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Assessing the Veracity of Pain Complaints and Associated Disability

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INTRODUCTION

Evaluation of pain complaints and pain-related disabilities presents a significant diagnostic challenge. In cases of clear, severe, and/or functionally disabling physical pathology and pain, the evaluations and opinions of most healthcare practitioners are fairly consistent. In most cases, however, where physical findings are less clear, practitioners who specialize in pain and disability assessments may express widely varying opinions. Medical evidence is often problematic or disputable and the relationship between physical findings and subjective report is frequently weak. Pain, ultimately, is a subjective complaint that is difficult to verify or refute (Hall & Pritchard, 1996; Merskey, 1986). Finally, despite recent biopsychosocial and psychophysiologic advances in terms of understanding, simplistic and dichotomous conceptualizations remain overly represented (Martelli, 2000).

Traditionally, pain and pain-related disability evaluations have been conducted by such specialists as physiatrists, orthopedists, neurologists, neurosurgeons, psychiatrists, psychologists, and neuropsychologists. Further, diagnosis and treatment are the presumed reasons for assessment, and the estimation of frank malingering, as well as significant exaggeration of symptoms, is generally estimated to be less than 20% (Martelli, Zasler, Mancini, & MacMillan, 1999). A recent review by Fishbain, Cutler, Rosomoff, and Rosomoff (1999) indicates that malingering might be present in between 1 and 10% of chronic

pain patients, but there are significant problems with the reliability of any such estimates and an absence of reliable methodologies for detecting malingering in chronic pain patients. However, it should be noted that pain evaluation referrals also frequently involve such contexts as health-care insurance policy coverage, disability insurance policy application, social security disability application, personal injury litigation, worker's compensation claims, functional capacity evaluations, and determination of capacity for work. In these medicolegal situations, the incidence of symptom exaggeration and malingering may be significantly higher (Rohling & Binder, 1995; Binder & Rohling, 1996).

Importantly, the evaluation of pain complaint and pain-associated functional impairment and disability may be one of the more confounded and misunderstood areas of healthcare-related work. The task of making determinations regarding pain severity, impact, and related functional impairment and disability is fraught with potential obstacles and pitfalls. This is, of course, due to the poorly understood and complex nature of the deficits involved, as well as the lack of formal, scientifically validated conceptual models and "rating systems." Disentangling the multiple contributors to subjective pain experience and associated functional disability requires careful scrutiny.

Chronic pain patients may present with some response bias to report pain or related disability. In the present chapter, response bias is defined as a class of behaviors that reflects less than fully truthful, accurate, or valid

symptom report and presentation. Importantly, response bias is a ubiquitous phenomenon affecting almost any domain of human self-report. However, in the context of medicolegal or insurance presentations, with which this chapter is primarily concerned, the prevalence or importance of such bias becomes more acute (Rohling & Binder, 1995; Binder & Rohling, 1996; Youngjohn, Burrows, & Erdal, 1995). Given frequent financial and other incentives to distort symptoms and performance during examinations conducted in medicolegal or insurance contexts (i.e., healthcare policy coverage, disability insurance, social security disability, personal injury litigation, worker's compensation claims, functional capacity, and work evaluations), assessment of examinee veracity and motivation to provide accurate and full effort during assessment becomes critical. The importance of detecting response biases or invalid symptoms is crucial with regard to increasing the likelihood of diagnostic accuracy. Accurate diagnosis is prerequisite to provision of appropriate and timely treatment and prevention of iatrogenic impairment and disability reinforcement; it is also critical to appropriate legal compensation decisions.

Medicolegal and insurance assessments, with their significant consequences of determination decisions regarding impairment and disability, require the following in order to ensure validity: (a) an accurate assessment of possible response bias to report pain or related disability; (b) an accurate assessment of attribution, i.e., whether pain and related problems are correctly being attributed to the compensable cause; and (c) sensitivity, specificity, utility, and ecological validity of assessment measures.

Blau (1984; 1992) has expounded on the importance of determining response biases and measuring true levels of impairment in medicolegal situations. Essentially, in this arena, an alleged victim of a wrongful act or omission attempts to establish (a) causality in order to demonstrate entitlement to compensation for damages, which is awarded based on (b) level of damages suffered. In cases of less obvious, clear-cut, and significant trauma with psychologic, neurologic, or soft tissue damage, causality and level of current and future damages are more difficult to prove and expert evaluation and opinion are heavily relied upon for making legal determinations. In the parallel insurance situation, the insured attempts to access entitlements to healthcare treatment and disability benefits, and expert evaluation and opinion are relied upon for making policy determinations. In both cases, financial and other incentives clearly represent motivational factors that increase the likelihood of response bias in the form of exaggerated or feigned symptoms.

EXAMINING PATIENT RESPONSE BIASES

Examinee response bias, or predilections toward less than fully valid, accurate, and effortful behavioral

responses, can take several forms. Bias ranges from interpretations of symptoms that may be minimized to exaggerated or feigned, and that may be accurately or inaccurately attributed to different events. For instance, pre-existing symptoms may suddenly be attributed to an accident, symptoms previously not noticed suddenly may be given such vigilant attention that attention and anxiety alone produce significant increases, or an accident may cause an aging person to do a self-inventory of health that reveals symptoms due to aging that were present but previously minimized. Further, a heightened awareness or vigilance effect can lead to focusing and sensitization to problems which would otherwise be innocuous. In addition, environmental realities also exert influence over response to injury and symptoms. For example, the vastly different consequences for diagnoses of cancer vs. mild traumatic brain injury or back injury produce differential reinforcement; the former is clearly undesirable and negative, while the latter can result in highly desirable monetary compensation.

Martelli et al. (1999) reviewed the literature and found that the following injury context variables were associated with poorer post-injury adaptation and recovery, and increased likelihood of response bias.

1. Anger or resentment or perceived mistreatment
2. Fear of failure or rejection (e.g., damaged goods; fear of being fired after injury)
3. Loss of self-confidence and self-efficacy associated with residual impairments
4. External (health, pain) locus of control
5. Irrational fear of injury extension, reinjury, or pain
6. Discrepancies between personality/coping style and injury consequences (e.g., very physically active person with few intellectual resources who has a back injury)
7. Insufficient residual coping resources and skills
8. Prolonged inactivity resulting in disuse atrophy
9. Fear of losing disability status, benefits, and safety net
10. Perceptions of high compensability for injury
11. Preinjury job (task, work environment) dissatisfaction
12. Collateral (especially if "silent")
13. Inadequate and/or inaccurate medical information
14. Misdiagnosis, late diagnosis, or delays in instituting treatment
15. Insurance resistance to authorizing treatment or delays in paying bills
16. Retention of an attorney
17. Greater reinforcement for "illness" vs. "wellness" behavior
18. Dichotomous (organic vs. psychologic) conceptualizations of injury and symptoms

These variables represent vulnerability factors that can reduce effective coping with post-injury impairments and increase the likelihood of maladaptive coping and response bias.

Importantly, these variables are not mutually exclusive, and, as with the variables presented below, more than one can contribute to symptom report and presentation

Additional review of the literature, balanced with clinical experience, identifies the following significant sources of response bias that can be seen during examinations (Martelli, 2000).

1. **Cultural Differences.** For example, many non-Western cultures mix emotional and physical pain and symptoms at a conceptual and phenomenological level. Also, some cultures see failure to impose severe penalty/extract significant compensation for harm as a sign of weakness and disgrace in God's eyes.
2. **Reactive Adversarial Malingering (RAM)** based on fear, mistrust of the opposing side's honesty, or mistreatment (e.g., from assumed "facts" in many work settings and cultures, including plaintiff attorney groups) resulting in a deliberate pendulum-like overplaying of symptoms. This may be especially characteristic in persons/groups with tendencies toward suspiciousness, including immigrants, outsiders, or those who feel chronically underprivileged.
3. **Conditioned Avoidance Pain-Related Disability (CAPRD),** or, roughly, phobic or extreme anxiety reactions wherein activity (mental or physical) is associated with anticipation of an exacerbation of pain, with such stress possibly resulting in an actual exacerbation. Kinesiophobia and cogniphobia are two types.
4. **Desperation Induced Malingering (DIM) or Desperation Induced Symptom Exaggeration (DISE),** e.g., insecure immigrant workers, aging workers, tired workers, workers insecure about work changes, immigrants who tried introjection and feel resentful that they were not rewarded, persons who recently climbed back on the horse only to get knocked off again without belief they can climb back in the saddle one more time, workers fearing their own limited or declining abilities, real or imagined abuse from employers, family, etc., immigrants who feel rejected by the culture and feel entitled, immigrants who feel disillusioned because the new land was not everything they had hoped — i.e., those who believe this to be a viable solution to a desperate situation. Probably also included are those making desperate pleas for

help and who, upon confronting tests that seem different and maybe easier than the real-life situations where they have problems, reduce effort to highlight their problems.

5. **Sociopathic, Manipulative, and Opportunistic (SMO) types.** These rather self-explanatory styles can be found in all groups.
6. **Passive Aggressive or Impatient or Rebellious types,** representative of those who resent others not listening to them and believing them at face value, and resent imposed evaluations or doctor's visits, especially ones that examine psychological function or motivation. They may play games with doctors by withholding or undermining procedures or treatments, and may especially alter performance or play games on tests that seem nonchallenging or unrelated to real life situations.
7. **Psychologically Decompensated types, i.e.,** the extremely dysfunctional patient who is usually easily recognizable.
8. **Those who don't take our examinations or tests as seriously as we do.** The authors have some very limited but relevant survey data suggesting that plaintiffs may take our examinations a little more seriously than defendants. In contrast, weathermen seem to be taken more seriously than independent examiners by persons who have not graduated high school, while independent examiners seem to fare better with those who have been to college.

Importantly, a too often overlooked form of bias is one that is iatrogenic to the nature of the insurance and adversarial legal system. In an effort to elucidate expectancy influences and bias for persons sustaining injuries, Martelli, Zasler, and Grayson (2000) collected survey attitudinal data from professionals who work with injured Worker's Compensation (WC) patients. A summary of the preliminary data is offered in Table 63.1, broken down by the three sample groups: (1) disability evaluators, comprised of physicians, chiropractors, physical therapists, and vocational evaluators; (2) staff from a rehabilitation neuropsychology service; (3) attendees at a case management conference, including over 50% WC case managers. These data are quite compelling. Overall, approximately 25% of WC patients are believed to be exaggerating or malingering, with higher rates evidenced by WC case managers. This suggests a general skepticism and distrust faced by injured persons during evaluations. In contrast, the majority of professionals filling out the survey believed they would be treated unfairly by the WC system if they were injured, suggesting a general skepticism and distrust of the extant systems that fund evaluation and treatment of injury and disability.

TABLE 63.1
Survey of Attitudes Regarding Worker's Compensation (WC)

Question	Respondent Sample (%)		
	Disability Evaluating Professionals (N = 17)	Medical Psychology Service Staff (N = 7)	Case Managers (N = 16) (including 7 WC employees)
% Injured workers who fake/exaggerate/malingering	19.2	24.7	28.5
% Injured workers that WC insurance treats < fairly	49.2	62.5	23.2
% Employers who treat injured workers < fairly	53.5	41.2	32.7
Likelihood your employer would treat you < fairly	43.75	54.2	46.4
Likelihood WC would treat you if injured < fairly	60.0	65.9	48.9

Despite the preliminary nature of these data derived from small samples and the fact that generalizability across situations cannot be assumed, they nonetheless seem compatible with the levels of diffuse distrust observed by the authors in medicolegal situations. These data highlight the importance of considering the much different set of motivational factors that operate on examinees that present to independent evaluation. In addition, they strongly argue for deliberate and thorough preparation of examinees prior to the examination.

In an interesting theory about a major type of response bias in chronically disabled workers, Matheson (1988; 1990; 1991a; 1991b) conceptualized a "symptom magnification syndrome" based on a careful analysis of injured industrial workers. He defined this syndrome as a conscious or unconscious self-destructive (e.g., blocks return to productive activity) and socially reinforced pattern of symptoms, which are intended to control life circumstances of the sufferer, but which impede health-care efforts. He further defines three major subtypes, and provides classification guidelines for evaluation via observation during performance of simulated work tasks completed within functional capacity evaluations. The Type I "refugee" is defined as displaying illness behavior that provides escape or avoidance of life situations perceived as unsolvable. Somatization, conversion, psychogenic pain, and hypochondriacal disorders are conceptualized as extreme subcategories for this type. The Type II "game player" employs symptoms for positive gain. Although this type seems associated with the psychiatric diagnosis of malingering, Matheson argues that true malingering is a medicolegal concept, while Type II symptom magnifying is a treatable self-destructive syndrome. The Type III "identified patient" is motivated by maintenance of the patient role as a means of life survival. Associated psychiatric diagnoses include factitious disorder (May, 1999).

Main and Spanswick (1995) also examined simulated or exaggerated incapacity in persons claiming physical disability. They identified a list of features associated with simulated or exaggerated incapacity. Features identified

as primarily suggestive of simulated or exaggerated incapacity included

1. Failure to comply with reasonable treatment
2. Report of severe pain with no associated psychological effects
3. Marked inconsistencies in effects of pain on general activities
4. Poor work record; history of persistent appeals against awards
5. Previous litigation

Features identified as not primarily suggestive included

1. Mismatch between physical findings and reported symptoms
2. Report of severe or continuous pain
3. Anger
4. Poor response to treatment
5. Behavioral signs/symptoms

A brief review of important sources of bias, or threats to objectivity, which require assessment during evaluation of physical, sensory, and neurocognitive impairments follows.

ATTRIBUTION AND BIAS

Examinee attribution bias can confound accurate diagnosis. Examples include mistaking clinical entities like depression or sleep disturbance and concomitant physical, memory, or motivational problems for physical injury and pain-related sequelae. This can occur due to misattribution or over-attribution or retrospective attribution, or illusory correlation, or heightened awareness due to vigilance biases. Importantly, conditions like depression and sleep disturbance are reversible and may have even been present pre-injury without producing significant limitations. Furthermore, these factors may be interacting with true physical injury symptoms to increase distress, prolong impairment, and interfere with recovery. Finally, such factors as

presence of vigilance to symptoms can increase physiologic arousal and lead to increased symptomatology and perceptions of impairment, in a vicious cycle.

