprehensive pain management program for individuals with TBI.

Physical modalities

A variety of physical modalities are used particularly for the treatment of pain complaints of musculoskeletal origin. Among these are manual manipulation (eg, joint manipulation, myofascial release techniques, strain/counterstrain, and massage), traction, and injection techniques (eg, intra-articular, periarticular, peritendinous, ligamentous/fibrous tissue, axial, and trigger point). An often overlooked, underappreciated, and much less frequently recommended form of treatment in persons with post-TBI pain complaints is exercise. In addition to affecting pain on both a peripheral and central basis, exercise can play a role in reducing additional factors that can have mediating roles on pain experiences such as weight gain, anxiety, and a general sense of well-being. In addition, exercise has been shown to be an effective treatment for depression,[74] a commonly comorbid condition of both chronic pain and brain injury.

Other physical agents used to modulate pain range from topically applied interventions such as superficial heat and cold and hydrotherapy to devices designed to work on deep muscle tissue (eg, ultrasound, phonophoresis, and shortwave and microwave diathermy). Electrical stimulation devices such as transcutaneous electrical stimulation (TENS) are also commonly employed as adjuvants for pain control. Another,

less well-known electrical stimulation device for pain management is cranioelectrotherapy stimulation (CES). This device is designed to act on the central nervous system by using ear clips that transmit a microcurrent of approximately 15-Hz cortical rhythm. While originally used to treat insomnia, anxiety, and depression, conditions commonly comorbid with both pain and brain injury, a number of controlled studies support that CES is a safe and useful treatment for pain, especially chronic pain.[75–77]

Psychological–behavioral management

There are relatively few outcome studies addressing the treatment of pain within the context of brain injury. Those that have been conducted have found general similarities in clinical presentation, pathophysiology, and treatment responses with other chronic pain conditions, with somewhat greater similarities for traumatic versus nontraumatic chronic pain, and some evidence suggesting greater treatment resistance for the latter type.[78] Especially in cases of posttraumatic pain, the severity and frequency of pain attacks and chronic pain-related sequelae such as coping abilities, depression, and anxiety can be of great concern to both the patient and treatment provider, and can be significantly improved by combined psychological treatment protocols.[35,79–82] Supportive counseling that begins early after trauma and is continuous results in better patient response,[35] and combination treatments appear to increase likelihood of benefit. Multidisciplinary and interdisciplinary treatment teams offering such treatments may be the treatment of choice for more challenging pain conditions (see Branca and Lake in this issue).

Martelli et al[4] reviewed various behaviorally based chronic pain treatment interventions for which efficacy data are available. A summary of the more useful behavioral treatments for chronic pain are included in Table 4.

It should be noted that patients seeing a psychologist for pain management are often referred by other treatment providers. Although the referring provider has hopefully explained the rationale for the referral and potential benefits, it is often helpful to begin the assessment process by addressing any patient concerns (eg, about being believed that their pain is real). Taking this time can greatly assist in developing rapport, establishing a therapeutic relationship, and engendering accurately informed and hopeful expectancies. Psychological management of persons with pain and a concomitant brain injury begins as any other assessment with examination of all variables relevant for treatment. In addition to areas such as personality and emotional status, coping style and strategies, social support, and pain specific factors (eg, interference, severity, response patterns to pain), assessment must also include specific sequelae associated with brain injury. An integration of this information is necessary to ensure that the full constellation of residual sequelae and strengths are considered to optimize an individually designed treatment plan that anticipates and implements compensatory strategies for all potential obstacles to benefit from behavioral interventions. For example, deficits in memory, attention, or executive functioning might be addressed through task-analytic instruction and compensatory memory notebooks with provision of external reminders for completion of at-home assignments.

CONCLUSIONS

The assessment and treatment of persons with pain is a complicated and challenging process. Pain can have a more disabling effect across a wider range of functions than brain or many other types of injuries. In addition to the absence of clear and objective measures for assessment and monitoring of pain, pervasive misconceptions continue to dominate modern medical and psychological practice. Hence, familiarity with many important issues and the current knowledge base in the specialty field of pain management is required to effectively prevent and manage chronic

Table 4. Summary of useful behavioral treatments for chronic pain

Patient education: The most modifiable pain-contributing factor is the stress reaction component. The best treatment packages generally contain elements targeting numerous factors. Posture may be addressed by awareness training. Stress management can assist with reducing sympathetic arousal/discharge that exacerbates pain. Accurate information and expectancies help with this and also assist with coping with pain more adaptively. Education about expected symptoms and course after MTBI has been shown to reduce the anxiety and selective attention and misattribution that can unnecessarily prolong symptoms.[83]

Biofeedback: Abundant research supports the utility of EMG or thermal biofeedback for both headache pain and chronic musculoskeletal pain disorders more generally. The forehead, trapezii, frontal-posterior neck, and neck areas are frequent EMG feedback sites. Patterns of pathophysiologic neuromuscular activity that underlie pain complaint and functional limitations, which can be remediated through feeding back physiologic information to allow self-correction, include (*a*) stress-related hyperarousal in musculoskeletal or other physiologic systems; (*b*) postural dysfunction; (*c*) hypertonicity or hypotonicity induced by reflex systems activated by inflammation, active trigger points, and cumulative strain or recurrent trauma; (*d*) learned guarding or bracing to mitigate anticipated pain or injury; (*e*) Learned inhibition or avoidance of muscle activation/activity; (*f*) chronic compensation for joint hypermobility/hypomobility (eg, muscles taking over the role of damaged joint tissue); and (*g*) faulty motor schema and muscle imbalance reflecting development of 1 or more of the preceding syndromes, and resulting in the lack of coordination and stability between typically coordinated muscle groups. In addition, pain associated symptoms such as sleep can potentially be helped. Finally, data is emerging which indicates that EEG biofeedback, and associated EEG-driven stimulation offers efficacy in treatment of some persistent pain and persistent postconcussive symptoms.[84]

Relaxation training: Progressive muscle relaxation (PMR) is the most studied relaxation procedure.[85] PMR involves the systematic tensing and relaxing of various muscle groups to elicit a deepening relaxation response, usually with combination of muscle groups and addition of diaphragmatic breathing to shorten the protocol. Meta-analytic reviews generally conclude that relaxation training and biofeedback training are equally effective. Relaxation training presumably serves to (1) reduce proprioceptive input ot the hypothalamus, thereby decreasing sympathetic nervous system activity, and (2) directly reduce muscle tension or preheadache vasoconstriction,[86] and can also facilitate improvement in pain-associated symptoms such as sleep disturbance, anxiety, and fatigue.

Operant treatment: Treatment based on the operant model[87] requires altering environmental contingencies to eliminate pain behaviors (eg, verbal complaints, inactivity, avoidance) and reward "well" behaviors (eg, incrementally increased exercise, activity level).

Cognitive-behavioral treatments: Cognitive approaches typically involve instruction in identification and refutation of maladaptive beliefs concerning pain. Specific cognitive strategies and skills are taught to replace inappropriate negative expectations and beliefs that maintain physiologic arousal and complicate symptom resolution.[88] Mittenberg et al[83] demonstrated successful treatment of postconcussive syndrome that included headache with a treatment package consisting of education about how expectations and misattributions can perpetuate symptoms, along with cognitive restructuring to shape more adaptive interpretations and expectancies.

Social and assertiveness skills training: Skills training may help some patients with more effective communication of needs. Increased need fulfillment decreases distressful emotions, which reduces physiological arousal that contributes to pain experience[69]

(*continues*)

Table 4. (Continued)

Imagery and hypnosis: Using some combination of autohypnosis, suggestions of relaxation, and visual imagery, patients are generally instructed to visualize the pain (ie, give it form) and focus on altering the image to reduce the pain. Sleep and associated symptoms can also be potentially helped. Imagery-based treatment is most effective following establishment of a good therapeutic alliance to facilitate compliance.[89–91]

Habit reversal: These treatment "packages" teach pain patients to detect, interrupt, and reverse maladaptive habits (eg, maladaptive head/jaw posture, jaw tension, negative cognitions). Specific skills are taught to both reverse poor functional habits and stressful thoughts as well as feelings that precipitate/perpetuate them.[92]

pain problems. This article has outlined an informed conceptual model intended to help in this regard.

Finally, the standards for specialty knowledge and training for treating pain parallel those for treating brain injury.[93] Similar expectancies apply to both, and neither should be compromised. At the core of all bioethical principles is the avoidance of harm. Virtually every ethics code issued by every healthcare profession and specialty enjoins its members to avoid doing harm by not practicing outside the limitations of their competence. Consistent with recent revisions and current ethical principles in medicine and psychology,[94–96] available options for brain injury specialists without specialized training and experience in pain management include referral to a professional with specialty competence; consultation with such specialists when referrals cannot be made; and acquisition of knowledge, supervision and training as indicated. When professions provide pain management services in the absence of specialty competence and without availing themselves of these options, they have ethical obligations to represent these limitations through tentativeness in opinions and conclusions and with complete transparency about the potential effects of these limitations.

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Posttraumatic Headache: Neuropsychological and Psychological Effects and Treatment Implications

[Posttraumatic Headache]

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Abstract

Posttraumatic headache (PTHA) is a frequent occurrence following trauma to the head, brain, and/or neck. Estimates of persistence for 6 months are as high as 44%. Review of available studies examining the effect of headache on neuropsychological test findings reveals that chronic headache pain, and chronic pain generally, exerts a significant and negative effect that poses a challenge to differential diagnostic efforts in the evaluation of mild brain injury. Given that PTHA is the most common postconcussive symptom and most frequent type of posttraumatic pain associated with mild traumatic brain injury (TBI), it follows that resolution of the postconcussion syndrome, and successful posttraumatic adaptation, may frequently rely on success in coping with PTHA symptomatology. Viewing PTHA from a biopsychosocial perspective, a general outline is offered for improving both assessment and treatment of PTHA. In addition, the most promising psychology-based treatment interventions are reviewed.

POSTTRAUMATIC headache (PTHA) is a frequent occurrence following trauma to the head, brain or neck; such trauma can be accompanied by a constellation of cognitive, emotional, and physical symptoms, as well as responses to injury in general and to headache more specifically. Notably, the terms *posttraumatic syndrome and postconcussion syndrome* [1] have both been used as

descriptions of the array of symptoms following motor vehicle accidents and have led to considerable confusion (see the article by Zasler regarding difficulties in nomenclature, elsewhere in this issue). What is not confusing, however, is the consistency of the symptoms following these injuries and the predominance of headache. Notably, the incidence of PTHA following head, brain, and neck trauma is estimated to be as high as 90%. [2] The incidence of chronic PTHA (CPTHA), or PTHA lasting more than 6 months, has been found to be as high as 44%, [3] and the incidence at 4 years has been estimated to be approximately 20%. [2]

PTHA AND IMPLICATIONS FOR NEUROPSYCHOLOGICAL ASSESSMENT

The assessment of neuropsychological functioning has become a common practice in the comprehensive evaluation of patients sustaining traumatic brain injury (TBI). Frequently, diagnosis of mild TBI is based on neuropsychological test findings as the primary and most sensitive measure for objective documentation of such injuries. Judgments based on diagnosis can be far reaching in terms of financial, vocational, treatment, and disability implications. Given the frequent presence of PTHA, however, the validity and utility of neuropsychological test based inferences necessarily depend on assurances that headache pain does not affect neuropsychological test results. [4]

Notably, several researchers who examined the prevalence of self-reported symptoms in persons with PTHA concluded that reported cognitive difficulties, when assessed, were quite prevalent (see Packard and Ham [5] for a review). Subsequently, several investigations of cognitive performance on more objective tests of neuropsychological performance have been conducted for persons with PTHA. A review of investigations identified through literature searches of several major medical and psychological databases is presented in Table 1. [6–14]

Authors	Summary of findings
Gfeller et al[6]	Evaluated 42 patients with persistent (≥ 3 mo) posttraumatic headache using a strategic battery of neuropsychological measures and found greater impairment for high versus low postconcussive symptom-reporting groups on 6/13 measures employed, including sensitive measures of verbal associative fluency, memory functioning, mental processing speed, and cognitive flexibility.
Gimse et al[7]	Evaluated 23 whiplash patients with headaches and positive findings for postural control disturbance against a matched control group and found significantly poorer scores on tests of learning and memory and tests of sustained divided attention and concentration. Findings were supported after efforts to rule out influences from pain, depression, medication, and premorbid health problems.
Radanov et al[8]	Compared 54 "common whiplash" patients with 28 patients with cervical spine syndrome caused by rheumatism and, despite the relative absence of self-reported symptom complaints in rheumatism versus whiplash patients, found impairment on measures of divided attention for both groups. Results suggested that headache due to cervical pathology is associated with impaired attentional functioning.
Radanov et al[9]	Compared 51 soft tissue injury patients with "cervicogenic" (complaint of headache, fatigue, dizziness, poor concentration, disturbed accommodation, and impaired adaptation to light intensity) versus "lower cervical spine" (cervical and cervicobrachial pain) syndromes following soft tissue injuries. They found similar reductions in speed of information processing for both syndromes, but significantly poorer divided attention scores for the cervicogenic patients were found, without relation to length posttrauma. Reduced working memory processing was interpreted as responsible for more global cognitive problems, as well as secondary neurotic reactions. Notably, the possibility of undiagnosed mild TBI was not excluded.
Tsushima & Tsushima[10]	Found an insignificant relationship (except for arithmetic) between headache and neuropsychological functioning when they divided 184 head injury patients into groups according to headache frequency; however, the Luria Nebraska Neuropsychological Battery (LNNB) was employed versus more sensitive and validated attention and information-processing measures.
Tsushima & Newbill[11]	Rated headache severity at time of testing for head injury patients and found no significant headache effects on neuropsychological testing, although they used the LNNB versus more sensitive and validated measures of attention and information processing.
Leijdekkers et al[12]	Found no differences between 37 female migraine patients and 34 nonheadache females on a fairly extensive battery of neuropsychological tests.

continues

Table 1. Summary of investigations of effect of headache pain on neuropsychological test results

Authors	Summary of findings
DiStefano & Radanov[13]	Compared groups of patients with cervical vertebrae dislocations and persistent symptomatology (n = 36) versus those subjectively recovered (n = 81) on measures of attentional functioning. They found no differences between groups at 1 wk or 6 mo on measures of attentional functioning, although the group that demonstrated persistent symptomatology at 6 mo demonstrated impaired divided attention at 1 week, along with increased headache, greater medication use, and higher age.
DiStefano & Radanov[14]	Followed 117 common whiplash patients with head and neck pain for 2 years and compared 21 symptomatic patients with pair-matched recovered patient controls on a well-designed battery of neuropsychological measures of attention and memory. Differential rates of recovery on measures of divided attention that could not be explained by medication or pain intensity were found. They concluded that selective aspects of attentional functioning in symptomatic patients may underlie cognitive complaints and cause adaptational problems in daily life.

Table 1. Continued.

This review supports the conclusion that when specific, sensitive neuropsychological measures are employed, headache pain is generally found to exert a significant and negative effect on neuropsychological test performance, at least for persons reporting persistent subjective complaints. Decrements in information processing speed and complex attention are most frequently observed, while reductions in cognitive flexibility and verbal associative fluency, as well as learning and memory, appear to represent secondary findings that may be mediated by decreases in information processing and complex attention.

Similarly, findings from a review of more general investigations of the effect of chronic pain on cognitive functioning and neuropsychological test performance are presented in Table 2. 15–21 This review supports the conclusion that pain and pain-related symptomatology typically produce impaired performance on selective neuropsychological testing.

