Assessing the Veracity of Pain Complaints and Associated Disability


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 psychiatrists, 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 healthcare 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 cognophobia 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 extent systems that fund evaluation and treatment of injury and disability.
TABLE 63.1
Survey of Attitudes Regarding Worker’s Compensation (WC)

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

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 healthcare 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
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The 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 pathologies, 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 reports 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 examination 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 premorbid 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 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 Stener'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, Bowknot and Currier 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 nonorganicity, 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 imprecation. 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 belabor 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 imprecation can be evaluated, as can any impairment for that matter, with appropriately designed forced choice testing. Additionally, examiners should realize that alleged pain imprecation 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, Paoloa, & Gray, 1984; Waddell, 1999). These are listed below:

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**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 wheeled to motor testing or regional sensory loss in a stocking or dermatomal 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 (Hadjistaurooulos & 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, Wil-liams, Zaslav, 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 tests 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, Pd, Pp, 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., Monsana 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
### TABLE 63.3
Response Bias Detection Measures and Strategies

<table>
<thead>
<tr>
<th>Pain Assessment Measures with Built-In Response Bias Indicators</th>
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<tbody>
<tr>
<td><strong>Pain Assessment Battery (PAB), Research Edition</strong></td>
</tr>
<tr>
<td>Proposed clinical hypothesis procedure evaluating</td>
</tr>
<tr>
<td>Millon Behavioral Health Inventory (MBHI)</td>
</tr>
<tr>
<td>Hendler (i.e., Mensana Clinic) Back Pain Test</td>
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<tr>
<td></td>
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<tr>
<td>Hoover’s test</td>
</tr>
<tr>
<td>Asatia-abasia</td>
</tr>
<tr>
<td>Nonorganic sensory loss</td>
</tr>
<tr>
<td>Nonorganic upper extremity drift</td>
</tr>
<tr>
<td>Stenger’s Test</td>
</tr>
<tr>
<td>Gait discrepancies when observed vs. not observed</td>
</tr>
<tr>
<td>Gait discrepancies relative to direction of requested ambulation</td>
</tr>
<tr>
<td>Forearm pronation, hand clasping, and forearm supination test for digit/finger sensory loss</td>
</tr>
<tr>
<td>Pain vs. temperature discrepancies</td>
</tr>
<tr>
<td>Lack of atrophy in a chronically paretic/paralytic limb</td>
</tr>
<tr>
<td>Impairment diminishes under influence of sodium amytal, hypnosis or lack of observation</td>
</tr>
<tr>
<td>Incongruence between neuroanatomical imaging and neurologic examination</td>
</tr>
<tr>
<td>Arm drop test</td>
</tr>
<tr>
<td>Presence of ipsilateral findings when implied neuroanatomy would dictate contralateral findings</td>
</tr>
<tr>
<td>Tell me “when I’m not touching” responses</td>
</tr>
<tr>
<td>Lack of shoe wear in presence of gait disturbance</td>
</tr>
<tr>
<td>Calluses on hands in “totally disabled” examinee</td>
</tr>
<tr>
<td>Assistive device “wear-and-tear” signs</td>
</tr>
<tr>
<td>Mankopf’s maneuver</td>
</tr>
<tr>
<td>Lack of atrophy in a limb that is claimed to be significantly impaired</td>
</tr>
<tr>
<td><strong>Medical Indicators</strong></td>
</tr>
<tr>
<td>Test for malingered lower extremity weakness associated with normal crossed extensor response</td>
</tr>
<tr>
<td>“Drunken type” gait with near-falls but no actual falls to ground</td>
</tr>
<tr>
<td>Patchy sensory loss, midline sensory loss, large scotoma in visual field, tunnel vision</td>
</tr>
<tr>
<td>Long tract involvement results in pronator type drift; proximal shoulder girdle weakness and malingering typically present with downward drift while in supination</td>
</tr>
<tr>
<td>Test for malingered hearing loss during audiologic evaluation</td>
</tr>
<tr>
<td>If organic, should be consistent regardless of whether observed or not</td>
</tr>
<tr>
<td>Gait for a patient with hemiparesis should present similarly in all directions; malingerers do not as a rule practice a feigned gait in all directions</td>
</tr>
<tr>
<td>Malingered finger sensory loss is difficult to maintain in this perceptually confusing, intertwined hand/finger position</td>
</tr>
<tr>
<td>Because both sensory modalities run in the spinotubular tract, they should be found to be commensurately impaired contralateral to the side of the CNS lesion</td>
</tr>
<tr>
<td>Lack of atrophy in a paralyzed/paralytic limb suggests the limb is being used or is getting regular electrical stimulation to maintain mass</td>
</tr>
<tr>
<td>All these observations are most consistent with nonorganic presentations including consideration of malingering or conversion disorder</td>
</tr>
<tr>
<td>Lack of any static imaging findings on brain CT or MRI in the presence of a dense motor or sensory deficit suggests nonorganicity</td>
</tr>
<tr>
<td>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</td>
</tr>
<tr>
<td>An examinee claiming severe right-brain damage who claims right-eye blindness and right-sided weakness and sensory loss</td>
</tr>
<tr>
<td>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.” 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)</td>
</tr>
<tr>
<td>An examinee who is unable to work should not present with signs of ongoing evidence of physical labor</td>
</tr>
<tr>
<td>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</td>
</tr>
<tr>
<td>Increase in heart rate commensurate with nociceptive stimulation during exam (some controversy exists on whether this always occurs)</td>
</tr>
<tr>
<td>If side-to-side measurements and/or inspection do not bear out atrophy consider other causes aside from one being claimed</td>
</tr>
</tbody>
</table>