Examiner misattribution can similarly occur. Only methodical medical and psychological assessment can differentiate sequelae secondary to, for example, brain injury from chronic pain. Tendencies toward over-diagnosis of brain injury-related disorders by brain injury specialists given abnormal neurocognitive findings and/or nonspecific somatic complaints not exclusively pathognomonic of brain injury can be avoided only through careful differential diagnosis. Brain injury specialists sensitized to neurologic symptoms have been observed by the authors to misdiagnose chronic pain sequelae as post-concussive symptoms, which may result in iatrogenic impairment associated with an escalation of medical costs, prolongation of inappropriate treatment, and eventual failure that produces helplessness and chronic disability (e.g., nonresolving post-concussive disorders) and misperceptions in the injured person. Conversely, similar observations have been made for psychiatrists and psychologists prone to infer psychiatric etiologies for all pathology, including pain and physical injury sequelae (Main & Spanswick, 1995; Martelli, Zasler, & Grayson, 1999a).

THE RESPONSE BIAS CONTINUUM

As indicated above, for the purposes of this chapter, response bias is defined as any behavioral predisposition involving less than fully truthful, accurate, and valid symptom report and presentation. This includes less than fully effortful behavioral responses displayed on formal and informal interview and examination procedures. Formal assessment of response bias, which is frequently lacking or only haphazardly attended to in most clinical examinations, represents the only assurance that clinical exam findings are accurate and valid reflections of pain severity, frequency, and functional impairment levels. Response bias appears best conceptualized on a continuum that extends from

1. Denial and unawareness of impairments,
2. Symptom minimization,
3. Symptom magnification/exaggeration to
4. Frank malingering.

Denial and unawareness refer to either psychologic or organic phenomenon wherein impairments are underappreciated due to dysfunction of brain operations subserving awareness or psychological repression to guard against distressful realizations. Symptom minimization is a related, but more consciously motivated desire, and usually involves an attempt to minimize the impact of undesirable functional restrictions or distress (Martelli, et al., 1999a).

Symptom magnification, in contrast, refers to exaggeration of impairment and can occur in relation to multiple factors and serves a wide range of psychological needs (e.g., efforts to legitimize latent dependency needs, resolve pre-existing life conflicts, retaliate against employer or spouse or other, reduce anxiety, exert a "plea for help," or solicit acknowledgment of perceived difficulties). Symptom exaggeration always promotes passivity and helplessness and an external locus of control and is a significant impediment to rehabilitation (Martelli, Zasler, & Grayson, 1999b).

Symptom exaggeration also can occur in patients with premonitory histories of psychologic problems who "latch on" to a specific diagnosis that not only becomes responsible for all life problems, but also promotes passivity and helplessness and an external locus of control. When patients are assessed for claims of major disability following uncomplicated mild whiplash or soft tissue injuries, nonorganic contributors should be closely scrutinized. Depression, post-traumatic stress disorders, anxiety conditions, and other psychiatric syndromes generally have a favorable psychological and functional prognosis given timely and appropriate assessment and treatment. Misdiagnosis of these conditions serves to promulgate misperceptions and amplify functional disability and healthcare costs.

Malingering, as defined in the DSM-IV (p. 683) (American Psychiatric Association, 1994) is "...the intentional production of false or grossly exaggerated physical or psychological symptoms, motivated by external incentives such as avoiding military duty, avoiding work, obtaining financial compensation, evading criminal prosecution, or obtaining drugs." Malingering should be suspected if any combination of the following is noted:

1. Medicolegal context of presentation
2. Marked discrepancies between claimed stress or disability and objective findings
3. The presence of Antisocial Personality Disorder

Therefore, measures of malingering, or deliberate symptom production for purposes of secondary gain, should always be administered in cases of medicolegal presentation, suspicion of any disincentive to exhibit full effort, or suspicion of sociopathic personality disorders.

According to Lipman's typology (1962), malingering can be categorized into four categories: (1) fabrication of nonexistent symptoms; (2) exaggeration of symptoms that are presented as worse than in actuality; (3) extension of symptoms that have actually improved or resolved; and (4) misattribution or fraudulent attribution of symptoms to an injury when they actually preceded, postdated, or are otherwise unrelated. Notably, exaggeration is considered much more frequent than fabrication, while more than one category can occur in a single person. Finally,

as Miller (2001) notes, different combinations of types can occur in persons with more than one problem (e.g., chronic pain, PTSD, post-concussion syndrome), which can further co-occur with other psychological syndromes (e.g., somatoform and personality disorders).

IDENTIFYING RESPONSE BIAS

The difficulty with defining pain, actual accentuation of the pain response (e.g., hyperpathia) seen in some chronic pain problems, response bias to reported pain, and its possible deception, makes the assessment of pain-related complaints extremely challenging. Pain, defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage (Merskey, 1986), is a complex multidimensional subjective experience mediated by emotion, attitude, and perception. Unlike other modalities, it is not possible to devise simple signal detection paradigms for the evaluation of response bias given that this is a subjective experience with no clear objective referents, especially in the case of chronic pain associated with actual injury and nociception or abnormal function of the nervous system. Clearly, multiple variables may impact on pain reporting and behavior. For example, arousal, stress, tension, and anger all may exacerbate subjective reporting of pain and pain behavior, as may depression, through effect on physiologic function. Psychoemotional and psychosocial concomitants of chronic pain must also be appreciated, including loss of self-esteem, lowered frustration tolerance, depression, sexual dysfunction and decreased libido, and anger and guilt. Further, situational factors make additional contributions to pain-related complaints. The context of an exam, however, typically requires that psychological and physical pain factors be addressed individually, despite the fact that these components are typically inextricably intertwined with one another, as well as such affective conditions as depression and anxiety.

Physicians should be familiar with exam strategies designed to evaluate disorders with (a) probable "functional" components, or symptoms that seem more strongly associated with psychosocial vs. structural factors, as well as (b) feigned symptoms, including bedside exam techniques for physical and cognitive "malingering." Examples include such strategies as Hoover's test for evaluation of malingered lower extremity weakness, sideways/backward walking for assessment of feigned gait disturbance, and a positive Stenger's test on audiologic assessment for nonorganic hearing loss. Other tests that might be of value in the context of response bias detection on the physical examination include Mankopf's maneuver, strength reflex test, arm and/or wrist drop test, hip adductor test, axial loading test, Gordon-Welberry toe test, Bowlus and Carrier test, Burns bench test, Magnuson's test, among others [(Babitsky, Brigham, & Mangraviti, 2000); see also Table 63.3 for a relatively comprehensive listing].

Some major exam findings that are inconsistent with structural lesions include patchy sensory loss, pain in a nondermatomal distribution, nonpronator drift, and/or astasia-abasia. Motor and other impairment inconsistencies that fluctuate or disappear under hypnosis, drug-assisted interviews or "presumed" nonobservation may certainly increase the index of suspicion regarding non-organicity, although exceptions to this rule do exist. Faked hemiparesis is typically more common on the left side, perhaps due to the fact that most persons are right-hand dominant. Consistency regarding laterality of symptoms, particularly with referred pain and/or neurologic impairment, should be evaluated.

Clinicians evaluating chronic pain must be familiar with psychosocial syndromes that may present as pain, including

1. *Factitious disorder*, or the intentional production or feigning of physical symptoms, or exaggerated expression of physical conditions in order to adopt a sick role
2. *Somatoform disorders*, characterized by preoccupation with physical symptoms and pain that exceeds possible organic pathology
3. *Hypochondriasis*, or preoccupation with pain as part of a conviction that it is a part of a pernicious disease process
4. *Conversion disorder*, or the expression of frank psychiatric disorder via some symbolic transformation

Clinicians should also be familiar with symptoms related to pain imperception. Pain complaints should be assessed, in part, when of CNS origin as opposed to psychogenic, by concurrently assessing temperature perception, given that the same neural pathways mediate these sensations. When temperature sensation is preserved in the presence of a loss of pain sensation, after either brain or spinal cord injury, the deficit is not likely to be organic (the loss should occur contralateral to and below the level of the lesion). This point also belabors the fact of understanding the neuropathology/pathology of the lesion based on imaging studies and the implications that these findings have for anticipated clinical exam findings. Alleged pain imperception can be evaluated, as can any impairment for that matter, with appropriately designed forced choice testing. Additionally, examiners should realize that alleged pain imperception or loss of sensation is difficult to fake upon repeated bilateral stimulation. This is due to the fact that examinees who exaggerate rely on subjective strategies rather than truly responding to the strength of the stimuli. Therefore, assessments such as Von Frey hairs could be utilized in the aforementioned

scenario to provide further objective evidence of feigned sensory deficits.

It is worth emphasizing that the presence of structural inconsistencies, a nonorganic syndrome and/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. Unfortunately, the science and art of methodic differential diagnosis are too often underappreciated in the evaluation process (Martelli, et al., 2000).

A relevant screening procedure frequently used by physical therapists, doctors, and chiropractors for estimating when psychological factors are significantly influencing pain-related responses is the assessment for Waddell's Nonorganic signs (Waddell & Main, 1984; Waddell, Main, Morris, Paolao, & Gray, 1984; Waddell, 1999). These are listed below:

Screening for Nonorganic Response Bias: Waddell Signs

1. Overreaction
Guarding/limping, bracing, rubbing affected area, grimacing, sighing.
 2. Tenderness
Widespread sensitivity to light touch of superficial tissue.
 3. Axial loading
Light pressure to skull of standing patient should not significantly increase low back symptoms.
 4. Rotation
Back pain is reported when shoulders and pelvis are passively rotated in the same plane.
 5. Straight leg raising
Marked difference between leg raising in the supine and seated position.
 6. Motor and sensory
Giving way or cog wheeling to motor testing or regional sensory loss in a stocking or nondermatomal distribution (rule out peripheral nerve dysfunction).
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Additional nonorganic signs include lower extremity giving way, no pain-free spells in the past year, intolerance of treatments; and emergency admissions to hospital with back trouble.

Importantly, the presence of Waddell's or other nonorganic signs does not exclude physical components as the cause of low back pain. Rather, they suggest only that psychological factors appear to be influencing the patient's responses and behavior. Notably, physical and psychological findings are not mutually exclusive, and psychological factors may more often be a result of low back pain than a cause (Simmonds, Kumar, & Lechelt, 1998). Further, recent reports strongly demonstrate the relationship between high levels of anxiety and nonorganic responses during physical exams in chronic low back pain (Hadjistauroopoulos & LaChapelle, 2000). As such, the importance of minimizing anxiety-related

response bias during exams should, therefore, be emphasized with regard to optimizing accurate performance and assessment. Clinicians should familiarize themselves with the wide variety of simple yet effective anxiety management interventions.

With regard to assessment of psychological/psychiatric, somatic, and neuropsychological impairments, including chronic pain, response bias represents an especially important threat to validity. Because these assessments usually begin with an interview about self-reported symptoms and subsequently rely heavily on standardized measures of performance on tests which are variably normed, their validity requires the veracity, cooperation, and motivation of the patient for obtaining valid performance measures. Recent evidence, however, strongly suggests that patients seen for presumptive injury-related impairments over-report preinjury functional status (Lees-Haley, Williams, Zasler, Margulies, English, & Steven, 1997). This is especially true with post-concussive and pain related deficits because these symptoms often appear with similar frequency in the general population (Lees-Haley & Brown, 1993). In addition, the demonstrated ability of physicians and psychologists to accurately detect malingering in examinations and test protocols has been less than impressive (Hall & Pritchard, 1996; Loring, 1995). Nonetheless, various instruments, techniques, and strategies are available that have demonstrated at least some utility in detecting response bias, especially malingering, as a means of increasing confidence in appropriate motivation during examination, and hence the validity of assessment findings.

In Table 63.2, a general summary of hallmark and selected signs of response bias are presented. The signs are predicated on examination of inconsistencies and can certainly be applied to most aspects of comprehensive medical and psychological evaluations for chronic pain.

In Table 63.3, a summary of some very specific response bias detection measures and strategies, along with guidelines, is presented in an integrated format. Importantly, these strategies are presented as illustrations of indicators of important information for interpreting examination findings and test data within a larger context of multidisciplinary evaluation for chronic pain. This approach integrates contextual information, history, behavioral observation, interview data, collaborative data, and personality data with measures of effort or performance (or symptom exaggeration or malingering) and physical examination, medical and neuropsychological examination performance data. This approach potentially offers increased reliability with regard to estimating the degree to which an examinee is responding truthfully and exerting full effort or withholding or distorting effort or performance, and the degree to which specific and general test results from multiple assessment areas are reliable and valid and reflect true abilities.