Authors	Summary of findings
Schwartz et al[15]	Found no significant differences between chronic pain patients with or without experience of head/neck injury on three sensitive neuropsychological measures.
Kewman et al[16]	Found a negative association between severity of pain complaints in musculoskeletal pain patients and performance on the Neurobehavioral Cognitive Status Examination.
Eccleston[17]	Found that severe chronic pain patients showed greater performance deficits on complex demanding attention-interference task versus low-pain patients or controls.
Eccleston[18]	Found that severe chronic pain patients showed significantly impaired performance on a sustained and shifting attention task compared to low-pain patients or controls.
Grigsby et al[19]	Compared chronic pain patients with a group of patients with mild and moderate head injury on motor functioning and information processing and found similarly poor performances on most measures for both groups, with the chronic pain group actually performing worse on measures of processing speed.
Goldberg et al[20]	Posttraumatic temporomandibular disorder (TMD) patients were significantly slower on simple and complex reaction time tests than nontraumatic TMD patients, and more susceptible to both proactive and retroactive interferences on immediate and delayed memory tests. SPECT differences suggesting greater abnormality in the traumatic group were noted.
Jarvis & Kooken[21]	Offered case study data, with referenced support from most of the non-PTHA chronic pain studies referenced herein, demonstrating pain as a significant factor affecting neuropsychological test performance that can lead to misdiagnosis of brain injury.

Table 2. Summary of investigations of effect of general chronic pain on neuropsychological test results

When the findings from Table 1 and 2 are considered together, it should be noted that the pattern of neuropsychological impairments observed appear quite similar to those produced by persons sustaining mild brain injuries. The implications with regard to differential diagnosis in cases of neuropsychological evaluation for brain injury, especially for suspected mild brain injury, clearly require that pain be considered a factor in neuropsychological test findings. Further studies are clearly indicated to help delineate this relationship, including how such factors as pain severity, type of chronic pain, and pain location specifically affect specific

neuropsychological test results. Finally, consistent recent findings regarding regional cerebral blood flow abnormalities in persons with chronic pain [22–25] offer added support for these conclusions.

THE PHENOMENOLOGY OF PAIN

Pain is a complex multidimensional subjective experience mediated by emotion, attitude, and perception. With pain experience, variability is often the rule rather than the exception, and variabilities in pain responses and health outcomes appear to be the result of complex biopsychosocial interactions among genetic, developmental, cultural, environmental, and psychological variables. [26–28] Notably, in the chronic pain literature, two critical distinctions are made. Acute pain, or pain occurring shortly after injury

* reflects relatively discrete neuroanatomic pathways and underlying acute somatic injury
* communicates useful new information that initiates protective physiological mechanisms (against injury extension)
* has survival value for the organism's adaptation by signaling the need for corrective attention.

In contrast, chronic pain, or pain that persists long (usually defined as 6 months) after injury

* reflects ambiguous pathways between injury sites and the central nervous system
* communicates useless information that perpetuates physiological protective responses long after removal of possibility of injury extension and despite lack of underlying tissue damage
* poses a liability to postinjury adaptation

Importantly, chronic pain is typically associated with response patterns involving decreases in, and avoidance of, activity. Decreased activity, in response, can prevent normal restoration of function; perpetuate painful experience; and, in cyclic disability-producing fashion, reinforce avoidance, inactivity, and increased pain. Finally, the longer pain persists, the more recalcitrant it becomes and the more treatment goals move toward management of pain and coping versus cure. [28]

IMPLICATIONS FOR PSYCHOLOGICAL/BEHAVIORAL TREATMENT OF PTHA

According to Miller, [29] chronic pain often represents the “weak link” in the cycle of “postconcussion invalidism.” Given that PTHA is the most common postconcussive symptom, [6,30,31] and hence the most frequent type of posttraumatic pain associated with mild TBI, it follows that resolution of the postconcussion syndrome, and successful posttraumatic adaptation, may frequently rely on success in coping with PTHA symptomatology. Even in the case of persistent symptomatology, Devore [32] notes, based on a review of the literature, that recovery and management of pain syndromes are quite likely, even with significant psychological comorbidity, provided that appropriate psychological consultation is provided.

In the field of chronic pain rehabilitation, the introduction of biopsychosocial models represent alternative theoretical approaches to dualistic and reductionistic biomedical conceptualizations that explain disease and health primarily in terms of measurable biological variables. Such models have helped explain the variability in health care outcomes and significantly advanced clinical treatment and outcome through the development of interventions for challenging health care situations. [33] Notably, a derived stress and coping formulation of postinjury adaptation conceptualizes adaptation to injury as a series of stressful demands that require coping, whether the injury is mild or more severe. Coping will, of course, represent an interaction between existing coping resources and injuryrelated demands. Bolstering of coping resources presumably allows for improved coping with stressful demands. Improved adaptation and reduced interference from symptoms should be expected, perhaps with the qualification that more or less bolstering of coping resources will be required. That is, the question should not be whether the symptoms will improve, but rather, how much coping assistance will be needed.

PSYCHOLOGICAL TREATMENT OF PTHA

PTHA does not occur in a vacuum. Rather, it occurs in a biological system within specific psychological and social contexts. It reflects an interaction of organic and emotional factors and, while similar to natural headaches in clinical presentation of subtypes and biochemical mechanisms, is typically resistant to traditional headache treatment. [30,34,35] Medication management alone may lead to unwanted side effects (eg, adverse effects on sleep, mental alertness, sexual functioning, or work performance) and does not address coping skills. [36] Conversely, PTHA patients have been reported to exhibit minimal response to nondrug (ie, psychological) treatments alone. [35] Treatments that are holistic in nature, targeting not only the pain directly, but also the patient's reaction to pain within his or her daily life, typically fare better than treatments with a more narrow focus (eg, medication management or nondrug therapies alone). Currently, multicomponent treatment packages are the preferred treatment choice for PTHA. [29,36,37]

Individual patient variables: Treatment issues and implications

Clearly, psychological factors affect the decision to seek treatment, type of clinical presentation, coping responses, and treatment efficacy for headache. [38,39] For example, Ziegler and Paolo [39] statistically controlled for headache severity and found significant elevations on the Hypochondriasis, Depression, Hysteria, Psychasthenia, and Social Introversion scales of the Minnesota Multiphasic Personality Inventory (MMPI) for treatment-seeking headache (HA) patients versus non-treatment seeking HA controls. These findings suggest that persons with HA who seek treatment may be more psychologically distressed relative to

non–treatment seekers. Karlsborg and colleagues [40] reported that stressful life events were more associated with distress and poor outcome than the presence of clinical findings (eg, positive results on magnetic resonance imaging [MRI]) in patients with whiplash injury.

The development of symptoms indicative of posttraumatic stress disorder (PTSD), including recurring nightmares, phobic avoidance, hypervigilance, and generalized anxiety, provides a special issue for PTHA treatment and illustrates the importance of differential diagnosis and individualized treatment protocols. Notably, Hickling et al [41] found that 15 of 20 consecutive patients referred to a psychological practice for PTHA had PTSD. Furthermore, Schreiber and Galai-Gat, [42] noting the prevalence of PTSD among injured versus noninjured survivors of stressful events, presented a case study demonstrating that secondary stressors (eg, severe uncontrolled pain, a prolonged state of acute anxiety, uncertainty regarding the immediate future, loss of control, and inability to monitor contact with the environment) may play an important role in the formation of PTSD. They recommended prompt and adequate pain management in hospitalized survivors of traumatic injury. Clearly, accurate differential diagnosis of previously undiagnosed PTSD late after injury is also necessary.

Gfeller and colleagues [6] found greater cognitive and emotional symptoms in depressed PTHA patients relative to nondepressed PTHA patients. DiStefano and Radanov, [14] however, suggest that cognitive complaints and dailylife adaptational difficulties may be the result of deficits in attentional functioning following traumatic injuries associated with headache pain. Conclusions concerning poor outcome following a posttraumatic accident are complicated by the “chicken and the egg” debate—that is, are PTHA patients predisposed to poor coping with daily life stress due to posttraumatic pain, depression, or attentional deficits, or do preexisting deficits in coping skills account for greater difficulties with distress following a traumatic injury?

Of course, depression, PTSDs, anxiety conditions, and other psychiatric syndromes often precipitate or exacerbate headache pain. They may also represent reactions to pain and associated life disruption and can, in some cases, represent a primary psychological disturbance. Generally, these disorders have favorable psychological and functional prognosis given timely and appropriate assessment and treatment. Misdiagnosis of these conditions can serve to promulgate misperceptions and amplify functional disability and health care costs.

Kay [43] offers evidence that personality disorders complicate adaptation after mild brain injury, while Miller [29] suggests that personality disorders and styles may complicate the administration of typical pain protocols and necessitate more extensive psychotherapy. Characterologically maladaptive coping styles (eg, exaggerated complaints from histrionic patients, excessive reliance on health care providers by the dependent patients, or witholding of information by schizoid patients) undoubtedly represent complications to both evaluation and treatment.

Treatment of PTHA may be further complicated by the presence of multiple pain sites. For example, Duckro and colleagues [44] reported comorbid myofascial irritation in the upper back, neck, and facial muscles in a sample of patients with PTHA. Furthermore, measures of anger, depression, and perceived disability were positively related to muscle tenderness at these sites. These findings suggest that treatments targeting the musculature (eg, relaxation and electromyographic [EMG] biofeedback), as well as cognitive behavioral therapy (CBT) to reduce anger and depression, may be beneficial in the treatment of multiple pain and emotional distress symptoms in persons with PTHA.

Interestingly, Martelli et al [33] presented a vulnerability, stress, and coping model and assessment methodology that incorporates psychological and social factors with biological factors as a schema for increasing understanding of variability in health care outcomes after TBI. They presented preliminary evidence for the utility of identifying vulnerability factors with a composite TBI Vulnerability Rating Scale (TBI-VRS) by reliably discriminating persons with better and poorer adaptation following brain injury. They also presented a more generalized vulnerability to disability scale with a report of preliminary findings suggesting the utility of identifying persons at risk for poor adaptation in mixed chronic pain patients. The Vulnerability to Disability Rating Scale (VDRS) parallels the TBI-VRS by providing for a composite rating of variables generally associated with poor adaptation to physical impairment (Table 3).

Increased complaint duration	Complaint inconsistency/ vagueness	Previous treatment failure	Collateral injury/ impairment	Pre/Comorbid medical history	Medication reliance
0 = < 6 mo 1 = < 12 mo 2 = > 12 mo Especially with expectation of chronicity, poor understanding of symptoms	0 = Little 1 = Mixed 2 = Mostly inconsistent Multiple, vague, variable sites; anatomically inconsistent; sudden onset without accident or cause; not affected by weather performing no work or chores, or avoiding easy tasks but performing most hobbies, enjoyments; pain only occasional	0 = Insignificant 1 = Mixed 2 = Mostly or all failures Especially with complaint of treatments worsening pain or causing injury, and expectation that future treatments will fail	0 = Insignificant 1 = Mild/moderate 2 = Significant Especially if silent and involving adaptation reducing impairments	0 = Insignificant 1 = Mild to < moderate 2 = Significant Seizure disorder/ diabetes; hypertension; brain injury, stroke, or other neurologic insult or vulnerability (esp. if undiagnosed); preinjury medication reliance; older; etc.	0 = Little 1 = Moderate 2 = Significant > 4 times/week narcotic, hypnotic or benzodiazepine tranquilizer; perceived inability to cope without medication
Severity of current psychosocial stress	**Psychological coping liabilities**	**Victimization perception**	**Social vulnerability**	**Illness reinforcement**	
0 = Nonsignificant 1 = Mild/moderate 2 = Significant Sum of personal, social, financial, emotional, identity, activity stresses, life disruption, premorbid coping style disruption, etc, and including injury/impairment ¥ coping style incongruence; persistent premorbid psychosocial stress levels	0 = Few 1 = Mild/moderate 2 = Significant Premorbid, comorbid: depression; posttraumatic anxiety; somatization (& repressive) defenses; emotional immaturity/ inadequacy with poor coping skills; hypochondriacal traits (eg, postinjury MMPI-3 > 85; preinjury > 70); passive coping style; childhood trauma (esp. death of parent; child or sex abuse); anger/ resentment; posttraumatic adjustment problems; alcohol, substance use/ abuse; limited premorbid intellect, education, skills; preinjury psychiatric treatment; poor premorbid work history	0 = Little 1 = Mild/moderate 2 = Significant Externalized "blame" for accident, disability, etc; perceived mistreatment; anger, fear, resentment, distrust regarding accident, treatment, understanding (family, employer, physician, etc—esp. given characterologic tendencies regarding victimization, resentment, suspiciousness, distrust, etc)	0 = Little 1 = Mild/moderate 2 = Significant Lack of family support, resources, romantic support (esp. if recent conflict, divorce); lack of community support/resources/ involvement; lack of employer, coworker, insurance manager support, etc.	0 = Little 1 = Mild/moderate 2 = Significant Secondary gain: attention, support in a dependency prone person; avoidance of stressful or displeasing life or job responsibilities or demands (esp. with recent or imminent job/job duty changes or reorganization); financial compensation (esp. if litigation; or current income = preinjury/ preimpairment)	VULNERABILITY SCORE _____ Total Pts (Max: 22) Preliminary Interpretive Guidelines *Scores of 13 or Above Suggest High Vulnerability to Chronic Disability*

Table 3. Vulnerability to Disability Rating Scale

Nonorganic symptoms and response bias

Although the frequency of occurrence is estimated to be relatively low, [38] clinicians evaluating PTHA should be familiar with nonorganic syndromes that may present as head pain, including

* *factitious disorder*, or the intentional production or feigning of physical symptoms or exaggerated expression of physical conditions in order to adopt a sick role
* *somatoform disorders*, characterized by preoccupation with physical symptoms pain that exceeds possible organic pathology
* *hypochondriasis*, or preoccupation with head pain as part of a conviction that it is a part of a pernicious disease process
* *conversion disorder*, or the expression of frank psychiatric disorder through head pain as a result of some symbolic transformation

Response biases, such as symptom magnification and malingering, should also be considered. *Symptom magnification*, or exaggeration of impairment, can occur in relation to multiple factors and serve a wide range of psychological needs, including efforts to legitimize latent dependency needs; resolve preexisting life conflicts; retaliate against employer, spouse, or other; reduce anxiety; exert a “plea for help”; solicit acknowledgment of perceived difficulties; adopt a physical versus psychological explanation for problems; or adopt a specific diagnosis responsible for all life problems that reinforces passivity and avoidant coping in persons with premorbid histories of psychiatric and psychoemotional problems. *Malingering*, or deliberate symptom production, should always be assessed through identification of opportunities for secondary gain (eg, increased compensation or avoidance of undesirable responsibilities) and should always be assessed in cases of medicolegal presentation or suspicion of any incentive for symptom exaggeration, malingering, or withholding of true performance on physical or cognitive tests. Any of

several memory measures designed to assess atypical or worse-than-chance performance can be employed to assess response bias in cognitive performance, [33] while consistency of reported symptoms across situations or time, or with known anatomic patterns, can help assess bias in physical symptom complaints.