*continued*
### TABLE 63.3 (CONTINUED)
Response Bias Detection Measures and Strategies

<table>
<thead>
<tr>
<th>Measure</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sudden motor give-away or ratchiness on manual strength testing</td>
<td>Considered to normally be a sign of incomplete effort or symptom exaggeration</td>
</tr>
<tr>
<td>Weakness on manual muscle testing without commensurate asymmetry of DTRs or muscle bulk</td>
<td>Suggests simulated muscle weakness if longstanding</td>
</tr>
<tr>
<td>Toe test for simulated low back pain</td>
<td>Flexion of hip and knee with movement only of toes should not produce an increase in low back pain</td>
</tr>
<tr>
<td>Magnuson's test</td>
<td>Have examinee point to area several times over period of examination; inconsistencies suggest increased potential for nonorganicity</td>
</tr>
<tr>
<td>Delayed response sign</td>
<td>Pain reaction temporally delayed relative to application of perceived nociceptive stimulus</td>
</tr>
<tr>
<td>Wrist drop test</td>
<td>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</td>
</tr>
<tr>
<td>Object drop test</td>
<td>Examinee claims inability to bend down yet does so to pick up a light object &quot;inadvertently&quot; dropped by examiner</td>
</tr>
<tr>
<td>Hip adductor test</td>
<td>Test for claimed paralysis of lower extremity, similar to Hoover's test yet looks for crossed adductor response</td>
</tr>
<tr>
<td>Disparity between tested range of motion (ROM) and observed range of motion of any joint</td>
<td>When ROM under testing is significantly disparate (e.g., less) from observed, spontaneous ROM, suspect functional contributors</td>
</tr>
<tr>
<td>Straight leg raise (SLR) disparities dependent on examinee positioning</td>
<td>Differences in SLR between sitting, standing, and/or bending may suggest a functional overlay to low back complaints</td>
</tr>
<tr>
<td>Grip strength testing via dynamometer</td>
<td>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</td>
</tr>
<tr>
<td>Sensory “flip” test</td>
<td>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</td>
</tr>
<tr>
<td>Pinch test for low back pain</td>
<td>Pinching the lumbar fat pad should not reproduce pain due to axial structure involvement; if test is positive, suspect a functional overlay</td>
</tr>
</tbody>
</table>