TABLE 63.2
Response Bias: Hallmark Signs

1. Inconsistencies within and between the following (given absence of significant psychiatric, attentional, comprehension, or other disorders where inconsistencies are not uncommon):
 - a. Reported symptoms
 - b. Examination/test performance
 - c. Clinical presentation
 - d. Known diagnostic patterns
 - e. Observed behavior (in another setting)
 - f. Reported symptoms and exam/test performance
 - g. Measures of similar abilities
 - h. Similar tasks or tasks within the same exam or test (especially when difficult tasks are performed more easily than easy ones)
 - i. Different examination sessions
2. Grossly impaired performance and extreme complaints
 - a. Poorer performance and more extreme complaints vs. established expectancies or normative data for similar injury/illness
 - b. Very poor performance on easy tasks (especially when presented as difficult)
 - c. Failing tasks that all but severely impaired perform easily
3. Lack of specific diagnostic signs of impairment
4. Specific signs of exaggeration/dissimulation/malingering on psychological testing
 - a. Minnesota Multiphasic Personality Inventory (MMPI/MMPI-2) Original and additional validity scales: L, F, Fb, Fp, Ds, K, VRIN, TRIN, F-K, Fake Bad, etc.
 - b. Personality Assessment Inventory (PAI) Validity scales (inconsistency, infrequency, positive and negative impression management) and 8 malingering and 6 suspected malingering patterns
 - c. Pain Assessment Battery (PAB): Symptom magnification, extreme beliefs frequency and other "validity" indicators
 - d. Millon Behavioral Health Inventory (MBHI) validity scales (3)
 - e. Hendler (i.e., Mensana Clinic) Back Pain Test: scores of 21–31 (exaggerating)
 - f. Cognitive malingering detection tests (e.g., memorization of 15 items test, Digit Recognition Tests, Computerized Assessment of Response Bias, Word Memory Tests, Word Memory Test, Word Completion Memory Test, etc.).
5. Interview evidence
 - a. Nonorganic temporal relationship of symptoms to injury
 - b. Nonorganic symptoms, or symptoms that are improbable, absurd, overly specific or of unusual frequency or severity (e.g., triple vision)
 - c. Disparate examinee history/complaints across interview or examiners
 - d. Disparate corroboratory interview data vs. examinee report
6. Physical exam findings
 - a. Nonorganic sensory findings
 - b. Nonorganic motor findings
 - c. Pseudoneurologic findings in the absence of anticipated associated pathologic findings
 - d. Inconsistent exam findings
 - e. Failure on physical exam procedures designed to specifically assess malingering

Empirical support exists indicating that each of these indicators has some utility in detecting dissimulation or suboptimal effort.* Examining the pitfalls and limitations of each of these procedures, both conceptual and methodological, is well beyond the scope of this chapter. However, increasing evidence exists for improved discrimination and increased reliability when multiple measures are employed. The conceptual approach offered by the proposed Motivation Assessment Profiling (MAP) is one where behavioral observation, interview, collaborative, historical, personality, and contextual data with neuropsychological and medical performance data and measures of effort or performance (or response bias) are integrated

as an optimal method for estimating the degree of effort or performance and the degree to which test results are reliable and valid and reflect actual abilities. Notably, in evaluation of response bias and malingering, as in evaluation of pre- and post-injury status, the following investigative tools may be used in conjunction with interviews and examination and testing: (1) school records; (2) medical records; (3) driver records; (4) service and criminal records; (5) employment records; (6) psychological/psychiatric records and reports; (7) interviews with family members, friends, teachers, and employers, etc.; (8) any other available materials (e.g., from attorneys through formal discovery).

Importantly, the strategies and guidelines offered in Table 63.3 are presented as important indicators for

* See starred references.

TABLE 63.3
Response Bias Detection Measures and Strategies

Pain Assessment Measures with Built-In Response Bias Indicators	
Pain Assessment Battery (PAB), Research Edition	Symptom Magnification Frequency (SMF) > 40%
Proposed clinical hypothesis procedure evaluating	Extreme Beliefs Frequency (EBF) > 35%
	Four other "validity" indicators (i.e., alienation, rating percent of max, % extreme ratings (2 scales))
Millon Behavioral Health Inventory (MBHI)	Elevations on 3-item validity scale
Hendler (i.e., Mensana Clinic) Back Pain Test	Scores of 21–31 (Exaggerating)
	Scores > 31 (Primary psychological influence)
Medical Indicators	
Hoover's test	Test for malingered lower extremity weakness associated with normal crossed extensor response
Asasia-abasia	"Drunken type" gait with near-falls but no actual falls to ground
Nonorganic sensory loss	Patchy sensory loss, midline sensory loss, large scotoma in visual field, tunnel vision
Nonorganic upper extremity drift	Long tract involvement results in pronator type drift; proximal shoulder girdle weakness and malingering typically present with downward drift while in supination
Stenger's Test	Test for malingered hearing loss during audiologic evaluation
Gait discrepancies when observed vs. not observed	If organic, should be consistent regardless of whether observed or not
Gait discrepancies relative to direction of requested ambulation	Gait for a patient with hemiparesis should present similarly in all directions; malingers do not as a rule practice a feigned gait in all directions
Forearm pronation, hand clasping, and forearm supination test for digit/finger sensory loss	Malingered finger sensory loss is difficult to maintain in this perceptually confusing, intertwined hand/finger position
Pain vs. temperature discrepancies	Because both sensory modalities run in the spinothalamic tract, they should be found to be commensurately impaired contralateral to the side of the CNS lesion
Lack of atrophy in a chronically paretic/paralytic limb	Lack of atrophy in a paralyzed/paretic limb suggests the limb is being used or is getting regular electrical stimulation to maintain mass
Impairment diminishes under influence of sodium amytal, hypnosis or lack of observation	All these observations are most consistent with nonorganic presentations including consideration of malingering or conversion disorder
Incongruence between neuroanatomical imaging and neurologic examination	Lack of any static imaging findings on brain CT or MRI in the presence of a dense motor or sensory deficit suggests nonorganicity
Arm drop test	An aware patient malingering profound alteration in consciousness or significant arm paresis will not let his own hand, when held over his head, drop onto his face
Presence of ipsilateral findings when implied neuroanatomy would dictate contralateral findings	An examinee claiming severe right-brain damage who claims right-eye blindness and right-sided weakness and sensory loss
Tell me "when I'm not touching" responses	An examinee with claimed sensory loss who endorses that he does not feel you touch him when you ask him to tell you "if you do not feel this."
Lack of shoe wear in presence of gait disturbance	An examinee with claimed longer term gait deviation due to orthopedic or neurologic causes should demonstrate commensurate wear on shoes (if worn with any frequency)
Calluses on hands in "totally disabled" examinee	An examinee who is unable to work should not present with signs of ongoing evidence of physical labor
Assistive device "wear-and-tear" signs	In any examinee using assistive devices for any period of time, e.g., cane, crutches, there should be commensurate wear on the device consistent with the claimed impairment and disability
Mankopf's maneuver	Increase in heart rate commensurate with nociceptive stimulation during exam (some controversy exists on whether this always occurs)
Lack of atrophy in a limb that is claimed to be significantly impaired	If side-to-side measurements and/or inspection do not bear out atrophy consider other causes aside from one being claimed

continued

TABLE 63.3 (CONTINUED)
Response Bias Detection Measures and Strategies

Sudden motor give-away or ratchitiness on manual strength testing	Considered to normally be a sign of incomplete effort or symptom exaggeration
Weakness on manual muscle testing without commensurate asymmetry of DTRs or muscle bulk	Suggests simulated muscle weakness if longstanding
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	Have examinee point to area several times over period of examination; inconsistencies suggest increased potential for nonorganicity
Delayed response sign	Pain reaction temporally delayed relative to application of perceived nociceptive stimulus
Wrist drop test	In an examinee with claimed wrist extensor loss, have him pronate forearm, extend elbow, and flex shoulder ... if upon making a fist in this position he also extends wrist, then nonorganicity should be suspected
Object drop test	Examinee claims inability to bend down yet does so to pick up a light object "inadvertently" dropped by examiner
Hip adductor test	Test for claimed paralysis of lower extremity, similar to Hoover's test yet looks for crossed adductor response
Disparity between tested range of motion (ROM) and observed range of motion of any joint	When ROM under testing is significantly disparate (e.g., less) from observed, spontaneous ROM, suspect functional contributors
Straight leg raise (SLR) disparities dependent on examinee positioning	Differences in SLR between sitting, standing, and/or bending may suggest a functional overlay to low back complaints
Grip strength testing via dynamometer	Three repetitions at any given setting should not vary more than 20% and/or bell-shaped curve should be generated if all 5 positions are tested
Sensory "flip" test	Sensory findings should be the same if testing upper extremity in supination or pronation or lower extremity in internal vs. external rotation, differences may suggest a functional overlay
Pinch test for low back pain	Pinching the lumbar fat pad should not reproduce pain due to axial structure involvement; if test is positive, suspect a functional overlay

Personality Instruments with Built-In Response Bias Designs

Personality Assessment Inventory (PAI)	Inconsistency (INC), Infrequency (INF), Positive Impression Management (PIM), and Negative Impression Management (NIM) scales. 8 score patterns thought to comprise a Malingering Index (Morey, 1996). > 2 patterns malingering suspected > 4 patterns likely malingering
Minnesota Multiphasic Personality Inventory (MMPI-2)	Validity indices (L, F, Fb, Fp, Ds, K, VRIN, TRIN), F-K (Gough, 1954) The Fake Bad Scale (Lees-Haley, 1991) Compare subtle to obvious items Rogers et al. (1994) – cutoff scores: Liberal: <ol style="list-style-type: none"> 1. F-scale raw score > 23 2. F-scale T-score > 81 3. F-K index > 10 4. Obvious – subtle score > 83 Conservative: <ol style="list-style-type: none"> 1. F-scale raw > 30 2. F-K index > 25 3. Obvious – subtle score > 190

Qualitative Variables in Assessing Response Bias

Time/response latency comparisons across similar tasks	Inconsistencies across tasks
Performance on easy tasks presented as hard	Low scores or unusual errors
Remote memory report	Difficulties, especially if less than recent memory, or severely impaired in absence of gross amnesia

TABLE 63.3 (CONTINUED)
Response Bias Detection Measures and Strategies

Personal information	Very poor personal information in absence of gross amnesia
Comparison between test performance and behavioral observations	Discrepancies
Inconsistencies in history and/or complaints, performance	Inconsistencies across time, setting, interviewer, etc.
Comparisons for inconsistencies within testing session (quantitative and qualitative)	A. Within tasks (e.g., easy vs. hard items) B. Between tasks (e.g., easy vs. hard) C. Across repetitions of same/parallel tasks (rule out fatigue) D. Across similar tasks under different motivational sets
Comparisons across testing sessions (qualitative, quantitative)	Poorer/inconsistent performance on re-testing
Symptom self-report: complaints	High frequency, severity of complaints and higher frequency, severity vs. significant other report or other collaborative report
Main & Spanswick, 1995	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 persistent appeals against awards Previous litigation
Symptom self-report: early/acute vs. late/chronic symptom complaint	Early symptoms reported late or acute symptoms reported as chronic
Response to typically helpful pain interventions	Failure to show any pain relief to at least one of the following: biofeedback, hypnosis, mild analgesics, psychotherapy, relaxation exercises, heat and ice, mild exercise Failure to show any pain relief in response to TENS
Assessment of Cognitive Effort:	
Performance Patterns on Existing Psychological/Neuropsychological Tests	
Full scale IQ	Low (vs. expected, estimated, etc.)
Arithmetic and orientation scale performance	"Near-miss" (Ganser errors)
WMS-R Malinger Index: Attention/Concentration Index vs. Memory Index	Attention-concentration index score < general memory index (AC-GMI)
Grip strength	Unusually low w/o gross motor deficit
Recognition memory (California Verbal Learning Test (CVLT))	< 13
Rey Complex Figure and Recognition Trial	Atypical recognition errors (> = 2); recognition failure errors
Word Stem Priming Task Performance	Poor or unusual performance
Specific Cognitive Effort/Response Bias Measures	
Word Memory Test (WMT)	< 50% chance responding
Test of Memory Malinger (TOMM)	< 50% chance level responding
Dot Counting Test (DCT)	Correct/incorrect responses
Computer Assessment of Response Bias (CARB)	< 89% raises suspicion
Rey Memory for 15 Items Test (MFIT)	Lezak (1983), < 3 complete sets, < 9 items
Symptom validity testing (SVT)	< 50% chance level responding

interpreting examinee data. Integration of contextual information, history, behavioral observation, interview data, collaborative data, personality data, with measures of effort or performance (or symptom exaggeration or malingering) and examination and test performance data provides the best information for estimating, for instance, the degree to which a person was responding truthfully and exerting full effort, and the degree to which test results are reliable and valid and reflect actual abilities and current status.

It also should be noted that the necessary recent increase in attention to response bias assessment has been accompanied by frequently haphazard and overzealous application of poorly validated detection models and single

assessment procedures regarding malingering. Further, some alarming trends have appeared that do not objectively or critically evaluate the weaknesses, as well as strengths, of these procedures. Based on a critical evaluation of the current state of the art, it appears that many common assumptions about response bias detection and malingering measures should be considered myths (Martelli, Zasler, Mancini, et al., 1999). Importantly, malingering (1) should not be considered dichotomous, or EITHER/OR (i.e., present/not; malingering/not); (2) should not be considered something that clinicians can reliably or validly assess with any high degree of certainty, even when serious efforts are made; and (3) should not be considered a discrete entity that symptom validity tests (SVT) measure.

TABLE 63.4
General Weaknesses of Response Bias Assessment Measures

1. Psychometric research inadequacies (e.g., basic test construction issues such as reliability, validity, as well as convergent and divergent validity studies are poorly addressed).
2. Limited generalizability of analogue research (i.e., simulated malingerers vs. externally or criterion-validated malingerers, unknown differences between simulated and real malingerers; cf. studying serial killers this way), as well as tendencies for measures with good discrimination to show less effectiveness in cross-validation and follow-up studies.
3. Variable group membership (i.e., wide variability in samples for both simulators and symptom/disorder groups).
4. Differential vulnerability to response bias (i.e., some tests are more obvious while others are more subtle).
5. Questionable generalizability of findings (i.e., from one measure to any other (response bias or real) test, or to actual symptoms, or across time; conversely, good effort on a response bias measure does not necessarily predict response on any other measure).
6. Absence of mutual exclusivity (i.e., poor effort can occur in presence of real disorder, symptoms).
7. "Law of the instrument" operational definitions wherein malingering becomes what malingering tests measure. Specifically, the definitions of "effort," and validation studies to examine the construct are missing. Further effort cannot be assumed uniform for mild traumatic brain injury (TBI), chronic pain, and depression diagnoses, for nonlitigating and litigating, etc.
8. Effects of fatigue, pain, disinterest, non-attended (computer) administration, mixing real tests and SVTs in a battery with unknown face validity, and other factors, on response bias tests, are not understood and have not been addressed.
9. Exclusive or even primary reliance on any current SVT/Index or combination potentially violates APA ethics and *APA Standards for Educational and Psychological Tests* with regard to making a diagnosis of malingered pain or in making decisions about recommending treatment termination, due to limited reliability and validity data.
10. Frequently high misclassification rates (i.e., false positive) when these are assessed through record review and detailed analysis.
11. Problems associated with inaccurate assumptions of nonorganic conditions based on inconsistencies or absence of peripheral findings. Notably, recent advances in our relatively poor understanding of pain and its mechanisms and associated sequelae have implicated central nervous system effects in many such cases. A growing body of evidence strongly associates central nervous system effects, especially central sensitization phenomenon, in cases where peripheral findings are inconsistent, weak, or even apparently nonexistent (Jay, Krusz, Longmire, & McLain, 2000; Miller, 2000; Nicholson, 2000; Nicholson, in review; Mailis, Papagapiou, Umana, Cohodarevic, Nowak, & Nicholson, in press).