When patients assessed for PTHA demonstrate specific coping vulnerabilities, non-organic contributors should be considered. In cases of poor response to treatment, suspicious presentation, inconsistency, or claims of major disability associated with PTHA, these factors should be more closely scrutinized. Of course, the presence of a non-organic syndrome or response bias does not necessarily exclude the diagnosis of another organic syndrome. This certainly complicates the process of disentangling multiple clinical entities that sometimes coexist. Integration of contextual information, history, behavioral observation, interview data, collaborative data, and personality data with measures of effort or performance (or symptom exaggeration or malingering) and psychological or neuropsychological performance data provides the best information for estimating, for instance, the degree to which self-reported symptom complaints, physical or cognitive test results, or demonstrated performances are reliable and valid. [33]

Assessment phase

General format

The assessment phase is the starting point of any psychological treatment protocol. Detailed individual assessment is necessary to consider specific treatment issues (eg, personality variables, social support) and facilitate the patient–therapist relationship. A thorough behavioral assessment may include a detailed clinical interview and paper-and-pencil assessment instruments such as pain diaries and various standard pain and headache questionnaires. Psychophysiological assessment is an additional option, if feasible, and typically involves examination of muscle tension or EMG for different muscle groups in the head (forehead, masseter, temporal, occipital) and neck (trapezius, cervical paraspinal). A protocol may also include three specific phases (eg, baseline, stressor, recovery). Some evidence suggests that headache patients are more physiologically aroused at baseline and slower to recover following a stressor than painfree patients. [45]

The assessment phase concludes when the results of evaluation have produced a specific case conceptualization that identifies a specifically tailored treatment plan. Feedback to the patient utilizing assessment results provides a framework for the treatment intervention, defines goals and patient/therapist expectations and sequences, and provides the forum for presenting general information concerning PTHA and the rationale for treatment and enlisting participation.

Specific measures

Penzien and colleagues [38] argue, based on a review of the headache literature, that although levels of psychological symptoms frequently have exceeded those of normal control samples, clinical levels of psychopathology are observed only among subsets of the headache population. Furthermore, emotional distress is frequently reactive to chronic pain. They recommend, therefore, that the most useful assessment of the typical headache patient include measures of stress, cognitive and behavioral strategies for coping with stress, beliefs about headaches, factors affecting perceived personal control of headaches, and headache-related disability. Of course, factors such as previous psychiatric history, drug use, and lifestyle variables should be considered. When screening suggests significant psychopathology, when treatment response is poor, or when inconsistencies in pain responses and behavior are noted, then more comprehensive and traditional psychological evaluation is warranted.

Several useful assessment instruments have arisen from biopsychosocial conceptualizations of chronic pain assessment and treatment. One of the most promising instruments is the Multiaxial Pain Inventory (MPI). The MPI is a comprehensive instrument for assessing relevant dimensions of the chronic pain experience, including pain intensity and interference, emotional distress, cognitive and functional adaptation, and social support. [46] Specific norms have been determined for different chronic pain subtypes, including head-ache. Notably, use of an inexpensive MPI scoring program [47] allows empirically based classification of patient responses into prototypic patient response subtypes: dysfunctional, adaptive coper, and interpersonally distressed. These classifications offer benefit for matching patients to types of pain management interventions, especially in terms of identifying those likely to need more intensive behavioral medicine services.

Several other instruments born from more general stress and coping models of behavior are adapted to more specifically assess chronic pain–related cognitive coping strategies. [27,38,48] Some of these have been applied to headache. Such instruments assess beliefs and use of painrelated coping strategies that are adaptive and maladaptive (eg, feelings of helplessness and external locus of control regarding pain, use of catastrophization, avoidance, selfblame). Not only can they predict response and adaptation to pain, but they represent instruments that can be used to prescribe treatment interventions (eg, relaxation procedures, disputation of catastrophic beliefs, adaptive cognitive reinterpretation to emphasize internal control, goal setting and activity quotas) with demonstrated efficacy for reducing painrelated distress.

Another useful conceptualization has been offered by Todd, [49] who proposed the concept of kinesiophobia as an explanation for the development and maintenance of avoid-anceconditioned pain-related disability. Kinesiophobia is defined as the unreasonable or irrational fear of pain and painful reinjury upon physical movement. Given that lack of patient participation and cooperation are the major factors contributing to poor progress (or relapse) in chronic pain treatment, it follows that pain phobias, as unhealthy pain-maintaining habits, are a major contributor to painrelated disability. The Kscale is used to quickly screen for unreasonable fear of movement or reinjury. Once malingering factors are ruled out, combination therapy is provided with emphasis on reeducation, countering maladaptive phobic responses, and promoting adaptive attitudes and treatment participation/cooperation.

Similarly, Todd et al [50] extended the concept of kinesiophobia to headaches. They observed that several cases of poor effort on neuropsychological assessment early after head trauma, initially mistaken for response bias, were reflective of phobic responses to anticipated headache. Some of these responses have been observed to persist for 1 or more years and required formal anxiety reduction procedures to counter. The concept of cogniphobia was subsequently proposed as an unreasonable or irrational fear of headache pain or painful reinjury upon cognitive exertion. The C-Scale, adapted from the kinesiophobia instrument and designed to assess anxiety-based avoidant behavior with regard to cognitive exertion, is included in fig 1. Preliminary findings suggest both good psy-chometric properties and utility. Like kinesi-ophobia, cogniphobia is treatable and can be eliminated through combination therapies that include such anxiety reduction procedures as graduated exposure, cognitive rein-terpretation, and systematic desensitization.

C-Scale: Survey of Headache Impact Key: 1 = Strongly disagree, 2 = Disagree, 3 = Agree, 4 = Strongly Agree		Disagree		Agree	
		1	2	3	4
1	I'm afraid that I might make the cause of my head pain worse if I concentrate too much.				
2	If I were to try to overcome it, my head pain would increase.				
3	My head pain is telling me that I have something dangerously wrong.				
4*	My pain would probably be relieved if I practiced concentration exercises.				
5	People aren't taking my medical condition seriously enough.				
6	My accident/injury has put my head and brain at risk for the rest of my life.				
7	Headaches always mean I have an injury or have done something to make it worse.				
8*	Just because something aggravates my pain does not mean it's dangerous.				
9	I'm afraid that I might make my medical condition worse by concentrating too much or being too mentally active.				
10	Simply being careful not to concentrate too hard or too long is the safest thing I can do to prevent my pain from worsening.				
11	I wouldn't have this much pain if there weren't something potentially dangerous going on in my head.				
12*	Although my condition is painful, I would be better off if I were more mentally active.				
13	Pain lets me know when to stop concentrating so that I don't injure myself.				
14	It's really not safe for a person with a condition like mine to engage in too much thinking and concentrating.				
15	I can't do all the things normal people do because it's too easy for me to cause harm to my condition.				
16*	Even though something is causing me a lot of head pain, I don't think it's actually dangerous.				
17	No one should ever concentrate on difficult mental tasks when he/she is in pain.				
* = Reverse Scoring / Suggested cutoff = 37		Total: _____			

Fig 1. Cogniphobia scale.

Treatment phase: Components of psychologically-based treatment protocols

While there is an abundance of headache treatment outcome studies available, there are relatively few studies specifically examining the psychological treatment of PTHA as a distinct subgroup of headache in general. The literature suggests that PTHA and natural headaches may share common pathways, and clinical presentations are generally very similar, if not identical. [51] Consequently, standard psychological treatments for headache are presumed to share common mechanisms of action. While PTHA treatment outcome studies suggest that combined psychological treatments are generally efficacious, evidence suggests that PTHA is often more recalcitrant to standard psychological treatment compared with natural headaches. [35] However, the severity and frequency of pain attacks and chronic pain–related sequelae such as coping abilities, depression, and anxiety may be significantly improved by combined psychologi-cal treatment protocols. [29,34,36,37,51] Supportive counseling that begins early after trauma and is continuous results in better patient response. [52]

Patient education

Packard [53] directly asked, “What does the headache patient want?” and detailed the stated treatment priorities of headache patients. Education concerning the causes of headaches was listed as a top priority. Information can be individualized for the patient and ideally presented while providing feedback after the behavioral assessment phase. It is especially important for psychologists to emphasize to the patient that the patient's pain is real. Some patients, when told by physicians that medical tests are inconclusive or that their headache pain is due to stress, may interpret this information as “it's all in my head.” Anecdotally, many patients are

confused or angry when referred to a psychologist for pain treatment. Explaining the cycle of stress and pain and validating their pain may help to gain client trust and commitment. The following paragraphs provide sample information that is helpful to convey to the PTHA patient. Etiological models of headache pain that may be drawn or given as patient handouts are provided in Fig 2.

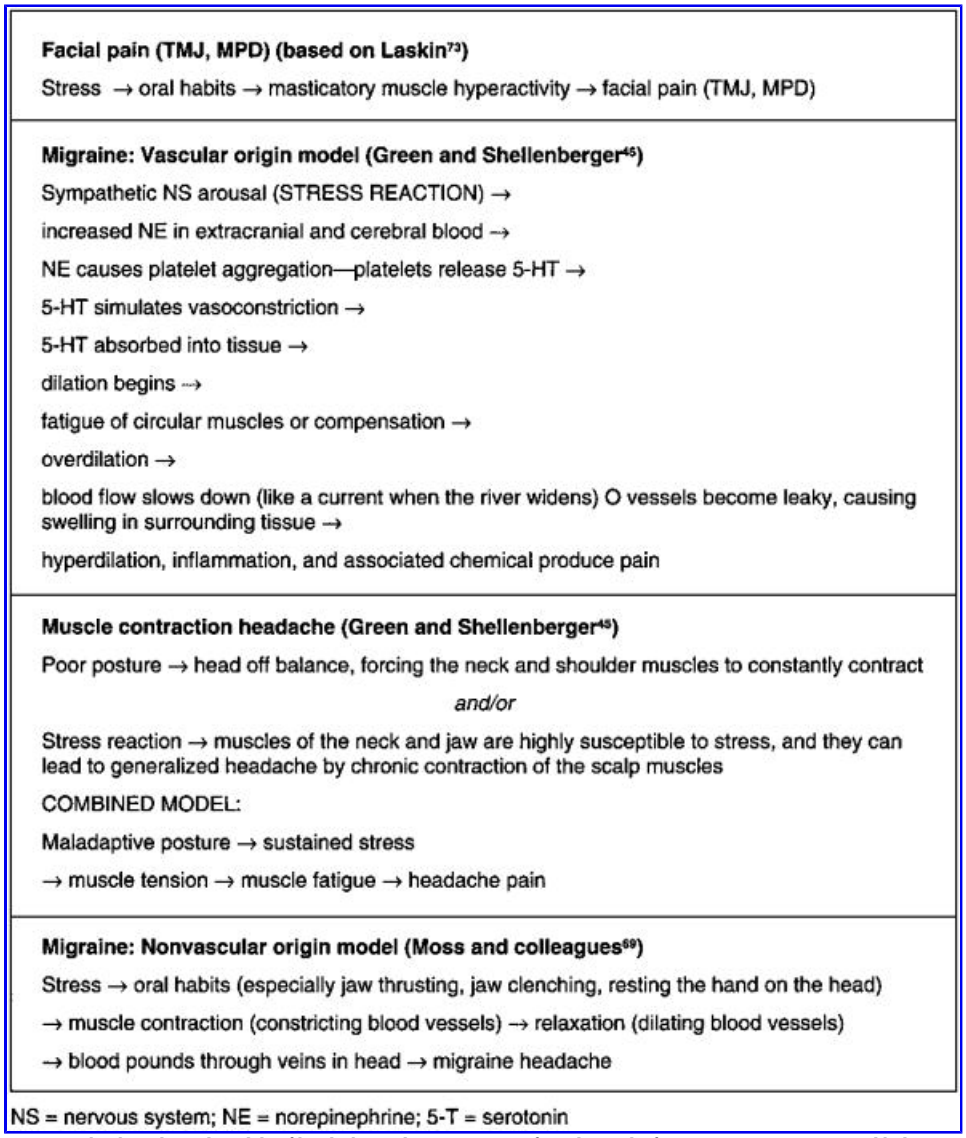

Fig 2. Psychophysiological models of headache etiology. MPD, myofascial pain dysfunction; TMJ, temporomandibular joint dysfunction, TMD, temporomandibular disease.

Regardless of the exact mechanisms in the origin of headache pain, one key component either directly leads to (via behavioral mediators) or exacerbates pain: sympathetic nervous system discharge (ie, the stress reaction). Emotional distress elicits sympathetic nervous system discharge, which results in increased muscle tension. Patients with head or neck trauma already tend to have elevations in head, neck, shoulders, and upper back. 37 Pain becomes a stressor in itself and increases emotional distress. Hence, a "vicious cycle" ensues between pain and emotional distress in the patient made especially vulnerable by trauma to the head or neck.

While many factors may contribute to headache pain (eg, trauma to the head or neck, posture, genetic factors, hormones, weather, foods containing tyramine), the factor that we have the most power to affect is the stress component. The best treatment packages generally contain elements targeting numerous factors. For example, posture may be addressed by awareness training (eg, letting the jaw go slack, not resting head on head, dropping the shoulders combined with deep breathing, resting the arms in the lap instead of on a table or the arms of a chair). Nutrition awareness is helpful to avoid certain foods in susceptible individuals (eg, wine and cheese containing tyramine, a vasocon-strictor). While there is not much we can do about the weather or genetics, tension induced by sympathetic discharge (ie, stress) can exacerbate the pain. The goal of a behavioral program for PTHA is to try and lessen pain, keep it from getting worse, or at least deal with it more adaptively.

Biofeedback

While an abundance of research reports the success of biofeedback for the treatment of tensiontype, migraine, and mixed migraine

and tensiontype headaches, [54] many studies list PTHA among the exclusionary criteria. [34] As a result, few studies have examined the efficacy of biofeedback for PTHA specifically. A number of studies utilized EMG biofeedback (forehead and neck sites) in combination with other treatment modalities (eg, cognitive–behavioral treatment, medication) and reported significant improvement in PTHA. [34,44,55] Ham and Packard [34] reported that combined EMG and thermal biofeedback resulted in at least moderate improvement for 53% of 40 chronic PTHA patients, most of whom had previously received medication, physical therapy, chiropractic treatment, or trigger point injections without significant success. However, it is difficult to make firm conclusions regarding the efficacy of biofeedback alone for PTHA, given the small sample sizes and the use of other simultaneous treatments in these studies.

Although empirical research examining the utility of biofeedback specifically for PTHA is sparse, many clinical researchers feel that biofeedback, when combined with medical treatment and/or psychotherapy, augments the treatment response for many persons with PTHA. Biofeedback-assisted relaxation is assumed to generalize to other aspects of the patient's life in addition to providing specific headache pain relief. Specifically, biofeedback can facilitate a perception of self-control and reduce feelings of helplessness. [37] In their retrospective biofeedback outcome study, Ham and Packard [34] reported that the greatest treatment effects were obtained for general relaxation and ability to cope with pain. Furthermore, most patients continued to use skills learned with biofeedback in their daily lives, supporting the contention that skills lead to generalizable benefits.