A specific method of response bias assessment that is worth mentioning is SVT, which typically refers to a forced-choice technique originally designed for assessing effort or symptom validity with respect to nonorganic blindness (Pankratz, 1988). This technique has been extended to assess effort in purported sensory loss and, more recently, memory complaints (Colby, 2000). The typical SVT paradigm involves presentation of a stimulus, followed by a distraction, and then presentation of the original stimulus with a novel stimulus with instruction to identify the original stimulus. With regard to memory assessment, a series of words is presented for recall and, following a delay, each word is presented with a sham, with the subject instructed to select the previously presented word. In the case of visual or sensory assessment, the simplest procedure entails exposing the subject to a series of visual or sensory stimuli (e.g., pinpricks while blindfolded, asking whether or not he or she perceived each). Performance is then compared with chance, which is the worst possible expected performance if sensory function or ability is completely absent. Below chance (i.e., below 50%) performance across a sample of numerous trials indicates negative bias and indicates that the symptom is feigned. Such performance provides strong and unambiguous evidence of conscious dissimulation or symptom malingering, because worse-than-chance performance requires recognition and suppression of true responses.

A summary of some of the major problems with extant response bias procedures is offered in Table 63.4 to (1) emphasize the necessary caution with regard to overinterpretation of response bias procedures; (2) emphasize the importance of employing multiple data sources and making thoughtful inferences only after integration of thorough historical information, interview, assessment, behavioral observations, collaborative interview, and data sources, and so on (Martelli, 2000).

Table 63.5 is presented to further caution against simplistic and dichotomous conceptualizations with regard to diagnosis, Table 63.5 is presented. Notably, this table represents just 64 of the possibilities with regard to injury-related presentations. The range of possibilities represented span from (a) persons with real, uncomplicated disorders with impairments on exam and in functional status, without exaggeration on either (but possibly minimization or denial) to (b) persons with no real physical pathology or impairments, but who exaggerate or feign impairments on exam and functional status.

Necessarily, a cautious approach is indicated with regard to estimating the probabilities regarding presence or absence of physical impairment and response bias. However, in many cases, it is not sufficient to integrate data from multiple sources and make inferences about which of the 64 possible combinations is most likely. Descriptive characterization is often relevant. For

TABLE 63.5
Diagnostic Realities in Assessment of Chronic Pain

Real Physical Pathology		Residual Functional Impairments		Residual Impairments On Exam, Testing	
1. Yes		1. Yes, and exaggerated		1. Yes, and not exaggerated	
2. Mixed		2. Yes, and not exaggerated		2. Yes, and exaggerated	
3. Indeterminate		3. No, and exaggerated		3. No, and exaggerated	
4. No		4. No, and not exaggerated		4. No, and not exaggerated	=
4	×	4	×	4	64

instance, if a person has both physical pathology and exaggeration, inferences must be generated about not only the degree of physical impairment, but also the degree of awareness of exaggeration on the part of the subject. Has the person adopted a sick role, and talked themselves into believing they cannot perform certain tasks or lack certain abilities (e.g., somatoform disorder), with conscious withholding of effort due to intending to demonstrating what they believe to be true disabilities? Or, are they less conscious and aware, as in a conversion disorder? Or are they completely aware, but coping in a way that may be adaptive as in the case of an aging worker with a chronic history of back failures who may be shy, have low self-esteem and self-confidence; be disconnected from or less than well liked by his/her employer, against the backdrop of believing that another back injury is inevitable and cumulatively painful and disabling, that uncomfortable interactions with others may be required, that the company sometimes fires previously injured workers, that the company did not make obvious safety precautions to prevent the individual's injury; and that no other job options are realistic?

CONCLUSIONS

To summarize, the major response bias detection strategies presented in Table 63.3 provide an illustrative summary of a constellation or profiling approach to response bias detection strategy use that relies on assessing relevant information for interpreting examination data. This conceptual model is also a methodological approach for constructing a profile of motivation and response bias, which (a) incorporates a wide array of findings from common instruments and procedures during evaluation; (b) summarizes empirically supported indicators with at least some purported utility in detecting suboptimal effort; (c) despite numerous pitfalls and limitations of each of these procedures, both conceptual and methodological, offers improved discrimination and increased reliability given multiple measures; (d) integrates behavioral observation, interview, and collaborative, historical, personality, and contextual data with medical examination and psychological performance data and measures of response bias, as

an optimal method for estimating the degree of effort or performance and the degree to which examination findings are reliable and valid and reflect actual abilities; and (e) allows estimation of motivation by incorporating currently available instruments and methods and the available published research for direct and indirect measurement of motivation and response bias.

Notably, these strategies are not offered individually and, again, are not intended to support a simple dualistic model that assumes examinees either try hard or malingering, or that evidence of less-than-full effort on any one test necessarily implies absence of impairment in other areas of examination or in real world abilities. Although they also are not offered with specific guidelines (e.g., failure on any one, or any two, or any three, etc. represents inadequate performance, or symptom exaggeration or malingering), they are offered with the suggestion that (a) examination performance can be influenced by multiple factors including a desire to be completely truthful and perform with full effort; (b) the degree of truthfulness and effort exerted on examinations exists on a continuum (vs. a dichotomy) and can be estimated by the extent to which indicators of unreliable report and poor/inconsistent effort are present; (c) reliability and validity of examination findings are dependent on relative assurances of full effort; and (d) interpretation and diagnostic impressions are dependent upon reliable and valid examination results.

It should be emphasized that "failure" on one measure of response bias or malingering does not mean that the entire set of complaints is biased or malingered. Ethical guidelines universally caution against overzealous interpretation of limited test data. In fact, the only reasonable evidence of certain or definite malingering is confession or admission. A secondary form of evidence, although somewhat less than perfectly reliable, is when the person or examinee is detected, via surveillance, performing an act he or she reported was absolutely impossible to perform under any circumstance.

It should be noted further that a great disparity exists between the adversarial legal process and the responsibility of attorneys to be client advocates vs. the dispassionate, objective scientific ethics expected and required of psychologists and physicians. The danger of attorney

“coaching” based on utilization of this material cannot be underestimated. This, of course, would then represent a form of “stealth” threat to the validity of examination data. This threat, or expected consequence of collision between disparate legal and scientific ethics, has recently been documented in a national publication noting a case of attorney–client coaching (Youngjohn, 1995). However, compared to simpler models where only a couple of isolated response bias measures are used, it seems extremely unlikely that the multiple measures, such as those outlined in the MAP approach, could be understood and manipulated.

Finally, enhancing response bias detection as a means of optimizing interpretability of examination results, critical as it is, should not be considered the final step. Decreasing response bias must certainly be considered a more efficacious and economic approach to enhancing utility of medical and psychological assessments.

The following explicit and comprehensive recommendations for enhancing motivation, assessing response bias, and increasing efficiency, utility, and ecological validity of examination procedures are offered (Martelli, et al., 1999b).

RECOMMENDATIONS FOR ENHANCING VALIDITY IN CHRONIC PAIN ASSESSMENTS

1. Establish rapport and attempt to establish a working relationship with patients. Even in cases of independent examinations where the referral source and expectation are extremely adversarial, valid data collection requires a collaborative effort. Be on guard by addressing potential sources of bias directly, and providing feedback, education, and clarification.
2. Prepare patients and examinees before beginning examination and testing. Employ understanding, as well as education, in order for examinees to be prepared to respond truthfully and to the best of their abilities. Emphasize that the procedures and tests don't always measure everything, but that they do assess poor motivation or effort. Emphasize that interview data, corroborative data, and functional abilities are just as important as examination data.
3. Spend time with patients/examinees and try to get to know them from a motivational, emotional status, personality, and coping style perspective. If motivation seems poor, confront and attempt to elicit more valid responses vs. ignore and/or proceed with collecting invalid data and/or attempting to interpret data of questionable validity. Such questioning of motivation/effort should not involve a “gotcha” attitude. We can't assume that everyone takes our tests seriously, will be as forthcoming, honest, or effortful as we would like, will not doubt our procedures or try to emphasize their problems, or that we won't have to work at getting them optimally motivated.
4. Ensure that important general situational and psychosocial variables affecting motivation are adequately assessed during an interview that is concluded prior to examination procedures. Specifically, assess the impact of anger or blame and feelings of resentment or victimization (Rutherford, 1989), as well as the other variables shown in the literature to be associated with poor recovery and adaptation to impairments (Martelli, Zasler, & Grayson, 2000).
5. In addition to emotional and motivational issues, always assess interest/disinterest in the examination and testing procedures process, and any obstacles or impediments to optimal effort and performance. Always assess anxiety level and ensure that measures are taken to minimize its effect and potential interference with valid assessment.
6. Rely primarily on M.D.s and Ph.D.s for all aspects of examination, including interviewing and testing, with limited use and reliance on technicians. Experienced M.D.s and Ph.D.s who conduct interviews, examinations, and test administration are infinitely more capable of
 - a. Integrating history, interview, personality, and emotional assessment data and inferences, with more sophisticated clinical observations during examination;
 - b. Adapting more creative modifications of testing procedures given suspicion of low motivation, as well as modifications to the testing process (e.g., provision of corrective feedback, instruction, anxiety reduction interventions) to increase motivation and optimize effort;
 - c. Benefiting from the probability that examinees will be less likely to believe they can “fake out the doctor”;
 - d. Avoiding the possibility of symptom exaggeration owing to fear that a technician or inexperienced clinician will miss legitimate problems.
7. Differentially utilize instruments with built-in response bias or symptom validity measures. Most major objective personality measures, some of the newer domain-specific pain assessment measures, and some neuropsychological measures (e.g., Memory Assessment Scales (Williams, 1992), and the Rey Complex Figure Test and

- Recognition Trial (Meyers & Meyers, 1995)) provide simulator performance data.
8. Apply multiple strategies for assessing motivation, especially when cutoff score approaches are employed, and include qualitative and qualitative measures. Integration of contextual information, history, behavioral observations, interview and collaborative data, and personality and coping data with measures of effort or performance and current test data, provides the best information for estimating the degree of effort exerted, and the degree to which test results are reliable and valid.
 9. Vary the response bias measures and procedures that are employed in order to prevent dilution of utility. Notably, publicizing of these tests has led to increased recognition by potential defendant attorneys, litigants, support groups, Internet groups, etc.
 10. Promote development of assessment procedures with built-in response bias or symptom validity measures and develop built-in measures for existing assessment procedures.
 11. Employ more sophisticated and less dichotomous continuous conceptualizations of motivation and response bias using multiple independent measures and estimated effort. Employ a reasonably sophisticated model that conceptualizes motivation and effort as continuous variables that can vary across tests, settings, and occasions. Utilize and devise models that measure degree of apparent motivation and effort, using multiple data sources, and estimate confidence levels in inferences given consideration of the multiple factors that contribute to test results. Employ similarly sophisticated models for assessing persistent impairments, adaptation to impairments, disability, and so on. Probability statements based on multiple measures are probably best.
 12. Do not freely share relevant trade secrets (e.g., information about response bias tests, or known patterns of performance on procedures and instruments) with referral sources, attorneys, and nonphysicians and nonpsychologists. They often adhere to a completely different set of professional ethics.
 13. Remain aware that in science and medicine things are rarely either-or, clear cut, or unidimensional. Avoid simplistic conceptual models that are compatible with dichotomous approaches to assessing motivation/effort and malingering. Such approaches usually rely on cutting scores for one or two measures. Note that cutting scores by their nature (Dwyer, 1996) always entail judgment, inherently result in misclassification, impose an artificial dichotomy on essentially continuous variables, and "true" cut scores do not exist.
 14. Promote utilization of independent examinations by clinicians who actually spend a significant portion of their time treating the type of patient being assessed. This helps assure more adequate clinical skills for accurate diagnosis and understanding, including detection and appreciation of suboptimal performance, as well as collection of internalized tracking data to validate previous inferences across time, and continuous self-correction and increased collection of internalized norms regarding ecological and predictive validity of available assessment measures.

REFERENCES

- * Allen, C.C., & Ruff, R.M. (1990). Self-rating versus neuropsychological performance of moderate versus severe head-injured patients. *Brain Injury*, 4(1), 7.
- * Allen, L.M., & Cox, D.R. (1995). *Computerized assessment of response bias* (revised ed.). Durham, NC: Cogni-Syst, Inc.
- * Allen, M. (1985). Review of Millon Behavioral Health Inventory. In J.V. Mitchell (Ed.), *The ninth mental measurements handbook* (p. 1521). Lincoln, NE: University of Nebraska Press.
- American Psychiatric Association (1994). Committee on Nomenclature and Statistics. *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, D.C.: American Psychiatric Association.
- * American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders*, (4th ed. revised). Washington, D.C.: American Psychiatric Press.
- * Arnett, P.A., Hammeke, T.A., & Schwartz, L. (1995). Quantitative and qualitative performance on Rey's 15-Item Test in neurological patients and dissimulators. *Clinical Neuropsychologist*, 9(1), 17.
- Babitsky, S., Brigham C.R., & Mangraviti, J.J. (2000). Symptom magnification, deception and malingering: Identification through distraction and other tests and techniques (VHS video). Falmouth, MA: SEAK, Inc.
- * Beetar, J.T., & Williams, J.M. (1995). Malingering response styles on the memory assessment scales and symptom validity tests. *Archives of Clinical Neuropsychology*, 10(1), 57.
- * Bernard, L.C., Houston, W., & Natoli, L. (1993a). Malingering on neuropsychological memory tests: Potential objective indicators. *Journal of Clinical Psychology*, 49(1), 45.