Studies that examined biofeedback for PTHA utilized either EMG or thermal feedback, or a combination of both. [34,37,56] The sites most commonly used for EMG biofeedback treatment of PTHA include the forehead, trapezii, frontal-posterior neck, and neck. Arena et al [57] suggest that electrodes may also be placed over muscular sites that are tender upon palpation. Generally, a reading of 2 μV or less is an indication of a relatively relaxed muscle. Thermal biofeedback sensors are generally placed on the second finger, third phalanx, palmar side. Daly and Wulff 56 utilized a protocol in which patients were taught to increase hand temperature at least 2° to 5° F within 30 s. Relaxation training utilized in PTHA biofeedback outcome studies includes progressive relaxation training (PRT), autogenics, deep muscle relaxation, and diaphragmatic breathing. [34] Generally, treatment effects are greater with increasing numbers of treatment sessions. Most protocols used between 9 and 24 sessions of biofeedback each. [34,57]

Relaxation training

Various forms of relaxation training have been utilized for the treatment of chronic headache (eg, autogenics, meditation); however, progressive muscle relaxation (PMR) has been most widely studied. [58] PMR involves the systematic tensing and relaxing of various muscle groups in order to elicit a relaxation response. Each muscle group is generally considered separately during initial training sessions in order to facilitate awareness of particular muscle tension levels. Typically, muscle groups are tensed for 7 seconds, followed by a 45-second relaxation phase. Once this has been practiced, the therapist typically combines muscle groups to shorten the protocol. It has been suggested that six sessions are generally sufficient to obtain proficiency; two daily home practice sessions are advised. [58] Diaphragmatic breathing is generally taught in combination with relaxation exercises.

Metaanalytic reviews generally conclude that relaxation training and biofeedback training are equally effective for headache reduction, producing improvement rates between 44.6% and 59.2% for tensiontype headaches and 52.7% for migraines. [60] The exact mechanism of headache reduction is not clear; however, it is presumed that relaxation training serves to (1) reduce proprioceptive input to the hypothalamus, thereby decreasing sympathetic nervous system activity, and (2) directly reduce muscle tension or preheadache vasoconstriction. [59,60]

Operant treatment

Fordyce [60] pioneered the behavioral approach to psychological assessment and treatment of chronic pain. [58] Although not specifically developed for use with PTHA, the concept follows the operant model to reduce general chronic pain behaviors. The operant model hypothesizes that painrelated behaviors may be positively reinforced by desirable consequences (eg, sympathy, nurturance) and simultaneously negatively reinforced by avoidance of aversive consequences (eg, undesirable work or social obligations). Treatment based on the operant model requires altering environmental contingencies to eliminate pain behaviors (eg, verbal complaints, inactivity) and reward “well” behaviors (eg, exercise, increased activity level).

Cognitive–behavioral treatments

Cognitive approaches for headache treatment are derived from several cognitive theorists and typically train the headache patient to identify and refute maladaptive beliefs concerning pain. [29] Specific cognitive strategies and skills are taught to replace inappropriate negative expectations and beliefs.

Holroyd and colleagues [62,63] have generally led the field in cognitive therapy for chronic headache. [58] Cognitive stress coping therapy has been successfully applied to tensiontype headache patients in group, minimal therapist contact, and homebased formats. [63] Cognitive stress coping therapy proposes that maladaptive cognitive responses are in place that contribute to keeping the headache patient stressed/tense by keeping the sympathetic nervous system activated. Pain pro-tocols based on this approach alter the maladaptive beliefs that mediate the stress reaction (ie, alter the way the individual perceives the stressor) to presumably alter the stress reaction (ie, muscle tension) that leads to increased pain.

Bakal [64] described a general treatment program for chronic headaches that emphasizes attention diversion and thought management as primary components. In essence, the PTHA patient is trained to shift attention from one aspect of the environment

(eg, internal pain) to another (internal or external). A number of attention-diversion techniques to reduce the intensity of pain have been described by Turk. 65 Thought management involves gaining control over negative headache-related thoughts that may increase the pain experience. Distressing thoughts and feelings are identified, and the patient is taught to use such thoughts as a cue to initiate a predetermined list of adaptive statements; the patient is also encouraged to develop his or her own list of personal statements.

Muse 66 described the utilization of systematic desensitization procedures for the treatment of PTHA with a concurrent PTSD diagnosis. Systematic desensitization involves the application of mastered relaxation skills to a devised a hierarchy of feared events, usually in vivid imagery, ordered from least anxiety-provoking to most anxiety-provoking. Anxiety encountered with imagery is countered by switching to relaxation, until each step of the hierarchy can be experienced without anxiety. Through a series of case studies, he noted that the methodological application of reciprocal inhibition techniques significantly improved debilitating anxiety and phobic avoidance of traumarelated events (eg, driving), although headache pain was somewhat less improved. While generalizations are limited due to small sample size, results suggest that systematic desensitization may be effective for the treatment of traumarelated symptomatology among PTHA patients. Clearly, reduction of anxiety can reduce the physiological arousal that contributes to headaches pain.

Social and assertiveness skills training. Miller recommended social skills training in a group format as an adjunct to standard psychotherapeutic interventions for chronic pain. Assertiveness training in particular may help some patients to communicate needs more effectively. This in turn increases the likelihood of need fulfillment and more desirable situational outcomes. Subsequent reduction of stressful events, anger, and other distressful emotional states associated with need frustration can reduce associated physiological arousal that contributes to headache pain.

Imagery and hypnosis. Several studies have reported success with imagery-based treatments for headache in general. 60 Procedures vary by study, but training generally includes autohypnosis and suggestions of relaxation and visual imagery. Generally, the patient is instructed to visualize the pain (ie, give it form) and focus on altering the image to reduce the pain. Imagery-based treatment is recommended following establishment of a good therapeutic alliance in order to facilitate patient compliance.

At least one study documented the application of imagery for PTHA in particular. 56 The authors described the case of a man previously treated unsuccessfully with opiod analgesics, biofeedback, and relaxation. Headache pain was visualized as a ball during an hypnotic trance, with direction given to “blast apart” the ball. Significant improvement was noted after seven sessions. Headache control with relaxation subsequently allowed reduced pain medication and eventual headache remission at treatment week 16, with maintenance at 1-year follow-up.

Biofeedback-assisted cognitive–behavioral therapy. The efficacy of EMG biofeedback and cognitive–behavioral therapy, singularly and in combination in multicomponent treatment packages, has been demonstrated for the treatment of various pain disorders (eg, headache, facial pain). However, the majority of multicomponent treatment packages in the literature to date utilize distinct techniques for biofeedback and cognitive–behavioral therapy. Grayson, 67 however, has presented a unique and promising single-case research design outlining a multicomponent treatment protocol (biofeedback-assisted cognitive– behavioral therapy [B-CBT]) that synthesizes the two in the treatment of chronic traumatic pain patients. The B-CBT protocol combines cognitive, emotional, and physiological (eg, muscle tension) elements to heighten awareness of selfcontrol. It provides immediate physiological feedback during the cognitive–behavioral therapy process to heighten awareness of psychophysiological reactions and facilitate change.

B-CBT, as originally presented by Grayson, can involve conducting situational analyses of specific life stressors simultaneously with psychophysiologic feedback to highlight a patient's specific reactions and shape more adaptive responses through reinforcement, relaxation, role playing, or exposure procedures. The visual and/or audio feedback from indicators of physiological arousal can be very salient, especially for patients with limited awareness of maladaptive responding (eg, increased muscle tension). Preliminary reports suggest that B-CBT may be especially helpful for the treatment of disorders with a high physiological reactivity component, including chronic head and neck pain, and disorders with strong anxiety or anger components.

Notably, the protocol for B-CBT is multifaceted, combining several established techniques. Situational analysis is utilized to focus on specific attributional, emotional, and behavioral response styles in situations within the patient's life. Psychophysiological monitoring provides feedback to the therapist and patient concerning the patient's reactions while simultaneously conducting the situational analysis during the relation of stressful events. The patient can be instructed to review the activating event with the therapist while attempting to maintain his or her physiological response below a preset threshold level. Through the process of shaping, the patient learns to monitor and control physiological reactions in conjunction with reviewing and modifying cognitive and emotional aspects of activating stressful events. In addition, a cognitive exposure method can be utilized by having the patient repeatedly relate the activating event, while attempting to maintain physiological response below a gradually reduced threshold level. Relaxation techniques such as deep breathing and progressive relaxation training may be utilized to achieve selfcontrol of physiological and emotional responses. Printouts of progress and handouts with reminders can be given to facilitate increased awareness of responses and generalization of selfcontrol strategies outside the therapy session.

Initial findings for this procedure have been very encouraging, and further reasearch is warranted. Currently, specific application of B-CBT to PTHA is being conducted by the authors within the context of utilizing simultaneous psychophysiologic feedback

with a variety of cognitive–behavioral treatment interventions.

POSTTRAUMATIC FACIAL PAIN AND HEADACHE: TREATMENT ISSUES

Head, neck, and facial pain are all possible sequelae of traumatic accidents, particularly whiplash injuries. 68 Facial pain (eg, temporo-mandibular disorder [TMD]) and headache frequently cooccur, and some researchers have proposed that TMD and headaches should be jointly considered. 69,70 The stomatognathic system, consisting of the dentition, periodontium, temporomandibular joint, associated musculature, and the cervical region from C1 to C3, is a region commonly affected by some types of headache and facial pain. 70 The fivepart system is closed, meaning that irregularity in one individual component (eg, from posttraumatic injury) will result in functional alterations in one or more of the remaining components.

Empirical evidence suggests that although trauma may produce more significant symptomatology, etiology of pain is not a significant factor in treatment outcome. For example, De Boever & Keersmaekers 71 reported that pain reduction via conservative treatment (eg, occlusal splint and psychological treatment) was equally effective for patients with traumatic (ie, whiplashinduced) TMD and those with nontraumatic TMD at 1-year follow-up. Treatment protocols for headache and facial pain reduction are generally similar and contain many of the same components (eg, biofeedback, relaxation training, cognitive– behavioral therapy). The efficacy of multicomponent packages suggests that such treatments may share a common mechanism of action for idiopathic headache and facial pain. Similarly, treatment packages may be equally effective for headache and facial pain resulting from traumatic injury.

Habit reversal

Gramling et al 72 utilized a habit reversal treatment package to teach facial pain patients to detect, interrupt, and reverse maladaptive habits (eg, maladaptive head/jaw posture, jaw tension, negative cognition). The main premise of the program is that participants can learn specific skills to reverse habits as well as the stressful thoughts and feelings that precipitate these habits. The treatment program begins by teaching exercises that increase awareness of the habit. Awareness training is facilitated by relaxation training exercises, which are taught in conjunction with deep breathing exercises. As pain patients become more aware of maladaptive habits and the situations in which they occur, they are taught to use specific exercises (eg, facial exercises) and deep breathing as competing responses. A similar process is used to help pain patients become more aware of habitual stressinducing thoughts and beliefs.

CONCLUSION

PTHA is the most frequent sequela following trauma to the head, brain, or neck, with an incidence estimated to be as high as 90%. 2 The incidence of chronic PTHA (CPTHA) or PTHA lasting more than 6 months has been found to be as high as 44%. 3 In the current article, relevant studies examining the effect of headache on neuropsychological test findings were reviewed. This review reveals that chronic headache pain, and chronic pain generally, usually has a significant and negative effect on persons reporting persistent subjective complaints. The fact that the pattern of neuropsychological impairments observed for chronic headache and other pain is similar to those produced by mild TBI poses a special challenge to differential diagnostic efforts. The implications with regard to differential diagnosis clearly require that pain be considered a factor in neuropsychological test findings. Further studies are needed to more clearly delineate this relationship, including how such factors as pain severity, type of chronic pain, and pain location specifically impact specific neuropsychological test results.

Furthermore, because PTHA is the most common postconcussive symptom and most frequent type of posttraumatic pain associated with mild TBI, resolution of the post-concussion syndrome and successful posttraumatic adaptation may frequently rely on success in coping with PTHA symptomatology. From a biopsychosocial perspective, a general outline is offered for improving both assessment and treatment of PTH. The most promising current psychologybased treatment interventions are reviewed. Notably, treatments that are holistic in nature, that target not only the pain but also the patient's reaction to it within his or her daily life and that emphasize selfcontrol, typically fare better than treatments with a more narrow focus (eg, medication management or nondrug therapies alone). Multicomponent treatment packages are currently the preferred treatment choice for PTHA. 29,36,37

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Cognitive Impairment in Patients with Chronic Pain: The Significance of Stress

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This review article examines the role of emotional distress and other aspects of suffering in the cognitive impairment that often is apparent in patients with chronic pain. Research suggests that pain-related negative emotions and stress potentially impact cognitive functioning independent of the effects of pain intensity. The anterior cingulate cortex is likely an integral component of the neural system that mediates the impact of pain-related distress on cognitive functions, such as the allocation of attentional resources. A maladaptive physiologic stress response is another plausible cause of cognitive impairment in patients with chronic pain, but a direct role for dysregulation of the hypothalamic-pituitary-adrenocortical axis has not been systematically investigated.

Introduction

This article discusses studies that examine cognitive functioning in patients with chronic pain with an emphasis on the role of emotional distress and the mechanisms of stress-related effects. The prevalence of chronic pain (*ie,* persisting for at least 6 months) in the general US population has been estimated to be in the range of 35 to 75 million [1]. Cognitive dysfunction is one component of the behavioral change that can occur in this common clinical condition. Chronic pain and associated symptoms complicate the presentation of other patients, including those with documented or presumptive brain injury. Although clarifying the impact of momentary pain is important, the concomitants of chronic pain, such as mood change, sleep disturbance, fatigue, and other aspects of suffering (*eg,* lifestyle interference secondary to disability), seem to be more closely related to cognitive impairment.

Sensory-discriminative and affective-motivational components of pain appear to be processed in parallel by different parts of the nociceptive system [2•,3,4]. Medial thalamic nuclei, the anterior and mid-cingulate, and related structures appear to mediate the affective-motivational component of pain. Some of the brain structures involved in processing affective-motivational dimensions of pain presumably are components of the neural system mediating the impact of pain-related stress on cognitive functioning.

Previous reviews have concluded that chronic pain is commonly associated with neuropsychologic impairment [5•,6]. Impairments are most evident on tests assessing attentional capacity, processing/psychomotor speed, and memory. Although many of the previously reviewed studies assessed emotional status or used symptom inventories that included items pertaining to characteristics such as fatigue, mood state, and distress, few studies have systematically explored the relationship between these variables and neuropsychologic test performance. The next section summarizes studies that have not been previously reviewed by Hart *et al.* [5•]. A later section focuses on previously reviewed and more recent studies that specifically address the role of psychologic distress and mood disturbance on cognition in patients with chronic pain.

Recent Neuropsychologic Findings in Chronic Pain Populations

A study of more than 100 patients with head, neck, or back pain who experienced decreased mental and physical functioning [7] underscores the importance of clarifying whether a patient with chronic pain is seeking financial compensation. Twenty-nine percent of those seeking financial compensation, but none of those not seeking compensation, failed two or more of the six neuropsychologic tests used to detect malingering. Heyer *et al.* [8] observed patients before and after they underwent lumbar spine surgery; 12.5% had suffered a previous stroke or transient ischemic attack, 8.3% had undergone carotid endarterectomy, and 42% were administered analgesic medications after surgery. Rated pain intensity correlated with performance on Trail-Making Part A and the Rey Complex Figure Test (RCFT) after the surgery, but not before; however, the relationship between pain intensity and RCFT performance was potentially confounded by medication use. Two other test measures did not correlate with rated pain intensity (Controlled Oral Word Association, Trail-Making Part B). Park *et al.* [9] compared patients with fibromyalgia recruited from a university-based clinic with healthy control subjects. Patients with other significant health conditions or major psychiatric disorders,

including depression, were excluded. The patients were impaired on measures of working memory capacity (reading span and computation span), free recall of a 16-item word list, and yes-no recognition memory of a word list and vocabulary, and performed marginally worse on a measure of verbal fluency. Measures of pain symptomatology, but not of mood or fatigue, correlated with performance on the working memory capacity and verbal memory tests. The patients were not impaired on measures of information processing speed (number, pattern, and letter comparison); however, these tests were simple and brief (*eg,* stimulus strings of three to nine segments and 30-second trials) compared with other tests of information processing speed that have been used to assess patients with chronic pain (*eg,* Paced Auditory Serial Addition Test [PASAT]). Furthermore, a measure of pain-related daily dysfunction correlated with the patient's performance on these measures of information processing speed and with performance on the tests of working memory capacity and verbal memory. Some of these investigators [10•] subsequently reported deficits in patients with rheumatoid arthritis on similar letter and pattern comparison tests of information processing speed in association with higher levels of pain and depression.

A negative finding in a small sample of 10 control subjects [11] is consistent with the type of clinical features identified in an earlier review [5•] that are most likely to be associated with pain-relative cognitive impairment. All 10 subjects were engaged in their normal work activities and had pain symptoms that were localized to one shoulder; the pain did not involve multiple sites or the head, neck, or back region. Pain was of low intensity (median visual analog scale [VAS], 9%) and as short as a 3-month duration. The Hopkins Symptom Checklist (SCL) did not indicate any mood changes or tendencies toward somatization. The three cognitive tests that were administered were brief (5 minutes). The pain group reported a higher level of perceived stress during the tests than the control subjects did, raising the possibility that deficits might have occurred if the tests were more cognitively demanding.

The Role of Emotional Factors and Chronic Stress

The effects of pain on cognitive functioning are not related in a simple fashion to its immediate sensory-discriminative features (*ie,* intensity and location) because the concomitants of chronic pain are the more important mediating variables [5•]. Specifically, cognitive impairment in patients with chronic pain has been associated with mood changes and emotional distress and with symptoms and clinical features such as increased somatic preoccupation, sleep disturbance, fatigue, and perceived interference with daily activities that are potential sources of chronic stress. A "cervicoencephalic syndrome," including dizziness, blurred vision, disturbed adaptation to light, and frequent headache, tends to be associated with cognitive impairment, perhaps as a result of chronic stress because even routine activities often require concentrated effort. The trend toward greater impairment for patients with post-traumatic pain (without evidence of brain injury) than for patients with other forms of pain also is suspicious for the influence of chronic stress related to perceived victimization.

Models of pain processing that distinguish the sensory, affective, cognitive-evaluative, and behavioral dimensions facilitate our understanding of the effects of chronic pain on cognitive functioning. The model that has guided our research distinguishes several stages of pain processing [12,13•,14]. The first stage, a sensory dimension, is commonly assessed by ratings of pain intensity. The second stage or immediate affective response commonly is assessed by ratings of pain unpleasantness. The third stage relates to the meaning and implications of pain for the patient and thus to associated emotional suffering, and is commonly assessed by measuring pain-related emotional states (*eg,* depression, anxiety, frustration) and beliefs (*eg,* perceived ability to endure pain). The fourth stage refers to illness behavior that can be assessed through ratings (*eg,* lifestyle interference) or observed by the examiner or collateral sources (*eg,* pain behaviors manifested at home or during clinical interview).

Considerable research, including studies reviewed in this article and previously [5•], has established that emotional distress frequently accompanies chronic pain. Emotional distress is related partly to specific symptoms, such as sleep disturbance, that are common in chronic pain [15]. Perhaps more importantly, mood change and chronic stress are not surprising because of the restrictions in daily activities, disruptions in preferred role functions, losses of sources of satisfaction and reinforcement, and changes in one's sense of identity and self-esteem that can occur because of chronic pain [16]. Avoidant behavior and reduced activity level can perpetuate a cyclic disability-enhancing pattern of further avoidance in activity and associated emotional distress. For example, Chapman and Gavrin [17] emphasize the distinction between pain and suffering. Suffering entails a disparity between what people believe themselves to be and what people are, which often occurs with poorly controlled chronic pain. Associated psychologic stressors may include feeling a loss of control, hopelessness, fear, and other negative beliefs and attributions. The idea that one's pain is uncontrollable is in itself a stressor. In addition to the stressors of pain and associated negative thinking, sustained maladaptive physiologic stress responses may leave a person feeling sick (*ie,* produce or exacerbate fatigue, dysphoria, muscle aches, sleep disturbance, somatic hypervigilance, and mental inefficiency).

Cognitive complaints for patients with chronic pain are more closely related to measures of emotional distress than to sensory-discriminative aspects of pain, and are

associated with motivational changes, such as a reduced desire for activities [18–20]. The following section discusses studies that relate cognitive impairment to emotional distress and later stages of pain processing.

Several studies have shown that psychologic distress and negative emotions are more closely associated with cognitive deficits in patients with chronic pain than is pain severity. Kewman *et al.* [21] found correlations between ratings of pain intensity, psychologic distress (composite measure of depression, anxiety, irritability, and energy level), interference with daily activities, and a composite score from a cognitive screening measure. When the effect of distress was partialed out, pain ratings no longer correlated with test performance. Grace *et al.* [22] found that pain intensity and trait anxiety (but not depression) correlated with measures of memory and processing speed. When the effect of mood was partialed out, pain intensity no longer correlated with test performance; however, after the effect of pain intensity was partialed out, trait anxiety still correlated with the Wechsler Memory Scale (WMS)-Revised Delayed Recall Index. Landro *et al.* [23] reported that only those patients with a history of major depression had memory impairments, and their rated pain intensity did not correlate with their test performance. Radanov *et al.* [24] found that poor performance on a test of processing speed was associated with lower ratings of emotional well-being and higher levels of self-reported nervousness. In a follow-up study [25], those patients who remained symptomatic and evidenced subtle attentional impairments 6 months and 2 years later continued to rate their emotional well-being lower. Because the latter studies did not include ratings of pain intensity, it is unclear to what extent psychologic distress predicted performance decrements independent of pain severity.

Studies that screened patients with chronic pain for psychiatric illnesses or otherwise that had a narrow range of scores on measures of psychologic distress did not find such associations, suggesting that distress or mood disturbance is only one factor that may contribute to cognitive impairment. For example, Eccleston [26,27] did not find an association between measures of distress from the McGill Pain Questionnaire or of mood disturbance (anxiety, depression) and performance on an attention-demanding numerical interference tasks, but his patients with pain had been screened for treatment of depressive symptoms and "severe emotional problems." In fact, his patients and control subjects had a similar frequency of mood disturbance [27]. Taylor *et al.* [28] reported subtle impairments in processing speed and short-term memory in two different groups of patients with chronic pain. Pain intensity and depression levels did not correlate with test performance, but the authors point out that there was a narrow range of scores on both measures.

Other findings suggest that psychologic distress or suffering in the form of increased somatic awareness or preoccupation is associated with cognitive deficits in patients with chronic pain. Eccleston *et al.* [29] found that only those patients reporting high somatic awareness (operationalized as a greater frequency and breadth of diffuse somatic complaints on a questionnaire) and higher pain intensity were impaired on a version of their attention-demanding numerical interference task. These patients also reported higher levels of depression and anxiety; however, the relationship between the level of emotional distress and test performance was not examined. Other studies have found associations between the level of somatic complaints and cognitive performance in patients with chronic pain, although clinical features that may covary with somatic complaints were not explored systematically. Patients whose temporomandibular disorder (TMD) occurred after a cervical whiplash injury (without a loss of consciousness) reported more somatic complaints on a modified SCL-90 and exhibited more impairment on tests of simple and choice reaction time and memory than patients with idiopathic TMD [30]. However, chronic pain related to whiplash injury may be relatively more severe and widespread [30] or associated with additional symptoms, such as dizziness and blurred vision [24]. Even if litigation is not a confounding issue, emotional reactions and negative attributions related to feelings of victimization also may be important factors. Furthermore, the study by Goldberg *et al.* [30] did not explore the relationship between emotional status and cognitive function. Cote and Moldofsky [31] found that the endorsement of somatic items from a depression scale covaried with ratings of pain intensity and fatigue, and with performance on a simulated multitask office procedure. None of these studies explored whether increased somatic awareness or complaints uniquely contributed to cognitive impairment. Nevertheless, these findings suggest that a somatic focus and associated emotional reactions may increase the disruptive influence of pain on cognition by facilitating access of pain into awareness [29].

Recent studies of patients with chronic pain implicate emotional distress and the later stages of pain processing in relation to cognitive impairment. Iezzi *et al.* [32] evaluated patients with chronic pain who were recruited consecutively from a hospital-based pain service. Pain was musculoskeletal in nature (*eg,* fibromyalgia, myofascial, osteoarthritis), and included patients with multiple pain sites and involvement of the neck or head. Approximately 50% of the patients were taking two or more classes of medication, including opioids. Patients with cancer, neuropathic pain, a history of major psychiatric illness or a history of traumatic brain injury (TBI), or other neurologic disorder affecting brain function were excluded. Statistical clustering procedures were used to identify groups reporting high, moderate, and low levels of emotional distress based on their SCL-90-R profiles. Those patients highest in emotional distress exhibited deficits in attention and processing speed (*eg,* Stroop Test, PASAT), memory (WMS-R Logical Memory and Visual Reproduction),

nonverbal intelligence (Wechsler Adult Intelligence Scale-Revised), and executive functions/abstraction (Wisconsin Card Sorting Test, RCFT Copy) compared with those patients lowest in emotional distress. The performance of the moderately distressed group tended to be intermediate to the other two groups. Deficits were not related to pain intensity ratings, disability/legal status, or medications. Wade and Hart [33] reported findings on the Digit Span Test in a large sample of patients with chronic pain (n = 736) consecutively evaluated at a pain management clinic in a medical center. Approximately 50% of the patients suffered from low back pain. The second and third most frequent diagnoses were myofascial dysfunction and complex regional pain syndrome. Most patients reported multiple pain sites. Patients with cancer-related pain or a history of TBI or neurologic disorders affecting cognition were excluded. The multidimensional aspects of pain were evaluated according to the four-stage model of pain processing. Patients completed VASs of pain sensation intensity (stage 1), pain-related unpleasantness (stage 2), and emotional states and negative illness beliefs associated with suffering (stage 3). A structured pain interview was used to assess illness behavior (stage 4). Step-wise multiple regression analyses were completed using measures of each pain stage as predictors and digit span as the criterion. A final regression analysis using only those predictor variables reliably related to attention in the first set of analyses indicated that measures of pain-related depression, perceived lifestyle interference, and the degree of social reinforcement for pain-related behavior were uniquely related to the deficits in attention span. Deficits were not related to pain intensity. Maladaptive beliefs and negative thoughts relating to perceived lifestyle interference contributed to pain-related suffering. Social reinforcement of pain behaviors (*eg,* solicitous responses) may serve to further increase somatic focus or preoccupation and, secondarily, psychologic distress. A subset of these patients (n = 274) also were administered the Verbal Paired Associate Learning subtest of the WMS. Similar analyses revealed that deficits in verbal learning were associated with pain-related anxiety after controlling for pain sensation intensity [34].

Brown *et al.* [10•] evaluated a large community-dwelling sample of patients with rheumatoid arthritis (n = 121). Measures of cognition included two tests of processing speed (timed letter comparison and pattern comparison), an inductive reasoning test that asked patients to determine the rule that made four of five sets of letters alike, two tests of working memory capacity (reading span and computation span), and the free recall of two lists of 25 words. Two scales were administered that assessed pain intensity (*eg,* pain at different times of the day, after physical activity) and some aspects of suffering or later stages of pain processing (how often pain interfered with activities). A composite measure of depression was derived from subscales of different instruments. High levels of pain and depression were associated with poor cognitive performance in all four areas of functioning (information processing speed, working memory capacity, reasoning ability, and verbal memory). Structural equation modeling indicated that depression mediated the relationship between pain and cognitive functioning (*ie,* chronic pain causes depression, which causes impairment in cognitive functioning). The effects of pain on cognition were no longer significant after controlling for depression. A model with "paths" from pain to depression and from depression to cognition, but not from pain to cognition, explained 55% of the variance in general cognition.

Although the study by Park *et al.* [9] did not find significant correlations between measures of depression and anxiety and cognitive performance, their findings are consistent with the role of later stages of pain processing in the cognitive impairment of patients with chronic pain. As the authors point out, their patients with fibromyalgia were screened carefully for depression; the mean symptom scores were below the cutoffs for even mild depression. Cognitive performance across multiple domains correlated with scores on a pain subscale that primarily measured the functional impact of pain; however, they did not correlate with scores on a pain questionnaire that measured pain intensity in a more focused manner [9]. The functional impact of pain is related in large part to its meaning and the implications for the patient (stage 3) and to resultant behavioral changes (stage 4). Pain-related suffering associated with maladaptive beliefs and with ongoing lifestyle disruption may not be reflected fully in a patient's current mood state.

The often observed relationship between measures of psychologic distress or negative emotions and cognitive performance for patients with chronic pain is perhaps not surprising because of the literature relating depression and anxiety to cognitive impairment. For example, two recent meta-analytic review articles indicate global neuropsychologic impairment in patients who are depressed. Veill [35] found effect sizes ranging from a standard deviation of 0.81 to 0.97 for measures of psychomotor speed (*eg,* Digit Symbol), verbal and nonverbal memory, and visuospatial/visuoconstruction. Veill also found an effect size of 2.0 for measures of mental speed and flexibility (*eg,* Trails B, Stroop Test) [35]. Christensen *et al.* [36] found an average effect size of 0.63 of a standard deviation after performing a wide range of neuropsychologic tests. The largest effect sizes by test category included "attention and tracking" (0.98), "memory mixed" (1.01), and vigilance (1.20). Effect sizes of approximately 1.0 or higher were found for such tests as Digit Symbol, Stroop Test, Benton Visual Retention Test, Buschke Selective Reminding Test, Animal Naming, and the Category Test. For patients with mild depression, the average effect size was 0.21. Prevalence rates for depression in clinic-based chronic pain samples range from 30% to 60% [37]. Some of the brain regions involved in processing the affective component of the pain

experience (anterior cingulate cortex [ACC]) also appear to play a role in the cognitive induction of negative affect in depression. The ACC is integrated with the dorsolateral prefrontal cortex, which is implicated in executive dysfunction in depressive illness [38].

Cognitive impairment and especially memory deficits have been found in combat-related and abuse-related posttraumatic stress disorder (PTSD) [39], and in rape survivors with PTSD who were screened for comorbid psychiatric illness, substance abuse [40], and other anxiety disorders, including obsessive-compulsive disorder, panic disorder, and social phobia [41–44]. Although memory deficits were most common, variable impairment was found for other abilities, including divided attention and executive functions. Studies of healthy subjects indicate that anxiety can negatively impact working memory and information processing [45], learning and memory [46,47], abstraction and problem-solving [47,48], and response inhibition [49], suggesting that subclinical levels of anxiety can be sufficient to interfere with functioning. Patients with chronic pain conditions often report anxiety levels that do not fall within the normal range; a significant amount of the variance in reported pain may be explained by anxiety [50–52]. In a recent study [53], patients with high anxiety reported greater affective responses to cold pressor pain and higher levels of sensory pain, suggesting that anxiety may predispose otherwise healthy people to have negative responses to painful events. Ploghaus *et al.* [54] compared activation responses with thermal stimuli using functional magnetic resonance imaging (fMRI) while varying visual signals to moderate anxiety level. One visual signal (low-anxiety condition) was followed consistently by thermal stimulation of moderate intensity. A second visual signal (high-anxiety condition) was followed by the same noxious stimulation on most trials, but occasionally by a much stronger thermal stimulus. The entorhinal cortex of the hippocampal formation (an area important for memory) responded differentially to the same thermal stimuli, depending on whether anxiety was induced; the activation pattern predicted activity in other brain regions associated with affective pain processing (perigenual cingulate) and intensity coding (mid-insula). The entorhinal cortex may be part of the neural network that mediates the impact of pain-related emotional distress on cognitive functions such as memory.