- * Bernard, L.C., McGrath, M.J., & Houston, W. (1993b). Discriminating between simulated malingering and closed head injury on the Wechsler memory scale-revised. *Archives of Clinical Neuropsychology*, 8(6), 539.
- * Berry, D.T., Baer, R.A., & Harris, M.J. (1991). Detection of malingering on the MMPI: A meta-analysis. *Clinical Psychology Review*, 11, 585.
- Binder, L.M., & Rohling M.L. (1996). Money matters: A meta-analytic review of the effects of financial incentives on recovery after closed-head injury. *American Journal of Psychiatry*, 153(1), 7.
- * Binder, L.M., & Pankratz, L. (1987). Neuropsychological evidence of a factitious memory complaint. *Journal of Clinical & Experimental Neuropsychology*, 9(2), 167.
- Blau, T. (1984). *The psychologist as expert witness*. New York: John Wiley & Sons.
- Blau, T. (1992). The psychologist as expert witness. Presented at the National Academy of Neuropsychology annual meeting, Reno, Nevada.
- * Brandt, J. (1988). Malingered amnesia. In R. Rogers (Ed.), *Clinical assessment of malingering and deception* (pp. 65-83). New York: Guilford Press.
- Colby, F. (2000). Does the binomial distribution stand falsely accused? *Brain Injury Source*, 4(4), 18-21.
- * Cullum, C., Heaton, R., & Grant, I. (1991). Psychogenic factors influencing neuropsychological performance: Somatoform disorders, factitious disorders and malingering. In H.O. Doerr, & A.S. Carlin (Eds.), *Forensic neuropsychology*. New York: Guilford Press.
- Dwyer, C.A. (1996). Cut scores & testing: statistics, judgment, truth, and error. *Psychological Assessment*, 8(4), 360.
- * Elmer, B.N., & Allen, L.M. (1995). *User's guide to the pain assessment battery* (Research ed.). Durham, NC: Cogni-Syst, Inc.
- * Faust, D. (1995a). The detection of deception. Special issue: Malingering and conversion reactions. *Neurologic Clinics*, 13(2), 255.
- Fishbain, D.A., Cutler, R.B., Rosomoff, H.L., & Rosomoff, R.S. (1999). Chronic pain and disability exaggeration/malingering research and the application of submaximal effort research to this area: A review. Poster presented at International Association of Pain 9th World Congress, Vienna, Austria.
- * Franzen, M.D., & Iverson, G.L. (1998). Detecting negative response bias and diagnosing malingering: The dissimulation exam. In P.J. Snyder & P.D. Nussbaum (Eds.), *Clinical neuropsychology - A pocket handbook for assessment* (pp. 88-101). Washington, D.C.: American Psychological Association.
- * Green, P., Iverson, G., & Allen, L. (1999). Detecting malingering in head injury litigation with the Word Memory Test. *Brain Injury*, 13(10), 813.
- Hadjistavropoulos, H.D., & LaChapelle, D.L. (2000). Extent and nature of anxiety experience during physical examination of chronic low back pain. *Behavior Research and Therapy*, 38(1), 13.
- Hall, H.V., & Pritchard, D.A. (1996). *Detecting malingering and deception: Forensic distortion analysis*. Delray Beach, FL: St. Lucie Press.
- * Hayes, J.S., Hilsabeck, R.C., & Gouvier, W.D. (1999). Malingering traumatic brain injury: current issues and caveats in assessment and classification. In N.R. Varney, and R.J. Roberts (Eds.), *The evaluation and treatment of mild traumatic brain injury*. Mahwah, NJ: Lawrence Erlbaum Associates.
- * Hendler, H.H., & Eimer, B.N. (in press). Psychological tests for assessing chronic pain and disability.
- * Hendler, N.H., Viernstein, M., Gucer, P., & Long, D. (1979). A preoperative screening test for chronic back pain patients. *Psychosomatics*, 20(12), 301.
- * Iverson, G.L. Qualitative aspects of malingered memory deficits. *Brain Injury*, 9(1), 35, 1995.
- Jay, G.W., Krusz, J.C., Longmire, D.R., & McLain, D.A. (2000). *Current trends in the diagnosis and treatment of chronic neuromuscular pain syndromes*. Las Vegas: American Academy of Pain Management and Elan Pharmaceuticals.
- Lees-Haley, P., & Brown, R.S. (1993). Neuropsychological complaint base rates of 170 personal injury claimants. *Archives of Clinical Neuropsychology*, 8, 203.
- Lees-Haley, P.R., Williams, C.W., Zasler, N.D., Margulies, S., English, L.T., & Steven, K.B. (1997). Response bias in plaintiff's histories. *Brain Injury*, 11(11), 79-99.
- Lezak, M. (1965). *Neuropsychological assessment*, (3rd ed.). New York: Oxford University Press.
- Lipman, F.D. (1962). Malingering in personal injury cases. *Temple Law Quarterly*, 35, 141.
- Loring, D.W. (1995). Psychometric detection of malingering. Presented at the annual meeting of the American Academy of Neurology, Seattle.
- Mailis, A., Papagapiou, M., Umana, M., Cohodarevic, T., Nowak, J., & Nicholson, K. (in press). Unexplainable widespread somatosensory deficits in patients with chronic nonmalignant pain in the context of litigation/compensation: A role for involvement of central factors. *Journal of Rheumatology*.
- Main, C.J., & Spanswick, C.C. (1995). Functional overlay, and illness behaviour in chronic pain: distress or malingering? Conceptual difficulties in medico-legal assessment of personal injury claims. *Journal of Psychosomatic Research*, 39(6), 737.
- Martelli, M.F. (2000). Psychological Assessment of Response Bias in Impairment and Disability Ratings. Presentation as part of Symposium #2031 (The Psychologist's Role in the Social Security Disability Process) at the American Psychological Association 2000 Convention, Washington, D.C.
- Martelli, M.F., Zasler, N.D., & Grayson, R. (1999a). Ethical considerations in medicolegal evaluation of neurologic injury and impairment. *NeuroRehabilitation: An Interdisciplinary Journal*, 13, 1, 45.
- Martelli, M.F., Zasler, N.D. & Grayson, R. (1999b). Ethical considerations in impairment and disability evaluations following injury. In R.V. May & M.F. Martelli (Eds.), *Guide to functional capacity evaluation with impairment rating applications*. Richmond: NADEP Publications.

- Martelli, M.F., Zasler, N.D., & Grayson, R. (2000). Ethics and medicolegal evaluation of impairment after brain injury. In M. Schiffman (Ed.), *Attorney's guide to ethics in forensic science and medicine*. Springfield, IL: Charles C Thomas.
- Martelli, M.F., Zasler, N.D., Mancini, A.M., & MacMillan, P. (1999). Psychological assessment and applications in impairment and disability evaluations. In R.V. May & M.F. Martelli (Eds.), *Guide to functional capacity evaluation with impairment rating applications*. Richmond: NADEP Publications.
- Matheson, L. (1988). Symptom magnification syndrome. In S. Isernhagen (Ed.), *Work injury*. Rockville, MD: Aspen Publishers.
- Matheson, L. (1990). Symptom magnification syndrome: A modern tragedy and its treatment. Part one: Description and definition. *Industrial Rehabilitation Quarterly*, 3(3), 1, 5, 8-9, 12, 23.
- Matheson, L. (1991a). Symptom magnification syndrome: A modern tragedy and its treatment. Part two: Techniques of identification. *Industrial Rehabilitation Quarterly*, 4(1), 1-17.
- Matheson, L. (1991b). Symptom magnification syndrome: A modern tragedy and its treatment. Part three: Techniques of treatment. *Industrial Rehabilitation Quarterly*, 4(2), 5-6, 22-24.
- May, R.V. (1999). Symptom magnification syndrome. In R.V. May & M.F. Martelli (Eds.), *Guide to functional capacity evaluation with impairment rating applications*. Richmond: NADEP Publications.
- Merskey, H. (1986). Classification of chronic pain, descriptions of chronic pain syndromes and definitions of pain terms. *Pain*, 3, S10-S11, S13-S24.
- * Meyers, J., & Volbrecht, M. (1999). Detection of malingerers using the Rey complex figure and recognition trial. *Applied Neuropsychology*, 6, 4, 201.
- * Meyers, J.E., & Meyers, K.R. (1995). *Rey complex figure and recognition trial*. Odessa, FL: Psychological Assessment Resources.
- Miller, L. (2000). Neurosensitization: A model for persistent disability in chronic pain, depression, and posttraumatic stress disorder following injury. *NeuroRehabilitation*, 14(1), 25-32.
- Miller, L. (2001). Not just malingering: Syndrome diagnosis in traumatic brain injury litigation. *NeuroRehabilitation*, 16, 1-14.
- * Millis, S. R. (1994). Assessment of motivation and memory with the recognition memory test after financially compensable mild head injury. *Journal of Clinical Psychology*, 50(4), 601.
- * Mittenberg, W., Arzin, R., Millsaps, C., & Heilbronner, R. (1993). Identification of malingered head injury on the Wechsler Memory Scale. *Psychological Assessment*, 5, 34.
- * Morey, L.C. (1996). *An interpretive guide to the personality assessment inventory (PAI)*. Odessa, FL: Psychological Assessment Resources.
- Nicholson, K. (2000). At the crossroads: Pain in the 21st century. *NeuroRehabilitation*, 14, 57.
- Nicholson, K., Psychogenic pain: Review of the construct, a novel taxonomy and neuropsychobiological model (in review).
- * Nies, K.J., & Sweets, J.J. (1994). Neuropsychological assessment and malingering: A critical review of past and present strategies. *Archives of Clinical Neuropsychology*, 9(6), 501.
- Pankratz, L. (1988). Malingering on intellectual and neuropsychological measures. In R. Rogers (Ed.), *Clinical assessment of malingering and deception*. New York: Guilford Press.
- * Rogers, R. (Ed.). (1988). *Clinical assessment of malingering and deception*. New York: Guilford Press.
- Rohling, M.L., & Binder, L.M. (1995). Money matters: A meta-analytic review of the association between financial compensation and the experience and treatment of chronic pain. *Health Psychology*, 14(6), 537.
- Rutherford, W. (1989). Postconcussion symptoms: Relationship to acute neurological indices, individual differences, and circumstances of injury. In H.S. Levin, H.M. Eisenberg, and A.L. Benson (Eds.), *Mild head injury* (p. 229). New York: Oxford University Press.
- Simmonds, M.J., Kumar, S., & Lechelt, E. (1998). Psychosocial factors in disabling low back pain: Causes or consequences? *Disability and Rehabilitation*, 18, 161.
- Waddell, G. (1999). Nonorganic signs or behavioral responses to examination in low back pain. *Hippocrates' Lantern*, 6(3), 1.
- Waddell, G., & Main, C.J. (1984). Assessment of severity in low back disorders. *Spine*, 9, 204-208.
- Waddell, G., Main, C.J., Morris, E.W., Paolao, M.D., & Gray, I.C. (1984). Chronic low back pain, psychologic distress, and illness behavior. *Spine*, 9, 209.
- * Williams, J.M. (1992). *The memory assessment scales*. Odessa, FL: Psychological Assessment Resources.
- * Youngjohn, J.R. (1995). Confirmed attorney coaching prior to neuropsychological evaluation. *Assessment*, 2(3) 279.
- Youngjohn, J.R., Burrows, L., & Erdal, K. (1995). Brain damage or compensation neurosis? The controversial post-concussion syndrome. *Clinical Neuropsychologist*, 9(2), 112.

Myths and Misconceptions about Chronic Pain: The Problem of Mind–Body Dualism

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As with most other phenomena, there are many myths or misconceptions about chronic pain. At the risk of being mundane, pretentious, or even reinforcing existing misconceptions, this chapter explores what some of these may be. Simply, myths or misconceptions arise out of an incomplete understanding and reflect our ignorance of the material at hand. Myths or misconceptions can also arise out of apparent but possibly false dichotomies. A fundamental dichotomy permeating many domains of human activity, from at least the time of Descartes, is mind–body dualism. This can be especially problematic for the understanding of chronic pain. Much of the current chapter is devoted to one aspect or another of this dichotomy and the misconceptions or confusion that flow from a biased perspective. Although some may think that reviewing this issue is akin to “beating a dead horse,” this may also represent an important misconception. Rather, it is argued that explication of what the interface between the two sides of this issue may be is a most important avenue of research. It should also be noted that many myths or misconceptions may be rooted in one’s professional training or background. In this regard, it is acknowledged that the predominant perspective of the current chapter is from that of psychologists working in the field of chronic pain.

Perhaps the most elemental myth or misconception is that we know what pain is. We do not. Pain is but a four-letter word or, preferably, a construct that facilitates understanding and communication of a number of related phenomena. One touches a hot stove, experiences “pain,” and pulls one’s arm away or says “ouch.” Certainly, there is a

shared intersubjective experience of many acute pain phenomena and, in this sense, acute pain is similar to the experience of a yellow banana or red apple. There is some understanding of the underlying neurobiology of the acute pain experience traditionally associated with the specificity theory of pain and thought to involve peripheral nociception with transmission of information about pain via the lateral pain system to the brain where it is experienced (somehow) as pain. In such situations, the nociceptive stimulus is usually withdrawn or there is healing of damaged tissue and the experience of pain resolves. Although there is certainly much to be learned about this acute pain response (e.g., why one may not experience pain in the midst of battle, during hypnosis or other activity), the problem becomes more complex and mysterious when one considers the transition to chronic pain.

Unlike the situation with color perception (i.e., where specific wavelengths of light may reliably be associated with the perception of particular colors) or many other domains of experience, there is often a poor relationship between the “subjective” experience of pain and “objective” or external referents. This may be most evident in the case of chronic pain where apparently similar peripheral pathology, injury, or nociceptive input can result in markedly different presentations. Whereas patient self-report, using verbal analogue or other rating scales, is perhaps the most straightforward and appropriate means of determining pain severity (or other aspects of the pain experience), this is prone to response bias like all self-reports. In this regard, it has been suggested that less than 10% of the chronic pain

population may consume as much as 70 to 80% of the resources (Linton, 1999), possibly due to a response bias to report pain and related problems, dependency behavior or other factors associated with the distinction between impairment and disability (Martelli, Zasler, & MacMillan, 1998; Martelli, Zasler, Mancini, & MacMillan, 1999).

A response bias to report pain may be suspected when there are extremely high severity ratings and exuberant pain behavior with high affective distress and (possibly exaggerated) suffering in the context of few, if any, clinical findings. In such cases, one might conclude that underlying emotional problems rather than pain are really the primary problem. On the other hand, many patients present with an apparent "belle indifference" and, despite giving extremely high pain severity ratings (e.g., 9/10 or even 10/10 — "the worst that pain could ever be"), they may appear entirely comfortable, in no apparent distress whatsoever, and often in much better spirits than the examiner. When challenged, such patients will typically maintain that their pain is very severe (e.g., worse than childbirth). It becomes very difficult to ascertain what the meaning of pain is in such discrepant presentations. However, it is likely that the problem of response bias, which reflects the meaning of the experience of pain, is not restricted to the unusual or extreme cases and may often confound presentation in even more "legitimate" cases.