Neuroanatomic Correlates and the Role of Stress Responses

Anterior cingulate cortex

Neuroimaging studies involving patients with clinical pain consistently have shown changes at the ACC, including areas associated with the emotional response to pain [2•,55]. The ACC is an area of the brain involved in the integration of affective-motivational dimensions of experience and various cognitive processes [56,57]. The affective subdivision of the ACC has connections to structures such as the amygdala, anterior insula, and orbitofrontal cortex and has been implicated in the modulation of affective states and emotional responses and in the evaluation of the emotional valence and motivational salience of information. For example, emotional responses (*eg,* fear and pleasure) may follow electrical stimulation in healthy subjects. Cingulectomy and cingulumotomy have been used to treat patients with pathologic aggression, obsessive-compulsive behaviors, and depression. Activation of this region has been associated with emotional processing (*eg,* recognition of affect in facial expressions, responding to emotionally valenced words) and symptom provocation in patients with psychiatric disorders. In contrast, the cognitive subdivision of the ACC has connections to structures such as the lateral prefrontal cortex, parietal cortex, and premotor areas and has been implicated in the executive control of attention, effortful information processing, and response selection, particularly under conditions that involve novelty, divided attention, conflicting information, working memory, or error detection. This area also is implicated in motor intention/control. Fernandez-Duque and Posner [58] emphasize the role of the ACC in "executive attention" that involves task switching, inhibitory control, error detection, processing novel stimuli, executing novel actions, and allocating attentional resources. Not surprisingly, activity is increased at the ACC in anticipation of a cognitively demanding task.

In regards to attentional control and pain processing, the ACC is part of a cortico-thalamo-mesencephalic network mediating selective attention to painful stimuli, but also is involved in attentional shifting [59]. The latter investigators found that painful stimulation during an auditory discrimination task intended to divert attention from pain produced a regional cerebral blood flow (rCBF) increase only in the mid-part of the ACC. The areas of the ACC involved in orienting responses to painful stimuli and those activated in response to increasing pain unpleasantness appear to be adjacent [60].

The integration of emotional and cognitive self-regulation at the ACC appears to include the executive control of attentional resources under conditions producing emotional distress [56,58,61]. For example, the ACC is activated after error detection or negative feedback, and is the likely source of an event-related potential called error-related negativity. This error-related activity partly reflects the level of motivation for error detection (and thus the allocation of attentional resources) and also is correlated with negative affect (and thus the amount of distress associated with making an error). The amount of the stress associated with making an error seems to depend on the personality dimension of "negative emotionality," which corresponds to the construct of trait neuroticism.

As an area of the brain that modulates emotional reactivity and contributes to the executive attentional system, it is not surprising that the ACC plays a role in pain process-

ing and appears to be an integral component of neural systems mediating the impact of pain-related stress on cognitive functioning. Specifically, the ACC may mediate the affective-motivational and cognitive-evaluative components of the pain experience related to suffering and the allocation of attentional resources under conditions of pain-related emotional distress. The activation of the ACC appears to reflect the degree of pain-related distress experienced [62]. The latter investigator had subjects immerse their hand in water under hypnotic suggestions for increased or decreased pain-related unpleasantness. Positron-emission tomography (PET) scans revealed greater activation during the condition of increased unpleasantness compared with the condition of decreased unpleasantness only in the ACC. Patients with chronic pain treated successfully with cingulumotomy may continue to experience painful sensations, but not the associated emotional suffering; anxious patients tend to be more likely to benefit from the surgery [63].

It has been speculated that, because of the extent to which the ACC is involved in pain processing and attentional mechanisms, the competitive demand may interfere with cognitive functioning [34]. Because painful stimulation and related emotional distress are attention-demanding, the reserved capacity of the ACC to allocate attentional resources may be limited. The plausibility that competitive demand on finite attentional resources may be expressed at the ACC is supported by neuroimaging studies. PET scan findings suggest that the interference effects in performing two attention-demanding tasks simultaneously can occur centrally at the ACC or prefrontal regions [64]. The areas of the ACC that are activated by painful stimuli partially overlap those activated during orienting responses and target detection [2•]. Another study found substantial ACC regional activation overlap on PET scans when patients performed a task that required sustained and divided attention during noxious heat stimulation [65].

The meta-analytic study of Bush *et al.* [56] indicates that the cognitive subdivision of the ACC is activated by various attention and cognitive-demanding tasks, and deactivated (*ie,* reduced blood flow or fMRI signal) by tasks that relate to emotional content; the affective subdivision shows the opposite pattern of activation and deactivation. The suppression of the cognitive subdivision during tasks with emotional content (and vice versa) may be a mechanism involving the ACC by which emotional distress disrupts attentional control and cognitive efficiency. In particular, cognitive subdivision activity may be suppressed during pain-related distress or the processing of the affective-evaluative components of the pain experience (*ie,* suffering). Depressive illness, experimentally induced emotional states in healthy subjects, and the anticipation of pain have all been associated with the deactivation of the cognitive subdivision of the ACC [56]. Because of the identified role of the cognitive subdivision of the ACC, effortful information processing likely is to be disrupted with pain-related distress, whether by mechanisms related to competitive demand on finite attentional resources or reciprocal suppression. These information processing demands include those related to the proposed role of the ACC in responding to pain (*eg,* selecting and organizing motor responses and mediating the learning associated with prediction and avoidance of noxious stimuli) [57]. In addition to its role in information processing, there is some evidence that the ACC contributes to a memory system [66,67]. Memory processes may be potentially disrupted with pain-related distress by the same mechanisms of competitive demand, interference, or reciprocal suppression at the ACC.

Stress-mediated mechanisms

The effects of chronic stress on cognition and the mechanisms by which stress results in changes in brain structure and function have important implications for cognitive functioning in patients with chronic pain. Manifestations of chronic stress, including excessive sympathetic nervous system activity, neuroendocrine response, and possibly immune system activity, may be part of a maladaptive response in patients with chronic pain [17]. Dysregulation of the hypothalamic-pituitary-adrenocortical (HPA) axis has been associated with mood disorders and PTSD, presumably reflecting the effects of chronic stress or maladaptive stress responses for patients with these disorders. Furthermore, patients with mood disorders and PTSD have reductions in brain volume, particularly in the region of the hippocampus [39,68,69]. For patients with a history of recurrent major depression, hippocampal volume showed an inverse correlation with the total days the patients were depressed [69]. The idea that chronic stress may produce reductions in brain volume is supported by recent findings in healthy control subjects [70]. Trait neuroticism, which reflects the general tendency to experience negative emotions or "stress reactivity" (as characterized by these investigators), showed a negative association with the ratio of the brain to the remainder of intracranial volume. Specifically, the tendency to experience anxiety and negative emotions (*eg,* shame or embarrassment) in social situations was related to reductions in brain volume. The authors note that trait neuroticism has been associated with hypercortisolemia and with an increased future incidence of affective and anxiety disorders. It has been reported that 20% of patients with chronic pain from a multidisciplinary pain program met predefined criteria for one of four McGill-Melzack Pain Index pain subgroup classifications ("emotionally overwhelmed") and had elevated scores on the same measure of trait neuroticism [71].

A considerable amount of evidence exists that implicates chronobiologic dysregulation of the HPA axis in patients with chronic pain. Elevated cortisol levels and reduced melatonin concentrations in patients with cluster and migraine headaches [72,73] have been interpreted as a disorder of hypothalamic circadian function [74]. In a

study of patients with TMD pain, Korszum *et al.* [75] found markedly increased daytime cortisol levels and a 1-hour phase delay in the timing of maximum cortisol levels relative to control subjects. Findings in patients with fibromyalgia include normal to increased plasma adenal glucocorticoid secretion in the morning and evening, but low 24-hour urine-free cortisol levels and an exaggerated pituitary response to challenge testing [76]. Extended studies at 10-minute intervals over a 24-hour period found significant differences in the pattern of circadian secretion of adrenocorticotropic hormone and cortisol, with a general hypoactivation. Crofford [77] has reviewed evidence of HPA axis involvement in acute and chronic pain, including the fibromyalgia spectrum of somatic pain and rheumatic diseases. Ehlert *et al.* [78•] noted that, in contrast to HPA axis hyperactivity in depression, the findings for PTSD, chronic fatigue, and stress-related bodily disorders, such as idiopathic pain syndromes, frequently suggest diminished HPA activity. Thus, findings of HPA dysregulation in chronic pain include hyperactivity and hypoactivity. This is consistent with a chronobiologic disturbance characterized by aberrant HPA activity and cortisol levels that are variably higher and lower in patients with chronic pain than in control subjects over the course of a day, and complex interactions with other neuroendocrine functions, especially those relating to melatonin, serotonin, and endogenous opioids [79].

Additional evidence of dysregulation of the HPA axis in pain syndromes comes from studies examining treatment effects for acute and chronic pain. Specifically, pain relief is associated with reduced cortisol levels and HPA activity. For example, local anesthesia can reduce immunologic and hormonal responses (*ie,* increases in plasma epinephrine and serum cortisol) to acute painful stimulation [80], and septal stimulation-inducing analgesia can produce a decrease in plasma cortisol and in gastric ulceration [81]. Preoperative acupuncture can reduce postoperative pain and nausea and plasma cortisol and sympathoadrenal system activity in patients who have undergone surgery [82]. Sugano and Nomura [83] found that water exercise and stretching programs produced lower salivary cortisol, subjective pain, and anxiety scores in patients with chronic low back pain. Bellometti and Galzigna [84] found that a combination of mud pack and antidepressant treatment positively rebalanced the stress response system, reduced pain symptoms, and improved the quality of life in patients with fibromyalgia. Pizzoferrato *et al.* [85] found that a single thermal mud treatment significantly decreased plasma cortisol and pain complaints for patients with osteoarthritis. Microcurrent cranial and body area stimulation studies have shown elevated plasma serotonin, β-endorphin, γ-aminobutyric acid, and dehydroepiandrosterone, along with diminished levels of cortisol and tryptophan and concomitant improvements in symptoms of pain, insomnia, and depression [86].

The hypothesis that maladaptive stress responses may be a cause of cognitive impairment in patients with chronic pain is plausible because of the evidence for a link between stress-related dysregulation of the HPA axis and neuronal plasticity. McEwen [87] reviewed animal and human research that supports a link between stress and neuronal plasticity at the hippocampus. Repeated stress suppresses neurogenesis of dendate gyrus neurons and produces atrophy of dendrites in the CA3 region. Dendritic atrophy appears to be the result of excitatory amino acid (glutamate) release during repeated stress, which is facilitated by circulating adrenal steroids. Chronic adrenal steroid (*eg,* glucocorticoid) exposure increases *N*-methyl-D-aspartate (NMDA) receptor binding. Serotonin released in response to stress also may facilitate excitatory amino acid activity at the NMDA receptor. Glutamate is a key neurotransmitter that mediates central nervous system hypersensitivity, including sensitization associated with chronic pain [88–90]. Glutamate increases intracellular levels of calcium ions (Ca^{2+}), which has been linked to excitotoxic effects. Sheline *et al.* [69] suggest that, in addition to glucocorticoid-induced neurotoxicity, other potential mechanisms include corticotrophin-releasing factor neurotoxicity and the loss of neuroprotective brain-derived neurotrophic factor.

Traumatic stress and elevated adrenal steroids are associated with signs of atrophy in the human brain [87], but reductions in brain volume in psychiatric disorders such as PTSD and recurrent major depression suggest that the human hippocampus is particularly sensitive. Prolonged glucocorticoid exposure reduces hippocampal cell number [91]. Chronically elevated levels of glucocorticoids in healthy elderly subjects may be associated with hippocampal atrophy [92]. The hippocampus is a primary target for adrenal steroids. Increased glutamate production and release in response to HPA activation has been implicated in reduced hippocampal volume in patients suffering from anxiety and depressive disorders [93,94]. A metabotropic glutamate receptor antagonist (2-methyl-6-phenylethynyl-pyridine) has been shown to possess antidepressant and anxiolytic properties [95]. In patients who are depressed, volume loss in the hippocampus correlates with volume loss in the core nuclei of the amygdala where glutamatergic pyramidal cells predominate [69]. The latter author suggests that over-excitation in one structure can produce damage in the other through reciprocal connections between the hippocampus and amygdala.

Animal models of stress-induced atrophy suggest that periodic HPA axis activity is sufficient to cause damage [87]. McEwen [87] postulates that repeated HPA and associated autonomic and neurochemical reactivity to experiences in the course of PTSD and recurrent depressive illness may underlie progressive neuronal structural changes and eventually cell loss. This same explanation would seem consistent with evidence of reduced brain volume in patients high in trait neuroticism who tend to

experience more life events as stressful and are more susceptible to psychologic distress.

Regional brain volume loss hypothesized to be a consequence of repeated stress and elevated cortisol levels have been associated with cognitive impairment in human subjects. For example, Bremner [39] reported that deficits in memory correlated with decreases in hippocampal volume in one of their PTSD patient samples; Sheline *et al.* [69] found impaired verbal memory and smaller hippocampal volumes in patients with a history of recurrent major depression who were in remission. Administration of the endogenous glucocorticoid cortisol can impair memory function in healthy subjects [96,97]. Newcomer *et al.* [97] did not find effects for tests of sustained or selective attention, but Kirschbaum *et al.* [96] found effects for tests that required spatial thinking and memory (judgements about spatial location after mental rotation or reversal of direction). Memory deficits occur in association with stress-induced (*eg,* public speaking, mental arithmetic) cortisol elevations in healthy subjects [96,98]. Lupien *et al.* [98] identified approximately one third of their group as cortisol "responders;" sampling over time showed that cortisol levels increased early-on, suggesting that anticipation of stress played a significant role in the observed memory deficits in this subgroup. Twelve-hour urinary-free cortisol excretion has been negatively associated with memory performance in healthy older women, but not men [99]. Longitudinal studies of healthy older adults have shown that increases in cortisol levels over a period of years is associated with cognitive impairment and decline in memory [99,100]. Lupien *et al.* [100] found that the slope of the change in plasma cortisol levels over a period of up to 4 years was correlated negatively with cognitive functioning. The patients who showed an increase in cortisol levels and had a high basal level in the final year were impaired on measures of verbal paired-associate learning and selective attention (visual search task), but not on measures of memory from the WMS or on tests of divided attention or naming. A 1-year follow-up study also demonstrated an impairment in vigilance in those patients with increasing cortisol levels who did not develop the same degree of hypersecretion [101]. Furthermore, prolonged cortisol elevations predicted hippocampal atrophy [92]. Seeman *et al.* [99] similarly found that increases in the level of urinary cortisol excretion over a period of almost 2.5 years was associated with actual declines in memory performance in women (but not men), although there were no effects on measures of abstraction and visuoconstruction. In a recent study, Lupien *et al.* [102] demonstrated some of the complexity in relating cortisol levels with cognitive impairment. Pharmacologic manipulation of glucocorticoids had different effects on memory function depending on a patient's cortisol history. Inhibition of cortisol secretion with administration of metyrapone impaired memory in those patients with a history of moderate cortisol levels; the deficit was reversed after hydrocortisone replacement. In contrast, metyrapone did not effect memory in those with a history of high cortisol levels and current memory deficits, but hydrocortisone treatment significantly decreased delayed memory. Memory problems associated with repeated stress and HPA axis dysregulation may be specifically mediated by glutamate's neurotoxic effect on the hippocampus. For example, Alvarez and Banzan [103] have shown that chemical stimulation of ventral hippocampus glutamate receptors inhibit learning and memory in adult rats. A metabotropic glutamate receptor antagonist can reverse the learning deficit.

The finding of Lupien *et al.* [98] that memory impairment was associated with early cortisol elevations corresponding to the anticipation of stress is particularly important for understanding the role of stress in the cognitive functioning of patients with chronic pain. The belief that pain symptoms are inevitable is a core feature of the chronic pain syndrome for many patients. The anticipation of pain is a significant psychologic stressor that may be associated with dysregulation of the HPA axis. Furthermore, the anticipation of pain has been associated with modified activity at ACC regions that play a role in effortful information processing [56] and at ACC regions that have been implicated in anxiety and stress reactions [2•]. Therefore, the anticipation of pain may disrupt information processing and the allocation of attentional resources through neuronal activity at the ACC. A recent study showed that anticipation of an impending and unpredictable pain stimulus increased rCBF in the caudal portion of the ACC and anticipation of an inevitable, but predictable pain stimulus resulted in a decrease of rCBF [104]. Subjects in this study reported promptly attending to or intentionally distracting their attention from pain. To the extent that aspects of chronic clinical pain are difficult to predict, this observation raises the possibility that a patient may become increasingly vigilant, emotionally aroused, and somatically focused, which could have the effect of reducing the capacity for effortful cognitive processing.

The negative effect of HPA axis dysregulation on cognitive function is consistent with the idea that the concomitants of chronic pain (*eg,* emotional distress) are related to neuropsychologic deficits. It is plausible that the patterns of HPA axis dysregulation in chronic pain represent variants of the periodic HPA and the autonomic and neurochemical reactivity that McEwen [87] postulates may underlie progressive neuronal changes. There is evidence from longitudinal studies [99] that a decline in cortisol levels may be associated with an improvement in cognition. Effective treatment of chronic pain symptoms may minimize repeated HPA axis activation, reduce cognitive inefficiency as a result of associated distress, and partially reverse any stress-related changes in brain structure or function.

Conclusions

The studies that have been reviewed suggest an association between psychologic distress and cognitive impairment in patients with chronic pain. In particular, pain-related negative emotions and variables that mediate suffering (*eg*, interference with activities and increased somatic vigilance) have been identified in a number of studies as correlates of cognitive impairment. Studies employing multiple regression analysis or structural equation modeling have extended earlier findings that suggested that psychologic distress is related to cognitive impairment independent of the effects of pain intensity. However, beyond this general distinction between sensory-discriminative and later stages of pain processing, research has not systematically and comprehensively explored the interrelationships among the numerous variables that potentially mediate the association between psychologic distress and cognitive impairment.

The ACC appears to be play an important role in pain processing, especially with regards to the affective-motivational dimension of the experience. As an area of the brain that modulates emotional reactivity and contributes to an executive attentional system, it may be an integral component of the neural system that mediates the impact of pain-related emotional distress on cognitive functioning, including the allocation of attentional resources. Mechanisms related to competitive demand on resources, interference effects, or reciprocal suppression between the affective and cognitive subdivisions of the ACC may underlie the disruption of cognitive function. Maladaptive physiologic stress responses and dysregulation of the HPA axis is another plausible cause of cognitive impairment in patients with chronic pain, including memory deficits secondary to effects on hippocampal function. Physiologic responses to repeated stress have been related putatively to changes in brain structure and function. A particularly intriguing possibility is that the anticipation of pain symptoms that are difficult to predict, especially in those patients who are high in trait neuroticism, is a significant stressor that repeatedly activates the HPA axis and ACC areas, resulting in cognitive impairment.

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Chapter 12

ETHICAL CHALLENGES IN THE NEUROPSYCHOLOGY OF PAIN, PART I

Michael F. Martelli

Scenario 1

A neuropsychologist performed an independent examination, the examinee's third, upon referral from a worker's compensation insurance company. The examinee was three years status post an unwitnessed injury in which a 175-pound steel beam fell ten feet, striking the worker's head, breaking his hard hat and causing him to fall to the ground and experience an uncertain loss of consciousness and an uncertain period of altered consciousness. Notably, he had been working while diagnosed with "walking pneumonia". After his accident, he was taken to a company nurse but refused medical treatment and went home for the rest of the day and the weekend. He reportedly slept most of the weekend, and apparently assumed that his symptoms of dizziness, nausea, confusion and several blackouts were results of his pneumonia. After the weekend, he attempted to return to work for several days but continued to have intermittent symptoms and was sent home each day. During the first week, his symptoms failed to improve, and he developed intense headaches. After a "blacking out" episode, his wife took him to the emergency room.

The examinee's headaches, dizziness, nausea and variable and intermittent confusion continued, but while his symptoms abated somewhat, his headache severity persisted. He subsequently received continuing assessment and treatment that over the next two years included multiple physicians with multiple specialties, without significant benefit in terms of primary persistent chronic head pain symptoms, as well as family complaints of problems with information processing, memory and irritability and anger. He did not return to work and was placed on short-term and then long-term disability.

On two occasions, the examinee received inpatient psychiatric hospitalization after bouts of severe depression with homicidal and suicidal thoughts and "personality deterioration". These were associated with reports of difficulty coping with pain, aggressive outbursts and fear of hurting family members. Diagnostic assessments continued and a tentative diagnosis of posttraumatic epilepsy was made based on variable EEG assessments, while CT and MRI findings revealed acute right frontal lesion that resolved on CT but not MRI, along with some evidence of an additional older (pre-injury) lesion also in the right frontal lobe. Sleep studies corroborated patient and family report of significant sleep disturbance. Primary treatments, however, were focused on chronic pain management and adjustment related problems, with perhaps overly aggressive medication management. At the time of this examinee's most recent independent examination, he had been enrolled in a residential, dual focused chronic

pain and brain injury rehabilitation program. He had made measurable overall progress, albeit against a backdrop of a sawtooth pattern of functioning, and was being transitioned to a modified intensive outpatient treatment program.

This third independent examination was scheduled by the worker's compensation insurance case manager after discharge from residential treatment. The insurance company refused authorization for outpatient treatment and made a settlement offer that was rejected by the patient, who refused to negotiate and insisted on continued treatment. The case manager selected and scheduled an independent examiner with no training, knowledge, experience or recognition in chronic pain disorders, despite recommendations from the treating physician and neuropsychologist for an examiner experienced with chronic pain disorders. Based on his record review and examination, this examiner diagnosed malingering and concluded that the examinee's behavior was best explained by sociopathy, opining that continued medical treatment was unnecessary and would not be beneficial.

Upon receipt of this independent examiner's report by the worker's compensation case manager, all workers compensation medical and wage benefits were immediately discontinued. The previously supportive employer terminated the patient upon recommendation of their attorney. The worker's compensation case manager informed the treating practitioners of the results of the independent examination and instructed them to discontinue all treatment and medications. Without medical benefits or compensation payments, the patient paid out of pocket for a reduced medication regimen and limited outpatient treatment. He appealed the case. Although he was granted social security disability benefits, he accrued increasing debt and interest charges from borrowing to pay bills. He also experienced drastically increased personal and family stresses, a significant interruption in his treatment, and complication of his symptoms and course. He deteriorated in physical, neuropsychological, and interpersonal status. After one year of enduring numerous insurance company attorney delays for an appeal hearing, he won the appeal and all benefits were restored.

The unqualified diagnosis of malingering and the conclusions and recommendations listed in the report were offered based on seemingly supportive evidence from interview, neuropsychological examination, and medical record review. Despite the appearance of providing sufficient support, a critical review of the examination and report revealed both a selective review that excluded more prominent disconfirmatory evidence, and several critical conceptual and methodologic errors.

With regard to this evaluation, the diagnosis of malingering, which had significant consequences for harming this patient, is considered problematic for several reasons. The examination was conducted by a professional in a subject area outside his expertise. The examiner conducted a very brief interview with selective medical record review. The examiner performed an incomplete review or consideration of historical information, including critical disconfirmatory information. The examiner failed to adequately consider differential diagnostic factors. The examiner did not include any of the many appropriate pain complaint response bias measures. And, the examiner offered a strong and unqualified opinion without appropriate recognition of the numerous important conceptual and methodological limitations.

Relevant Ethical Issues

Competence

Performance of an examination, especially a more demanding medicolegal examination for which chronic pain is the primary complaint, despite lacking specific training, experience and competence in this area violates General Principles A, B and D. These relate to efforts regarding promoting benefit and avoiding harm (A), managing

conflicts of interest to avoid harm (B), and protecting fairness and justice through precautions against insufficient competence and experience (D). It also fails the specific obligations prescribed in at least standards 2.01 (services only within boundaries of competence, education, experience), 2.04 (basing judgments on knowledge of the discipline) and 3.04 (avoiding potential harm).

Assessment

In this case, the examiner conducted a brief and insufficient interview based on report content, listing of one hour by the examiner, and wife report of it actually lasting thirty minutes or less. The examiner also failed to obtain and/or adequately review sufficient premorbid medical, work, military records, the records of the most recent treating rehabilitation neuropsychologist, or family records. Notably, evidence from pre-injury military, employment records and family report indicate an adaptive history that is inconsistent with sociopathy and malingering. He played on an army baseball team even with a fractured fibula, had very good work performance reviews, an average of fifteen hours overtime per week for the six months prior to his injury, and was working with pneumonia at the time of his injury; positive family functioning reported by family and coworkers is notable for absence of behavioral or emotional problems or conflicts. Therefore, in addition to violating Principles A, B, C (Integrity: ensuring accuracy), and D, the examiner also violated Standard 3.04 (avoiding potential harm), in addition to obligations relating to assessment.

Standard 9.01 (Bases for Assessments) requires that opinions be based on sufficient information and techniques to substantiate findings. Further, the failure to include appropriate, direct measures of pain response bias that could support a diagnosis of malingering additionally deviates from obligations in Standard 9.02 (Use of Assessments) relating to use of appropriate techniques with demonstrated utility (9.02a) and established reliability and validity (9.02b). For example, Martelli, Zasler, Nicholson, Pickett, & May (2001) list numerous indicators of response bias in pain symptom report. This examiner did not use any, and instead, made leaping generalizations about malingering of pain on the basis of measures of cognitive response bias. Further, the failure to acknowledge and report any of the multiple limitations in procedures and methodology transgresses Standard 9.06 (Interpreting Assessment Results), which prescribes the taking into account limitations of interpretations.

Even greater concern must be raised in light of the additional observation of apparent confirmatory bias and selective medical record review. Notably, all evidence potentially consistent with malingering was considered much more strongly than the preponderant contradictory opinions and evidence. The examiner was notably vigilant to secondary gain while completely ignoring (assessment of) secondary losses (e.g., life disruption of strong premorbid coping style for deriving reinforcement, self esteem and identity, and coping with stress through traditional male role activities at work and home). The examiner also failed to mention or consider important reactive contextual factors (e.g., credible perception of insurance company "games"). This, again, transgresses Standard 9.06, which requires taking into account the situational and personal factors that might affect or reduce the accuracy of interpretations.

The examiner demonstrated conspicuous predilection for dichotomous sociopathic explanations for behavior, at virtually every point in inferential reasoning. Posttraumatic organic and reactive explanations for behavior were summarily discounted. For example, irritability and anger outbursts were attributed to sociopathy. However, the combination of negative premorbid history and neuroimaging evidence of a right frontal insult, especially when combined with evidence of a high association between chronic pain disorders, anger, and violence (e.g., Bruns, Disorbio, & Hanks, 2003), were not considered through

differential diagnostic review, apparently due to a combination of bias and lack of competence in chronic pain. Further, no apparent consideration was made that true organic impairment can co-occur with exaggeration or malingering. Finally, the examiner engaged in unethical behavior by offering strong and unqualified opinions despite lack of expertise in the subject area (Standards 2.02 and 2.04); by performing an inadequate assessment (Standards 9.01 and 9.02); and through his failure to express resulting limitations of the assessment procedures (Standard 9.06), interpretations, and the resultant potentially harmful consequences of the conclusions. These behaviors also clearly infringed on General Principles A (avoidance of harm) and D (Justice), which prescribes taking "precautions to ensure that their potential biases, the boundaries of their competence, and the limitations of their expertise do not lead to or condone unjust practices."

Conflict of Interest

Finally, conducting this evaluation seems to breach Principle A (managing conflicts of interest to avoid harm) and Standard 3.06 (Conflict of Interest). The latter proscribes taking on professional roles when objectivity, competence or effectiveness might be impaired, or where it might risk harm. Because this examiner accepted an evaluation outside of his areas of competence and the area where he conducts clinical assessments, motivation and desire for more lucrative medicolegal work presumably influenced his judgment and decision, resulting in compromised competency of procedures, non-objectivity, and harm. The enticing financial incentives in medicolegal work are being increasingly recognized as underappreciated threats to objectivity (e.g., Martelli, Bush, & Zasler, 2003; Martelli, Zasler, Nicholson, Hart, & Heilbronner, 2001, 2002; Martelli, Zasler, & Grayson, 1999). In summary, the neuropsychologist's behavior violated, on multiple counts, the spirit and the letter of the Ethics Code.

Case Resolution

After the examinee's worker's compensation benefits were discontinued, his attorney procured a copy of the "independent examination" report. His treating physician and his neuropsychologist prepared a lengthy letter addressing justification for continuing treatment and concern for potential harm that could result from treatment termination. As part of this justification, the numerous methodological problems with the report on which the insurance company based its decision were outlined in detail. Recommendations were made for continued treatment at least until an independent examination was performed by a qualified expert. The letter, which included references to all of the concerns delineated in this scenario, was copied to the independent examiner, along with an note expressing concern that the issues raised in the letter addressed several apparent ethical breaches and that discussion seemed necessary.

When the insurance company refused to reconsider their decision, the patient's wife, after collaboration with the patient, family, attorney and state board of disability rights, appealed the decision and began completing a formal complaint against the examiner with the state licensing board. Because of the family decision to file formal complaint, no further contact or action was taken by the treating doctors, and the examiner also made no attempt at contact.

Conclusions and Recommendations

In this scenario, a neuropsychologist performed an independent examination and diagnosed malingering in an individual who was experiencing chronic pain and persistent cognitive sequelae following a blow to his head. This diagnosis produced significant negative consequence for the subject in terms of health care access, finances, relationships within the family, and general functioning. However, the assessment methodology,

interpretations, conclusions and recommendations offered in the independent examination report clearly failed to satisfy several aspirational guidelines and several specific Ethical Standards outlined in the 2002 APA Ethics Code. An inadequate evaluation that transgressed numerous ethical standards served as the basis for a diagnosis that produced significant life consequences and the penultimate ethical breach – the careless and unjustified infliction of harm.

Martelli and Zasler (2001), in one of the major volumes on pain management, addressed ethical issues relating to competency and offered a checklist summary of guidelines for evaluating professional expert qualifications in chronic pain. This checklist was employed to rate the neuropsychologist in this scenario. The neuropsychologist's score of 0 of 48 was included in the letter written to the insurance company, and copied to the neuropsychologist, to argue against the acceptance of the malingering diagnosis and termination of benefits. A successful appeal took one year to reverse the insurance decisions that were based on the deficient examination of the neuropsychologist, at a considerable psychological cost to the individual and his family.