Most clinicians have encountered patients who complain that some doctors think their pain is "all in their heads," that is, it is just psychological, has no organic basis, or is not real. Many doctors may also sometimes hold such an opinion especially if there are no signs of any relevant pathological process, the presentation is otherwise inconsistent with expectation, or the patient appears somehow quirky and with obvious psychological problems. Of course, most would readily accept that lack of a discernible pathological process does not mean that there is not one. There is obviously much to learn about the underlying biological processes involved in chronic pain as well as in many other medical conditions. It is sometimes suggested (to patients or others) that pain is always experienced in the brain and thus is actually "all in the head." However, this begs the question about whether the pain is more "psychological" or "organic" as any psychological phenomena (e.g., reading this sentence or reading the next sentence) is presumed to have a neurobiological substrate. It should be remembered that the definition of pain accepted by the International Association for the Study of Pain (IASP) states that pain is always a psychological state (Merskey & Bogduk, 1994) but this should not be misinterpreted to mean that it is only a psychological state devoid of any physical basis in reality.

Another way of expressing the question of whether pain is in the mind or body is in terms of it being either functional or organic. Organic here implies structural damage or aberration generating "real" pain, whereas functional is com-

monly meant to imply that presentation is associated with (usually preexisting) psychological or psychiatric problems and that the corresponding perception or self-report of pain may be distorted or magnified. However, there is increasing realization that chronic pain problems may truly be functional but, in this sense, functional refers to the effects of distributed neural networks involved in the processing of pain in contrast with the view of pain as involving a static structural (peripheral or central) lesion generating some nociceptive or neuropathic process (Wall, 2000). This is perhaps especially true of chronic nonmalignant or idiopathic pain. Notably, distributed neural networks underlie many (or most) neuropsychological functions such as motor, vision, or language. In this regard, pain might be considered akin to vision where there is clearly a peripheral and central biological apparatus mediating function but where psychological factors are also clearly pertinent and, therefore, we tend to see what we want to see or perception is otherwise colored by experience. Much of the brain has been shown to be involved in processing of painful information in one manner or another (Besson, Guilbaud, & Ollat, 1995; Bromm & Desmedt, 1995; Chundler & Dong, 1995). There is limited understanding of how several more dedicated areas actually operate. There is also very poor understanding of how the "pain system" interacts with other systems (e.g., endocrine, immune, motor, cognitive, etc).

With regard to the functional-structural distinction, Mailis, Amani, Umana, Basur, & Roe (1997) have documented a dissociation of separable components of pain in a sample of neuropathic pain patients, i.e., a deep pain component mediated by peripheral nociceptors plus a cutaneous component (allodynia) considered to be a product of central sensitization. Several further studies by this group utilizing sodium amytal or other techniques have purported to document structural-functional dissociations in several other patient samples and that functional aspects of presentation appear to be associated with specific psychosocial factors (Cohodarevic, Mailis, & Montanera, 2000; Mailis & Nicholson, 1997). Whereas the concept of central sensitization has certainly come into vogue, most study remains at the level of the spinal cord or periphery whereas the most important effects may well be supraspinal. Of course, it is much more difficult to conduct experiments at this level. It is also important to recognize that a focus upon how the brain is processing pain does not resolve the mind-body problem, although it may take it a step closer. Caution should also be exercised about neglecting peripheral factors in favor of how the brain is processing pain. It would be unfortunate if the pendulum were to swing and one misconception about chronic pain was displaced only to be replaced by another.

There is a widespread belief that functional neuroimaging will allow us to unlock the secrets of pain or at least provide for some better "objective" indices of pain. Unfortunately, most functional neuroimaging studies of pain

have involved acute pain challenges with normal controls. Results have revealed widespread patterns of activation and deactivation in multiple cortical and subcortical sites (Coghill, Sanf, Maisog, & Ladoralo, 1999; Hsieh et al., 1995; Treede, Kenshalo, Gracely, & Jones, 1999). Indeed, almost all of the brain has been shown to respond in one paradigm or another. There has been more consistent (albeit not always found) patterns of activation in the anterior cingulate, insula, somatosensory cortex (S1 and/or S2), and somatosensory thalamus, findings that might have been anticipated given what is known about the neuroanatomy of the pain system. Notably, however, the anterior cingulate is activated by almost any behavioral challenge (Cabeza & Nyberg, 1997) and the insula is activated by somatosensory stimulation whether noxious or not (Craig, Reinan, Evans, & Bushnell, 1996), although it may be that processing of pain takes place within discrete parts of these cortical areas (Davis, Taylor, Crawley, Wood, & Mikulis, 1997). Furthermore, activation may be seen in the same areas if the subject merely anticipates pain (Drevets et al., 1995; Porro, Francescato, Cettolo, & Baraldi, 1995), although one study has found that the sites of activation with actual vs. anticipated pain can be distinguished (Ploghaus et al., 1999). It remains unclear what the significance of most of these findings are, or whether this technique will fulfill the promise of providing an important inroad to the understanding of chronic pain. However, this technology by itself cannot be expected to resolve mind-body problems. Rather, such issues will only successfully be addressed with relevant psychological analysis of the phenomena of interest (e.g., fear avoidance, response bias to report pain, etc.) coupled with appropriate behavioral challenge during neuroimaging with groups of patients who have been well defined (psychologically and medically) prior to imaging.

There is a long tradition in distinguishing sensory-discriminative, motivational-affective, or cognitive-evaluative components of pain (Melzack & Casey, 1966). Such distinctions often resolve into the dichotomy between the sensory-discriminative vs. the motivational-affective, the latter subsuming the cognitive-evaluative. It seems to often be assumed, at least implicitly, that these are distinct or independent components of pain but that "real" pain necessarily involves the sensory-discriminative component, the motivational-affective or cognitive-evaluative components merely being the emotional or cognitive overlay. More recently it has been suggested that such distinctions are too simplistic or misleading, again leading into problems of Cartesian dualism (Treede et al., 1999; Wall, 2000). There has also been a tradition of distinguishing between the lateral and medial pain systems, generally corresponding to the distinction between the sensory-discriminative vs. motivational-affective and cognitive-evaluative components of pain (Vogt, Sikes, & Vogt, 1993). It remains unclear to what extent these systems are independent and

processing information in parallel or how there is interdependence of function and unification of the experience of pain (Vogt, et al., 1993; Treede et al., 1999). Notably, this same issue, integration vs. independence or differentiation of function, has also been a subject of debate with regard to the function of basal ganglia-thalamocortical loops mediating motor, visual, cognitive, affective or other functions (Alexander, DeLong, & Strick, 1986; St-Cyr, Taylor, & Nicholson, 1995).

Many instances or types of central pain occur (e.g., post-stroke central pain) when central, often supraspinal, activation of the pain system produces the experience of pain independent from any peripheral pathology (Nicholson, 2000a). Such pain can be very severe and very real. As indicated previously, chronic pain can be expressed independent from any structural lesion (central or peripheral) as a consequence of central sensitization or functional neural networks. This might account for many puzzling presentations, perhaps especially in those cases of idiopathic pain or when there is no indication of any significant peripheral pathology, but mechanisms of effect remain largely unknown.

The gate control theory initially provided great promise of an integration of peripheral and central, ascending and descending, or neurobiological and psychological factors (Melzack & Wall, 1965). Unfortunately, this paradigm has failed to generate such integration, although there has been an immense amount of work devoted to understanding the microcircuitry of the spinal cord. In the recent past, several other models with a focus on supraspinal mechanisms have been suggested (Birbaumer, Flor, Lutenberger, & Elbert, 1995; Chapman, 1995, 1996; Flor et al., 1995; Flor, Braun, Elbert, & Birbaumer, 1997; Lenz, Gracely, Zirh, Romanowski, & Dougherty, 1997; Melzack, 1999; Wood, Alpers, & Andrews, 1999). These all accept, more or less explicitly, the importance of some neuropsychobiological interface. Some have provided good detail of possible neurobiological mechanisms but there has been very poor, if any, actual integration of psychological and neurobiological factors. Rather, there has been only general suggestion of how psychological processes (e.g., emotion, memory, conditioning, stress, personality) may be related to underlying neurobiological processes. Unfortunately, the neurobiological underpinning of personality is extremely rudimentary (Grigsby & Stevens, 1999).

Gabriel (1990, 1993, 1995) has developed a model system of discriminative avoidance learning that may prove useful in providing for future integration of psychological and biological components involved in chronic pain. Gabriel distinguishes between an anterior and posterior thalamocingulate circuit. The anterior thalamocingulate circuit, centered on the medial dorsal thalamus and the anterior cingulate (area 24b), is specialized for the rapid and flexible acquisition of conditioned avoidance responses. In contrast, the posterior thalamocingulate circuit, centered

upon the posterior cingulate with afferents from anterior thalamic nuclei, is specialized for the maintenance and retention of responses involved in discriminative avoidance learning. These circuits are heavily interdependent and also dependent upon inputs from several other structures (e.g., the amygdala). This model appears of interest, especially given that psychological dimensions associated with the development or expression of chronic pain problems (e.g., active-passive, motor-sensory, independent-dependent) might be mapped onto this neurobiological substrate (Nicholson, in preparation). The structure of such neuro-psychobiological circuits underlying chronic pain may also be seen to be associated with aspects of social behavior and psychosocial development (i.e., maternal behavior, separation cry, etc.) (MacLean, 1986, 1993; Nelson & Panksepp, 1998). Increasing understanding of the neurobiology of attachment or other aspects of social or interpersonal behavior can be expected to facilitate understanding of chronic pain problems. It is suspected that development of animal models that explore the psychobiological substrate may be especially useful but this would require raising laboratory animals in an environment that would produce psychological vulnerability.

Whereas there has been much investigation about the possible role of psychological factors in the presentation of chronic pain patients, there is very poor understanding and even poorer empirical documentation of what this might entail. One approach to this issue is that the psychology of chronic pain is merely a matter of how people react to or cope with the ("real" or "physical") pain they have. This appears to represent what has been the dominant cognitive-behavioral perspective prevalent in at least North America. Many who adopt this approach seem to think that psychological distress, as can be measured by various brief questionnaires, is the only pertinent psychological phenomenon to be assessed. On the other hand, others consider that psychosocial factors may contribute to a vulnerability for development or expression of chronic pain problems, or that there is some "psychogenicity" in the expression of a pain problem. In such cases, a more detailed psychological analysis is usually considered. It is likely that these two approaches are often two sides of the same coin. For example, fear avoidance is a prominent problem with many chronic pain patients who may be unwilling to try to be doing things because of a fear of increasing pain, whereas other patients who do not have such fear may be coping better with pain and related problems or engaging in activity that may help to reduce the pain. Although this phenomenon is usually interpreted as a coping mechanism, it can also be considered a disposition or premorbid vulnerability.

It is likely that there are many shades of meaning to the term "psychogenic pain," some involving a weaker or stronger sense of how psychosocial factors may be causal. It is unknown to what extent any pain problem may be

primarily (or even exclusively) psychogenic or independent of peripheral factors. Even in such cases as sympathetic labor pain of a man whose wife is expecting, i.e., the *couvade syndrome* (Bardhan, 1965), there may be associated gastrointestinal effects related to the stress and anxiety of this event and focus upon gastrointestinal sensation that might generate some peripheral nociception. However, especially if such pain is severe, one might suspect there is central magnification of any actual peripheral nociception. Again, in general, it remains very poorly understood what pertinent psychosocial factors may be or what any specific mechanisms of effect might be. There is also very poor understanding of the interaction of psychosocial factors with the pain system or other systems (i.e., motor, immune, endocrine, autonomic, etc.). Nonetheless, there has recently been increasing attention devoted to these and related issues (Block, Kremer, & Fernandez, 1999; Gatchel & Turk, 1999; Grzesiak & Ciccone, 1994; Nicholson, 2000b).

It is suspected that there is typically an interaction of biomedical and psychological factors contributing to presentation in most cases of chronic pain. As is true for many traits, it might be expected that psychological predisposition or vulnerability is normally distributed with a minority (perhaps 5 to 15%) having marked disposition, another minority (again, perhaps 5 to 15%) being very resistant, and most of us somewhere in between. Thus, for someone with strong disposition, it may require little in the way of peripheral pathology/injury or peripherally generated nociception to activate central functional components associated with psychological factors. In others who are more resistant, it may require marked injury, perhaps under conditions of extreme stress, for a central sensitization effect associated with psychosocial factors or vulnerability to be activated. This is consistent with several recent vulnerability-diathesis-stress models of chronic pain (e.g., Dworkin & Banks, 1999). Returning to the example of fear avoidance, an individual with marked vulnerability may react with extreme fear avoidance to even little actual nociception (e.g., slight musculoskeletal strain). In an individual with little disposition, it may require much more severe injury and substantive nociception to limit activity due to fear avoidance. Notably, other vulnerability factors (e.g., genetic) should also be considered; but, again, an association or interaction with psychological factors can often be expected (e.g., gender, temperament, or other effects).

The nosological system of the American Psychiatric Association (APA, 1994) distinguishes between Pain Disorder Associated with Both Psychological Factors and a General Medical Condition versus Pain Disorder Associated with Psychological Factors. In the latter, psychological factors are considered to play the primary role in the onset, maintenance, severity, or exacerbation of chronic pain, whereas both psychological and medical factors are

considered to contribute to the former. Pain Disorder Associated with a General Medical Condition is not a psychiatric diagnosis and indicates that pain is associated with medical factors alone. Whereas these distinctions are certainly heuristic and useful, it should be noted that this nosological system provides no guidance about what psychosocial factors should be considered, how these might be measured, what any specific mechanisms of effect might be, or how any interaction effects between psychological and biomedical factors might operate. Again, just because psychological factors can be associated with onset, maintenance, exacerbation or severity of pain, and although they may well be primary, it does not mean that pain is not "real" or that there is not a neurobiological substrate to this disorder. This appears to be a common misconception (Teasell & Merskey, 1997). Again, psychological states or processes (e.g., pain, fear, responsibility, reading this sentence) are not merely figments of one's imagination but are presumed to have an underlying neurobiological substrate. "Functional" pain associated with psychological factors may be quite "real" and should perhaps be considered a variant of central pain as has previously been suggested (Nicholson, 2000b). Psychiatry has grappled with similar problems of what constitutes organic, functional, and psychologic factors in the understanding of several other disorders (e.g., depression) where both psychosocial and biomedical etiological factors can be identified and both biomedical or psychosocial treatments may be indicated.