The positive changes in the 2002 Ethics Code more specifically define competence and emphasize disclosing limitations and promoting more just, equitable and transparent practice. The qualifications summaries offered by Martelli and Zasler (2001) in the areas of both chronic pain and brain injury (available at http://villamartelli.com) can assist in specifically assessing extent of competency and in prescribing activities to increase or maintain professional competency in the neuropsychology of pain.

Scenario 2

An experienced, well-known and respected neuropsychologist who specializes in forensic neuropsychological assessment agrees to perform an injury related clinical evaluation on a local chiropractor. The neuropsychologist had received treatment at the chiropractor's office over at a five-year period and had even been treated directly by him on at least couple of occasions. The chiropractor was seen for a neuropsychological evaluation one week after being hurt accidentally while shopping at an industrial construction store. He was struck in the head by a malfunctioning metal spring-loaded security camera and was knocked to the floor. He sustained a loss of consciousness of approximately fifteen minutes, experienced a three-hour period of posttraumatic amnesia (PTA), and sustained a back injury, per emergency room records. He was released from the emergency room several hours after arrival. The neuropsychological report, which was copied to the client's attorney, indicated twenty-four hour period of PTA based on interview of the patient's wife, and reported significant cognitive deficits, significant emotional distress, and head and back pain. Despite a reasonable neuropsychological battery, only very weak checklist measures of pain and emotional status were administered, along with a single measure of response bias. A diagnosis of mild to moderately severe traumatic brain injury (TBI) was given. Emotional distress and pain were not considered as possible influences on neuropsychological test findings. However, pain and distress were offered as explanations for a borderline performance on the symptom validity measure. No recommendation or referral for psychotherapy, or any strategies for reducing emotional distress, were offered. The neuropsychologist followed the patient and reported in a note a couple of weeks later that he was limiting scope of treatment to assessment and individual and family consultation to avoid a "dual relationship" that would be incurred with psychotherapy provision.

Several months later, the neuropsychologist (NP1) made a referral to another neuropsychologist after the patient's insurance changed to a company for which he was not a provider. Up to that point, no recommendation was made for reducing emotional distress. The patient was

seen by the second neuropsychologist (NP2), an experienced TBI specialist, and evaluated two more times over a two year period. He was also referred to a psychiatrist for pharmacologic treatment of depression and to a multidisciplinary chronic pain management clinic. He subsequently underwent corrective back surgery.

A third neuropsychologist (NP3) was retained by the defendant's attorney to do an independent exam (IE) as a personal injury trial date approached. He was retained with the understanding from an initial record review that NP1, who was a friend, had withdrawn from treatment. NP3, on record review of repeated previous testing found indices of reduced motivation, inconsistent and improbable performances, somatic hypervigilance, and even observations that pain interfered with attention during testing and interview. On independent examination, he found that the chiropractor demonstrated: (a) Failure on several less well known response bias indicators; (b) stark inconsistencies throughout the evaluation; (c) clearly disruptive pain behavior that interfered inconsistently with attention during simple interview and testing (e.g., scores up to twice as impaired on similar tasks when he appeared to be having exacerbation of pain); (d) significant emotional distress, including interview-elicited evidence of a fairly extreme persistent rumination about perceived mistreatment from his injury and need to seek justice for the wrong; and (e) generalized severe impairments worse than usually seen in severe TBI and without evidence of any improvement across time. A report was issued that reported persistent neuropsychological deficits being due primarily due to an interaction of emotional distress, chronic pain, and motivation to exaggerate impairment. Aggressive psychological intervention was recommended.

After completing the independent examination, and while reviewing additional records, NP3 learned that NP1 had recently been retained by the plaintiff's attorney and performed a complete forensic examination that included reviewing of all records. He testified in a deposition that the patient was permanently disabled with severe neuropsychological impairments due to the TBI. His test performance was deemed valid, despite some suspicious symptom validity test scores, because he consistently scored in the same poor range across all testings post injury. Moreover, NP1's deposition testimony asserted that he had known the patient pre-injury, understood his personality and cognitive functioning, had good comparative data, and therefore had special qualification for more validly assessing post injury changes. In response to questioning about why a recommendation for psychotherapy and/or pain management was not made, he asserted that it was because he knew the patient's personality was consistent with a need to appear normal and feared suggestion of psychotherapy might make him "worse".

Relevant Ethical Issues

Multiple Relationships

There are several important ethical problems in this complex scenario. The most salient problem is the engagement in a multiple relationship that initiated a host of subsequent breaches related to assessment and conclusions. NP1 chose to initiate a clinical relationship with a person with whom he had a pre-existing professional relationship. Standard 3.06 (Conflict of Interest) proscribes taking on a professional role when personal (or scientific, professional, legal, financial, or other) interests or relationships could be expected to impair objectivity or expose the client to risk of harm. Similarly, Standard 3.05 (Multiple Relationships) proscribes entering into a multiple relationship if the relationship might reasonably impair objectivity, competence, or effectiveness in performing psychological duties, or otherwise risk harm to the person. No reasonable exceptions to compliance with these standards existed in this case, and several other competent neuropsychologists were readily available, including

some with much more pain experience. No indications were given that the multiple relationship was considered even potentially problematic, and the report did not indicate any potential limitations or dangers as a result of this preexisting relationship. Only in later notes was the issue of multiple relationship conflicts raised, but only regarding the provision of psychotherapy. A subsequent report indicates that NP1 reasoned that because neuropsychological assessment and consultation services were "objective", multiple roles presented no conflict.

NP1's reasoning and interpretation of ethical standards regarding multiple relationships is clearly problematic. Perhaps only in an ideal world, if one assumes that two neuropsychologists could produce exactly the same interview and test results, would we expect all neuropsychologists to reach identical conclusions. In the real world, there is frequently disagreement about even the same test results, situations are often complex, and lawsuits involving mild brain injury are frequently accompanied by widely discrepant findings and opinions. In this case, examination of the assessment findings and recommendations from NP1 strongly suggest that this experienced and competent neuropsychologist's objectivity was significantly compromised by the multiple relationships. This lack of objectivity was manifest in inadequate assessment procedures, an uncritical diagnostic approach, and uncritical judgment that compromised his findings and the welfare of the patient in several ways, especially in terms of delayed treatment and prolonged disability. Hence, in addition to Standard 3.06 (Conflict of Interest), Standard 3.04 (Avoiding Harm) seems to have been breached. The spirit of the Ethics Code was also violated in terms of Principles A (striving to promote benefit and avoid harm), B (managing conflicts of interest to avoid harm), C (promoting accuracy and truthfulness), and D (protecting justice and fairness and avoiding bias).

Further, suspicion must be raised that the failure to recommend treatment reflects not only nonobjectivity and poor judgment due to contradictory relationship role influences, but also the conflicting interests of the examiner. Chart notes indicating examiner intention to avoid a "dual relationship" by not providing psychotherapy indicates an awareness of need for this treatment. A failure to make a referral to someone with whom there would not be a dual relationship suggests the possibility that such a referral was avoided for one of more of the following reasons: (a) overemphasis on brain injury interpretations of cognitive symptoms to justify report findings and/or reach findings favorable to the familiar client's preference for organic explanations of difficulty and/or his lawsuit; (b) avoidance of exposing NP1's questionable dual relationship and reasoning regarding it; and/or (c) avoiding the possibility of losing income (both existing clinical income and anticipated more lucrative future medicolegal income) by referring to someone not hampered by a dual relationship. These likelihoods are supported by the following events: (a) when the client's insurance was changed to a network to which NP1 did not belong, a referral to another provider was made, and that provider initiated both psychological treatment and specialty pain management referrals; (b) NP1 became re-involved when he could again be reimbursed, by providing even higher paid medicolegal assessment and testimony; and, (c) NP1 asserted, at both points of his service provision with this client, that his previous relationship was advantageous (i.e., providing more pre-injury baseline information for comparison), without consideration of disadvantages (e.g., nonobjectivity from a previous non-clinical relationship or nonobjectivity from a previous clinical relationship).

NP1 failed to prevent his own personal interests from competing with those of his client. By entering into risky multiple relationships with role conflicts (3.06), he clearly compromised his professional objectivity, competence, and effectiveness (3.05), and did not take reasonable steps to avoid harm (3.04). By accepting this client a second time for medicolegal evaluation, NP1 expanded the ethical conflict resulting from

multiple roles and interests. The following potential sources of bias existed: (a) a preexisting personal relationship that included having received health care treatment services at this persons office, and even by him, which could reasonably be expected to affect objectivity in a clinical evaluation of that person; (b) the preexisting personal relationship that could reasonably be expected to affect objectivity in a medicolegal evaluation; (c) the preexisting clinical treatment relationship that could reasonably be expected to affect objectivity in a medicolegal evaluation; and (d) apparent financial interests which initially, as a clinical assessment provider, seemed to compromise patient need for treatment, and later, as a highly paid expert, ignored precautions against possibility of nonobjectivity from both previous personal and clinical relationships (the fact that the former most likely contributed to the latter is also consistent with financial interests that conflict with appropriate assessment and treatment). NP1 failed to adequately consider and safeguard against potential conflicts and negative consequences of his decisions, and hence failed to take any precautions to avoid the harm that seems to have resulted. This harm included compromised objectivity and ineffectiveness in assessment, diagnosis and treatment planning that complicated recovery and contributed to prolonged distress and disability.

Assessment

In terms of NP1's initial assessment, numerous problems are evident. Inadequate checklist versus objective measures of emotional status and pain were employed. No measures of emotional status or pain complaint veracity were administered, and on the one measure of cognitive symptom exaggeration employed, a borderline score was produced but minimized and attributed to pain and emotional distress. These problems breach requirements in Standard 9.02 (Use of Assessments) regarding both failure to use appropriate techniques with demonstrated utility, reliability and validity, and failure to indicate resulting limitations.

In terms of diagnostic opinions, the requirement in Standard 9.01 that opinions be based on sufficient information and techniques to substantiate findings was not met. Not only were insufficient measures employed, but there was then no consideration that pain, emotional status or motivation may have influenced neuropsychological test performance (e.g., Hart, Martelli, & Zasler, 2000; Hart, Wade, & Martelli, 2003; Martelli, Zasler, Nicholson, & Hart, 2001; Nicholson, 2000). The inconsistency in interpreting pain and distress as causes of a suspicious score on a very easy symptom validity test, yet not considering that they could affect performance on much harder neuropsychological measures is glaring evidence of bias. Further, pain and distress were not even considered as possible barriers to adjustment that required prompt and aggressive treatment. The failure to recommend prompt treatment to someone presumed to be in acute emotional distress and pain violates the primary bioethical principle of beneficence and nonmaleficence (APA, 2002; Beauchamp & Childress, 2001; Martelli, Zasler, & Johnson-Greene, 2001).

Case Resolution

After learning that NP1 had become re-involved as an expert with his former health care provider and then clinical patient, NP3 called NP1 to express concerns about apparent conflicts of interest and multiple relationships. With regard to accepting the subject as a clinical patient, he minimized how well he knew the subject and explained his rationale. He noted that he had consulted a colleague (a clinical psychologist/psychotherapist) who agreed that neuropsychology was objective, that having a pre-injury baseline of cognitive and personality functioning was a unique advantage, and that avoiding psychotherapy would avoid a conflict. Regarding

re-involvement for medicolegal examination, NP1 explained that the subjects attorney requested a re-evaluation and that he was only performing clinical duties.

In a subsequently scheduled meeting, NP3 delineated the ethical concerns. NP1 did not express complete agreement, and even questioned whether NP3's opinions were conflicted by his involvement as an expert "from the other side" of an adversarial court proceeding. NP1 nevertheless noted the following: (a) He did not fully critically consider, explicitly indicate, or explicitly make efforts to safeguard against potential conflicts inherent in such an examination, invited questioning of his objectivity, and did not indicate the potential conflicts or limitations in his reports; he admitted that if another neuropsychologist had conducted an evaluation under similar circumstances, he probably would have been suspicious; (b) this was the first evaluation of someone with whom he had a pre-existing relationship, and he would not perform a similar evaluation in the future, and he would be on guard more generally to issues relating to conflicted interests; (c) neuropsychologists are not always objective, and certain situations require greater scrutiny – e.g., he periodically uses the Sweet and Moulthrop (1999) self-examination questions, and these could have been used in this situation and perhaps should be used more frequently; and, (d) he would request a withdrawal from testifying in the legal case.

NP1 subsequently reported that a discussion between his business attorney and the retaining plaintiff's attorney determined that he would risk legal action if he withdrew from expected involvement in the plaintiff's case. Problem-solving discussions between NP1 and NP3 were planned. Prior to further discussion, a settlement was reached, obviating the need for decisions about NP1's continued involvement in the plaintiff's legal case.

Overattribution of post-injury problems to brain injury despite weak evidence reduced the credibility of the patient's complaints and misdirected treatment. Further, not recommending psychotherapy or pain management early after injury diluted recognition of the importance of these problems, delayed treatment and almost certainly protracted distress, complicated recovery, and prolonged disability. The small settlement that was awarded hardly seemed desirable compensation for the apparently harmful initial assessment and treatment of this patient that likely would not have happened absent the multiple conflicting relationship influences.

Conclusions and Recommendations

In the present scenario, the neuropsychologist employed poor judgment by entering into multiple relationships with multiple contradictory professional, personal and financial influences. He did not make reasonable efforts to consider the potential negative effects on his objectivity and effectiveness or the potential harm to the patient. He performed an inadequate assessment that very poorly assessed and poorly addressed the role of pain and emotional distress factors, as well as motivation, and overattributed problems to brain injury. He failed to (a) indicate any of the many potential limitations or qualify any opinions, (b) protect against compromising objectivity, (c) employ more reliable and valid instruments for pain and emotional assessment status, (d) appropriately interpret instruments, and (e) protect against the harm that eventuated.

NP1's inadequate assessment and treatment recommendations delayed appropriate treatment of pain and emotional distress symptoms, complicated recovery, and almost certainly protracted distress and disability. Whether by coincidence or subtle reinforcement, his initial involvement increased the likelihood of his seeing this patient later for lucrative work in his preferred specialty of forensic neuropsychology.

It should be considered an extremely difficult and underappreciated challenge to resist the highly reinforcing incentives associated with

lucrative medicolegal work in an otherwise increasingly restrictive reimbursement environment. Although these incentives often exert an overt influence, it may be the more subtle and less conspicuous reinforcement that is the more dangerous threat, and there is increasing evidence that bias is as prevalent in forensic examiners as is it is in personal injury claimants (e.g., Martelli, Zasler, Nicholson, Hart, & Heilbronner, 2001).

The changes to APA Ethics Code help address the problem of inappropriate examinations by tightening standards in the areas of competence, validity and objectivity, consideration and indication of limitations, attention to individual factors, protections against harm, promotion of more equitability and justice, and transparency (Adams, 2003). Clearly, more attention is needed in order to parallel the increasing prominence of forensic neuropsychology specialists and services, particularly given the high frequency of pain complaints by personal injury litigants and those seeking disability benefits. It may be incumbent upon neuropsychologists to seek out additional readings that provide strategies for protecting against these potent yet often subtle threats to objectivity (e.g., Martelli, Bush, & Zasler, 2003; Sweet & Moulthrop, 1999).

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