There is a widespread misconception that what constitutes psychological factors contributing to presentation in chronic pain must be gross psychopathology, psychiatric disorder, or sexual/physical abuse during childhood. Whereas there is some evidence that physical or sexual abuse may play a part in the etiology of gastrointestinal or pelvic pain problems, there is otherwise little evidence that this is a relevant etiological factor in other pain problems (Drossman, 1994; Roy, 1998), although it may be that concurrent histories of abuse plus a significant nociceptive or neuropathic pain problem can result in increased affective distress and difficulty coping. There is mixed evidence on the causal relationship between pain and depression (more generally considered negative affect and certainly not always involving pre-morbid clinical depression), different studies suggesting that the relationship may be causal, reactive, or recursive (Robinson & Riley, 1999). On the other hand, it has also long been noted that many chronic pain patients appear to be model citizens and, although perhaps very active, with underlying dependence-independence conflicts or other identifiable characteristics, do not have gross pre-morbid psychosocial problems (Blumer & Heilbronn, 1982). In some cases, there are clear indications that psychological factors are playing a major role in presentation, such as when there is complete resolution of pain

problems on administration of a placebo, when there is marked exacerbation under stress or complete resolution in a calming environment, or when there is dramatic pain behavior when attention is focused on pain but no pain behavior when distracted. In many cases, however, the markers may be much more subtle.

Another important and often very contentious issue associated with many misconceptions about chronic pain is whether patients might be malingering (Martelli, Zasler, et al., 1999; Fishbain, et al., 1999a). Although it may be cruel to suggest that (actual) pain is just in the head, suggesting that it is not real or valid, it may be a greater insult to suggest that one's (actual) pain is the product of active dissimulation or malingering, that is, that the individual is just pretending to have pain (or greatly exaggerating pain) to obtain some financial or other benefits. On the other hand, some patients may be actively malingering and this can be very costly, diverting resources from those who need them. A recent review of the literature indicates that malingering might be present in from 1.25 to 10.4% of chronic pain patients but that estimates are not considered reliable and, furthermore, that there is no reliable method for detecting malingering with chronic pain patients (Fishbain, et al., 1999a).

Some signs, often considered to be "non-organic" (i.e., non-dermatomal sensory deficits) (Mailis et al., 2001; Waddell, McCulloch, Kummel, & Venner, 1980), have been found to be associated with actual abnormalities on functional neuroimaging, that is, lack of activation or deactivation of contralateral S1 cortex and other areas on stimulation of the affected side (Mailis et al., 2000). In addition, such nondermatomal somatosensory deficits are prevalent in several pain populations and are suspected as being associated with psychosocial factors (Fishbain, Goldberg, Rosomoff, & Rosomoff, 1991; Mailis & Nicholson, 1997).

Whereas symptom validity testing has been used in other domains (i.e., assessment of memory complaints) to quite unambiguously identify conscious dissimulation, e.g., when level of performance is statistically significantly below what would be expected on the basis of random responding, this is not possible with pain because there is no objective external criteria to evaluate the subjective report. Although there appears to be a trend to use certain cut-off scores on symptom validity testing with those pain patients who concurrently complain of cognitive problems, this is considered inappropriate because such techniques have not been normed on appropriate populations (i.e., chronic pain patients with affective or other problems). Furthermore, there is a large literature documenting cognitive deficits associated with either acute pain challenges in normal volunteers or chronic pain patients (Hart, Martelli, & Zasler, 2000; Martelli, Grayson, & Zasler, 1999; Nicholson, 2000c).

Although it may be very difficult to ascertain whether there is deliberate malingering, there is much stronger

evidence that compensation has the potential to influence presentation, for example, severity or duration of complaints (Cassidy et al., 2000; Loeser, Henderlite, & Conrad, 1995; Loeser & Sullivan, 1995; Nachemson, 1994; Rohling, Binder, & Langhinrichsen-Rohling, 1995). There is also consistent evidence that secondary gain, a concept that is crudely akin to social reinforcement of illness behavior, is an important factor affecting presentation (Fishbain, Rosomoff, Cutler, & Rosomoff, 1995). Again, it is often very difficult to disentangle the effect of these or other specific psychosocial factors from biomedical factors in individual cases.

Many myths and misconceptions about treatment for chronic pain also arise from our poor understanding of the phenomena or are specifically associated with a biased perspective of the mind-body problem. There appears to be a predominant misconception, on behalf of both patients and professionals, that medical science will solve the problem of pain and suffering. On the part of the patient, this may be associated with the idea that medical science is omniscient or omnipotent and can fix any and all problems. Some patients may relegate all responsibility for their pain problem(s) to their doctors. These or related attitudes can lead to persistent medical treatment seeking behavior. After repeated temporary successes (or failures) with numerous medical interventions, it might be questioned whether patients expect to be "cured" by medical science or if they really just want someone to take care of them, perhaps as their parents may once have done. Unfortunately, patients who present with much suffering and desire for treatment can usually find some physician who will provide treatment, whether or not there are good indications for any such intervention. Many interventions, especially surgical, can lead to very serious iatrogenic effects. Some patients may then end up with pain problems far worse than they previously had, wishing they had never had surgery. Iatrogenic effects are certainly not limited to medical interventions. Physiotherapy, chiropractic, or other physical therapies can also result in serious iatrogenic effects. Furthermore, psychological treatments, especially perhaps insofar as they enhance invalidism, can also greatly exacerbate problems. It should also be noted that many practitioners (whether they be plumbers, mechanics, physiotherapists, physicians, psychologists, lawyers, or others) will engage in their professional activity, applying the tools of their trade, often with little regard for the need or effectiveness of their interventions. It should not be forgotten that there are tremendous financial benefits on the part of practitioners. In this regard, it may be more pertinent to question the issue of compensation of practitioners than patients. It should also be recognized that there is a massive medical-industrial complex propagating biomedical research and treatments. Undue medicalization of a problem is not unique to the field of pain. For example, many people would rather have liposuction

for weight control than maintain a proper regimen of diet and exercise or accept a less than ideal body weight. It may be easier to administer Ritalin or other stimulants to school children rather than provide appropriate structure or stimulation for problems of activity level. Patients or their doctors may prefer to take a pill for problems of depression rather than pursue cognitive-behavioral change that may be more effective.

Although it might be demonstrated that there is an organic or biomedical substrate for a pain problem, this does not necessarily mean that there should be medical treatment. For example, there may be some mild degenerative spinal changes demonstrated on CT, or functional neuroimaging might demonstrate that there are some patterns of brain activation (or deactivation) associated with low-back pain. However, it might be that pain in this case is primarily associated with inactivity, poor posture, or poor back hygiene. The treatment of choice may be to have the person engage in an appropriate exercise regimen or other activity rather than perform back (or brain) surgery. In this context, it should be noted that a recent meta-analysis indicates that opioids provide some good effect with nociceptive pain, are less effective with neuropathic pain, and are not effective with idiopathic pain (Graven, de Vet, van Kleef, & Weber, 1999). Although no attempt will be made to review the literature, it is apparent that there are many myths or misconceptions regarding opioid treatment, ranging from the extremes of believing opioids should never be prescribed because this will lead to drug addiction, to the other extreme where no consideration is given for this possible problem and opioids are heavily prescribed whether or not there is beneficial effect.

As previously suggested, whereas it might be demonstrated that psychosocial factors are involved in the etiology, maintenance, exacerbation, or severity of pain problems, this does not mean that such pain is not "real." It also does not mean that such pain should be treated with psychological methods alone, nor that psychological interventions would necessarily be helpful at all. Indeed, pharmacological or other medical treatment might be the treatment of choice with these patients. It remains largely unknown to what extent any such pain problems would respond to psychosocial interventions or to what extent medical treatments may be required. In addition, whereas psychosocial factors may play an important or even primary role in the pain problem, with relatively little apparent peripheral pathology/nociception, it might be that alleviation of this minor peripheral component via medical intervention would be sufficient to completely resolve the problem.

Although psychosocial interventions have been shown to be effective for a wide variety of chronic pain problems, whether or not there is demonstrable peripheral pathology (Flor, Fydrich, & Turk, 1992; Holroyd & Lipchik, 1999; Morely, Eccleston, & Williams, 1999; Van Tulder, Koes, & Bouter, 1997), effect sizes are often limited and many

TABLE 38.1
Myths and Misconceptions about Chronic Pain

We know what pain is.
We know what the biological basis of pain is.
We know what the psychology of pain is.
Pain is either in the body or in the mind.
Pain has either sensory or affective and/or cognitive components.
Pain is psychogenic or pain is not psychogenic.
If there is no discernible organic basis, then pain must be “functional,” that is, “only psychological.”
Psychological means somehow not real or without any basis in physical reality.
If there is a psychological component, it is all in your head.
If there is a marked psychological component contributing to presentation, there is no organic substrate.
We have reliable tests that are specifically sensitive to “organic” vs. “non-organic” conditions, or we can accurately measure biomedical or psychological components contributing to presentation.
Patient self-report of pain severity or other problems is unbiased.
We know when a patient is malingering or to what extent compensation issues are affecting presentation.
Practitioners are not biased or are not influenced by compensation issues.
Medical science or biomedical treatments will solve all the problems of pain and suffering.
Psychological treatments are all that is necessary or you just need to be a better person or of better character.
If there is a major psychological component contributing to presentation, there should be psychological but not biomedical treatment.
Psychological presentation in chronic pain patients is either causal or reactive.
Psychological treatments are not helpful for real (organic) pain.
Because psychological factors may be associated with onset, maintenance, exacerbations, severity, etc., means that it is not a real.
Functional neuroimaging will allow us to unlock the secrets of pain and establish the organic vs. psychological basis of the pain.
Opioid use causes addiction or does not result in problems of addiction.
Pain does not cause cognitive problems.
All of a patient's problems are because of an accident/injury and pain.

patients do not find these helpful. It is largely unknown what specific techniques are effective for which patients. In addition, it has been suggested that any systematic treatment delivered with enthusiasm appears to be helpful, leading one to suspect placebo (or at least nonspecific) effects (Blanchard & Galovski, 1999). Indeed, placebo effects are common and prominent (Turner, Deyo, Loeser, Von Korff, & Fordyce, 1994). Again, the mechanism of effect is not well understood (Price & Fields, 1997), although it has been demonstrated that the placebo effect may be associated with a discrete neurobiological response (Benedetti, Arduino, & Aanzio, 1999; Harrington, 1997). There is clearly need for further study about how psychosocial or related interventions work.

Finally, there is a trend toward emphasizing the results of systematic reviews and meta-analyses (i.e., evidence-based medicine) to establish whether or not any specific treatment is effective. Although this is certainly laudatory and may help to weed out the “junk science” or inappropriate and possibly iatrogenic treatments, caution should be exercised about taking such reviews/analyses as “gospel.” These may not always be of adequate quality, thus raising concern about conclusions (Fishbain, Cutler, Rosomoff, & Rosomoff, 1999b). Furthermore, just because a set of studies does not provide evidence for something, it does not mean that this is necessarily so. For example, although one could line up the studies indicating that there is no peripheral biological basis for fibromyalgia, this does not mean that some such process will not be discovered. Following a strict set of guidelines based on the results of such reviews/analyses could prematurely limit the range of options for treatment or future research.

In conclusion, many myths or misconceptions about chronic pain exist today. These largely arise out of our poor understanding of the phenomena. This chapter focused on myths or misconceptions that are associated with the problem of mind–body dualism or the tendency to view pain problems from either an “organic” or a “psychological” perspective. In contrast, it has been repeatedly suggested that explication of the interface between these domains may be of critical importance in the understanding and treatment of chronic pain. Table 38.1 presents a summary of the primary myths and misconceptions that have been discussed in this chapter. It would be another misconception to think that many other myths and misconceptions do not exist.

REFERENCES

- Alexander, G.E., DeLong M.R., & Strick, P.L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience*, 9, 357.
- American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, D.C.: American Psychiatric Association.
- Bardhan, P.N. (1965). The couvade syndrome. *British Journal of Psychiatry*, 111, 908.
- Benedetti, F., Arduino, C., & Aanzio, M. (1999). Somatotopic activation of opioids systems by target-directed expectations of analgesia. *The Journal of Neuroscience*, 19, 3639–3648.
- Besson, J.M., Guilbaud, G., & Ollat, H. (1995). *Forebrain areas involved in pain processing*. Paris: John Libbey Eurotext.
- Birbaumer, N., Flor, H., Lutenberger, W., & Elbert, T. (1995). The corticalization of chronic pain. In B. Bromm & J.E. Desmedt (Eds.), *Pain and the brain: From nociception to cognition* (pp. 331–343). New York: Raven Press.

- Blanchard, E.B., & Galovski, T. (1999). Irritable bowel syndrome. In R.J. Gatchel & D.C. Turk (Eds.), *Psychosocial factors in pain* (pp. 270–283). New York: Guilford Press.
- Block, A.R., Kremer, E.F., & Fernandez, E. (Eds.). (1999). *Handbook of pain syndromes*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Blumer, D., & Heilbronn, M. (1982). Chronic pain as a variant of depressive disease: The pain-prone personality. *Journal of Nervous and Mental Disease*, 170, 381–486.
- Bromm, B., & Desmedt, J.E. (Eds.). (1995). *Pain and the brain: From nociception to cognition*. New York: Raven Press.
- Cabeza, R., & Nyberg, L. (1997). Imaging cognition: An empirical review of PET studies with normal subjects. *Journal of Cognitive Neuroscience*, 9, 1–26.
- Cassidy, J.D., Carroll, L.J., Cote, P., Lemstra, M., Berglund, A., & Nygren, A. (2000). Effect of eliminating compensation for pain and suffering on the outcome of insurance claims for whiplash injury. *New England Journal of Medicine*, 342, 1179–1186.
- Chapman, C.R. (1995). The affective dimension of pain: A model. In B. Bromm & J. E. Desmedt (Eds.), *Pain and the brain: From nociception to cognition* (pp. 283–301). New York: Raven Press.
- Chapman, C.R. (1996). Limbic processes and the affective dimension of pain. *Progress in brain research*, 10, 63–81.
- Chandler, E.H., & Dong, W.K. (1995). The role of the basal ganglia in nociception and pain. *Pain*, 60, 3–38.
- Coghill, R.C., Sanf, C.N., Maisog, J.M., & Ladoralo, M.J. (1999). Pain intensity processing within the human brain: A bilateral, distributed mechanism. *Journal of Neuropsychology*, 82, 1934–1943.
- Cohodarevic, T., Mailis, A., & Montanera, W. (2000). Syringomyelia: Pain, sensory abnormalities, and neuroimaging. *Journal of Pain*, 1, 54–66.
- Craig, A.D., Reiman, E.M., Evans, A., & Bushnell, M.C. (1996). Functional imaging of an illusion of pain. *Nature*, 384, 258–260.
- Davis, K.D., Taylor, S.J., Crawley, A.P., Wood, M.L., & Mikulis, D.J. (1997). Functional MRI of pain- and attention-related activations in the human cingulate cortex. *Journal of Neurophysiology*, 77, 3370–3380.
- Drevets, W.C., Burton, H., Videen, T.O., Snyder, A.Z., Simpson, J.R., & Raichle, M.E. (1995). Blood flow changes in human somatosensory cortex during anticipated stimulation. *Nature*, 373, 249–252.
- Drossman, D.A. (1994). Physical and sexual abuse and gastrointestinal illness: What is the link? *The American Journal of Medicine*, 97, 105–107.
- Dworkin, R.H. & Banks, S.M. (1999). A vulnerability-diathesis-stress model of chronic pain: Herpes zoster and the development of postherpetic neuralgia. In R.J. Gatchel & D.C. Turk (Eds.), *Psychosocial factors in pain* (p. 247–271). New York: Guilford Press.
- Fishbain, D.A., Cutler, R.B., Rosomoff, H.L., & Rosomoff, R.S. (1999a). *Chronic pain and disability exaggeration/malingering research and the application of sub-maximal effort research to this area: A review*. Poster presented at International Association of Pain 9th World Congress, Vienna, Austria.
- Fishbain, D.A., Cutler, R.B., Rosomoff, H.L., & Rosomoff, R.S. (1999b). What is the quality of the implemented meta-analytic procedures in chronic pain treatment meta-analyses? *The Clinical Journal of Pain*, 16, 73–85.
- Fishbain, D.A., Goldberg, M., Rosomoff, R.S., & Rosomoff, H.L. (1991). Chronic pain patients and the nonorganic physical sign of nondermatomal sensory abnormalities (NDSA). *Psychosomatics*, 32, 294–303.
- Fishbain, D.A., Rosomoff, H.L., Cutler, R.B., & Rosomoff, R.S. (1995). Secondary gain concept: A review of the scientific evidence. *The Clinical Journal of Pain*, 11, 6–21.
- Flor, H., Braun, C., Elbert, T., & Birbaumer, N. (1997). Extensive reorganization of primary somatosensory cortex in chronic back pain patients. *Neuroscience Letters*, 224, 5–8.
- Flor, H., Elbert, T., Wienbruch, C., Pantev, C., Knecht, S., Birbaumer, N., Larbig, W., & Taub, E. (1995). Phantom limb pain as a perceptual correlate of cortical reorganization. *Nature*, 357, 482–484.
- Flor, H., Fydrich, T., & Turk, D.C. (1992). Efficacy of multidisciplinary pain treatment centers: A meta-analytic review. *Pain*, 49, 221–230.
- Gabriel, M. (1990). Functions of anterior and posterior cingulate cortex during avoidance learning in rabbits. *Progress in Brain Research*, 85, 467–482.
- Gabriel, M. (1993). Discriminative avoidance learning: A model system. In B.A. Vogt & M. Gabriel (Eds.), *Neurobiology of cingulate cortex and limbic thalamus: A comprehensive handbook* (pp. 478–523). Boston: Birkhauser.
- Gabriel, M. (1995). The role of pain in cingulate cortical and limbic thalamic mediation of avoidance learning. In J.M. Besson, G. Guilbaud, & H. Ollat (Eds.), *Forebrain areas involved in pain processing* (pp. 197–211). Paris: John Libbey Eurotext.
- Gatchel, R.J., & Turk, D.C. (Eds.). (1999). *Psychosocial Factors in Pain*. New York: Guilford Press.
- Graven, S., de Vet, H., van Kleef, M., & Weber, W. (1999). *Opioids in chronic non-malignant pain: A meta-analysis of the literature*. Poster presented at International Association of Pain 9th World Congress, Vienna, Austria.
- Grigsby, J., & Stevens, D. (1999). *Neurodynamics of personality*. New York: Guilford Press.
- Grzesiak, R.C., & Ciccone, D.S. (Eds.). (1994). *Psychological vulnerability to chronic pain*. New York: Springer-Verlag.
- Harrington, A. (Ed.). (1997). *The placebo effect: An interdisciplinary exploration*. Cambridge, MA: Harvard University Press.
- Hart, R.P., Martelli, M.F., & Zasler, N.D. (2000). Chronic pain and neuropsychological functioning. *Neuropsychology Review*, 10(3), 131–149.
- Holroyd, K.A., & Lipchik, G.L. (1999). Psychological management of recurrent headache disorders: Progress and prospects. In R.J. Gatchel & D.C. Turk (Eds.), *Psychosocial factors in pain* (pp. 193–212). New York: Guilford Press.
- Hsieh, J.C., Stahle-Backdahl, M., Hagermark, O., Stone-Elander, S., Rosenquist, G., & Ingvar, M. (1995). Traumatic nociceptive pain activates the hypothalamus and the periaqueductal gray: A positron emission tomography study. *Pain*, 64, 303–314.

- Lenz, F.A., Graceley, R.H., Zirh, A.T., Romanowski, A.J., & Dougherty, P.M. (1997). The sensory-limbic model of pain memory. *Pain Forum*, 6, 22-31.
- Linton, S.J. (1999). Prevention with special reference to chronic musculoskeletal disorders. In R. J. Gatchel & D.C. Turk (Eds.), *Psychosocial factors in pain* (pp. 374-389). New York: Guilford Press.
- Loeser, J.D., Henderlite, S.E., & Conrad, D.A. (1995). Incentive effect of worker's compensation benefits: A literature synthesis. *Medical Care Research and Review*, 52, 34-59.
- Loeser, J.D., & Sullivan, M. (1995). Disability in the chronic low back pain patient. *Pain Forum*, 4, 114-121.
- MacLean, P.D. (1986). Culminating developments in the evolution of the limbic system: The thalamocingulate division. In B.K. Doane & K.E. Livingston (Eds.), *The limbic system: Functional organization and clinical disorders* (pp. 1-26). New York: Raven Press.
- MacLean, P.D. (1993). Perspectives on cingulate cortex in the limbic system. In B.A. Vogt & M. Gabriel (Eds.), *Neurobiology of cingulate cortex and limbic thalamus: A comprehensive handbook* (pp. 1-15). Boston: Birkhauser.
- Mailis, A., Amani, N., Umana, M., Basur, R., & Roe, S. (1997). Effect of sodium amytal on cutaneous sensory abnormalities, spontaneous pain and algometric pain pressure thresholds in neuropathic pain patients: A placebo-controlled study II. *Pain*, 70, 69-81.
- Mailis, A., Downar, J., Kwan, C., Nicholson, K., Mikulis, D., & Davis, K.D. (2000). FMRI in explainable widespread somatosensory deficits (WSDs) in patients with chronic pain. Nineteenth Annual APS meeting, Atlanta, CA, *American Pain Society Abstract*, 760, 158.
- Mailis, A., & Nicholson, K. (1997). Effect of normal saline controlled intravenous administration of sodium amytal in patients with pain and unexplained widespread non-anatomical sensory deficits: A preliminary report. *American Pain Society Abstract*, 708, 138.
- Mailis, A., Papagapiou, M., Umana, M., Cohodarevic, T., Nowak, J., & Nicholson, K. (2001). Unexplainable widespread somatosensory deficits in patients with chronic nonmalignant pain in the context of litigation/compensation: A role for involvement of central factors. *Journal of Rheumatology*, 28, 1385-1393.
- Martelli, M.F., Grayson, R., & Zasler, N.D. (1999). Post traumatic headache: Psychological and neuropsychological issues in assessment and treatment. *Journal of Head Trauma Rehabilitation*, 14(1), 49-69.
- Martelli, M.F., Zasler, N.D., & MacMillan, P. (1998). Mediating the relationship between injury, impairment and disability: A vulnerability, stress & coping model of adaptation following brain injury. *NeuroRehabilitation: An Interdisciplinary Journal*, 11(1), 51.
- Martelli, M.F., Zasler, N.D., Mancini, A.M., & MacMillan, P. J. (1999). Psychological assessment and applications in impairment and disability evaluations. In R.V. May & M.F. Martelli (Eds.), *Guide to functional capacity evaluation with impairment rating applications*. Richmond, VA: NADEP Publications.
- Melzack, R. (1999). From the gate to the neuromatrix. *Pain Supplement*, 6, S121-S126.
- Melzack, R., & Casey, K.L. (1966). Sensory, motivational and central control determinants of pain: A new conceptual model. In D. Kenshalo (Ed.), *The skin senses* (pp. 423-443). Springfield, IL: Charles C Thomas.
- Melzack, R., & Wall, P.D. (1965). Pain mechanisms: A new theory. *Science*, 150, 971-979.
- Merskey, H., & Bogduk, N. (Eds.). (1994). *Classification of chronic pain* (2nd ed.). Seattle: IASP Press.
- Morely, S., Eccleston, C., & Williams, A. (1999). Systematic review and meta-analysis of randomized controlled trials of cognitive behaviour therapy and behaviour therapy for chronic pain in adults, excluding headache. *Pain*, 80, 1-13.
- Nachemson, A. (1994). Chronic pain—The end of the welfare state? *Quality of Life Research*, 3, S11-S17.
- Nelson, E.E., & Panksepp, J. (1998). Brain substrates of infant-mother attachment: Contributions of opioids, oxytocin and norepinephrine. *Neuroscience and Biobehavioral Reviews*, 22, 437-452.
- Nicholson, K. (2000a). An overview of pain problems associated with lesions, disorder or dysfunction of the central nervous system. *NeuroRehabilitation*, 14, 3-13.
- Nicholson, K. (2000b). At the crossroads: Pain in the 21st century. *NeuroRehabilitation*, 14, 57-67.
- Nicholson, K. (2000c). Pain, cognition and traumatic brain injury. *NeuroRehabilitation*, 14, 95-103.
- Nicholson, K. (in prep.) Psychogenic pain: Review of the construct, a novel taxonomy and neuropsychobiological model.
- Ploghaus, A., Tracey, I., Gati, J.S., Clare, S., Menon, R.S., Matthews, P.M., & Rawlins, J.N.P. (1999). Dissociating pain from its anticipation in the human brain. *Science*, 284, 1979-1981.
- Porro, C.A., Francescato, M.P., Cettolo, V., & Baraldi P. (1998). *Cortical activity during anticipation of a noxious stimulus: A fMRI study* [Abstract]. 4th International Conference on Functional Mapping of the Human Brain, Montreal, Quebec.
- Price, D.L., & Fields, H.L. (1997). Where are the causes of placebo analgesia. *Pain Forum*, 6, 44-52.
- Robinson, M.E., & Riley, J.L. (1999). The role of emotion in pain. In R.J. Gatchel & D.C. Turk (Eds.), *Psychosocial factors in pain* (pp. 74-88). New York: Guilford Press.
- Rohling, M.L., Binder, L.M., & Langhinrichsen-Rohling, J. (1995). Money matters: A meta-analytic review of the association between financial compensation and the experience and treatment of chronic pain. *Health Psychology*, 14, 537-547.
- Roy, R. (1998). *Childhood abuse and chronic pain: A curious relationship?* Toronto: University of Toronto Press.
- St-Cyr, J., Taylor, A., & Nicholson, K. (1995). Behavior and the basal ganglia. In W.J. Weinger & A.E. Lang (Eds.), *Behavioral neurology of the movement disorders, Advances in neurology* (Vol. 65, pp. 1-28). New York: Raven Press.
- Teasell, R.W., & Merskey, H. (1997). Chronic pain disability in the workplace. *Pain Research Management*, 2, 197-205.

- Treede, R., Kenshalo, D.R., Gracely, R.H., & Jones, A.K.P. (1999). The cortical representation of pain. *Pain*, 79, 105-111.
- Turner, J.A., Deyo, R.A., Loeser, J.D., Von Korff, M., & Fordyce, W.E. (1994). The importance of placebo effects in pain treatment and research. *Journal of the American Medical Association*, 271, 1609-1614.
- Van Tulder, M.W., Koes, B.W., & Bouter, L.M. (1997). Conservative treatment of acute and chronic nonspecific low back pain. *Spine*, 22, 2128-2156.
- Vogt, B.A., Sikes, R.W., & Vogt, L.J. (1993). Anterior cingulate cortex and the medial pain system. In B.A. Vogt & M. Gabriel (Eds.), *Neurobiology of cingulate cortex and limbic thalamus: A comprehensive handbook* (pp. 313-344). Boston: Birkhauser.
- Waddell, G., McCulloch, J.A., Kummel, E.G., & Venner, R.M. (1980). Non-organic physical signs in low back pain. *Spine*, 5, 117-125.
- Wall, P.D. (2000). Pain in context: The intellectual roots of pain research and therapy. In M. Devor, M.C. Rowbotham, & Z. Wiesenfeld-Hallin (Eds.), *Proceedings of the 9th World Congress on Pain: Progress in pain research and management* (pp. 19-34). Seattle: IASP Press. Inclusive pages??
- Wood, J.D., Alpers, D.H., & Andrews, P.L.R. (1999). Fundamentals of neurogastroenterology. *Gut*, 45(Suppl. 2), II6-II16.