### 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
  - Validity indices (L, F, Fp, Fp, Da, K, VRIN, TRIN), F-K (Gough, 1954)

- **Minnesota Multiphasic Personality Inventory (MMPI-2)**
  - The Fake Bad Scale (Lees-Haley, 1991)
  - Compare subtle to obvious items
  - Rogers et al. (1994) – cutoff scores:
    - *Liberal:*
      1. F-scale raw score > 23
      2. F-scale T-score > 81
      3. F-K index > 10
      4. Obvious – subtle score > 83
    - *Conservative:*
      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
- Performance on easy tasks presented as hard
- Remote memory report

- Inconsistencies across tasks
- Low scores or unusual errors
- 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

<table>
<thead>
<tr>
<th>Personal information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comparison between test performance and behavioral observations</td>
</tr>
<tr>
<td>Inconsistencies in history and/or complaints, performance</td>
</tr>
<tr>
<td>Comparisons for inconsistencies within testing session (qualitative and qualitative)</td>
</tr>
<tr>
<td>Comparisons across testing sessions (qualitative, quantitative)</td>
</tr>
<tr>
<td>Symptom self-report: complaints</td>
</tr>
<tr>
<td>Main &amp; Spanwick, 1995</td>
</tr>
</tbody>
</table>

| Very poor personal information in absence of gross amnesia |
| Discrepancies |
| Inconsistencies across time, setting, interviewer, etc. |
| 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 |
| Poorer/inconsistent performance on re-testing |
| High frequency, severity of complaints and higher frequency, severity vs. significant other report or other collaborative report |
| 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 |
| Early symptoms reported late or acute symptoms reported as chronic |
| 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 |
| Arithmetic and orientation scale performance |
| WMS-R Malingering Index: Attention/Concentration Index vs. Memory Index |
| Grip strength |
| Recognition memory (California Verbal Learning Test (CVLT)) |
| Rey Complex Figure and Recognition Trail |
| Word Stem Priming Task Performance |
| Specific Cognitive Effort/Response Bias Measures |
| Word Memory Test (WMT) |
| Test of Memory Malingering (TOMM) |
| Dot Counting Test (DCT) |
| Computer Assessment of Response Bias (CARB) |
| Rey Memory for 15 Items Test (MFIT) |
| Symptom validity testing (SVT) |

| Low (vs. expected, estimated, etc.) |
| “Near-miss” (Gaser errors) |
| Attention–concentration index score < general memory index (AC-GMI) |
| Unusually low w/o gross motor deficit |
| < 13 |
| Atypical recognition errors (> = 2); recognition failure errors |
| Poor or unusual performance |
| < 50% chance responding |
| < 50% chance level responding |
| Correct/incorrect responses |
| < 89% raises suspicion |
| Lezak (1983), < 3 complete sets, < 9 items |
| < 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 malingers vs. externally or criterion-validated malingers, 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 symptoms/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 a 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 sequela 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, Papagapous, Umanna, Cohodasovic, 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

<table>
<thead>
<tr>
<th>Real Physical Pathology</th>
<th>Residual Functional Impairments</th>
<th>Residual Impairments On Exam, Testing</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Yes</td>
<td>1. Yes, and exaggerated</td>
<td>1. Yes, and not exaggerated</td>
</tr>
<tr>
<td>2. Mixed</td>
<td>2. Yes, and not exaggerated</td>
<td>2. Yes, and exaggerated</td>
</tr>
<tr>
<td>3. Indeterminate</td>
<td>3. No, and exaggerated</td>
<td>3. No, and not exaggerated</td>
</tr>
<tr>
<td>4. No</td>
<td>4. No, and not exaggerated</td>
<td>4. No, not exaggerated</td>
</tr>
</tbody>
</table>

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 of profiling or a profiling approach to response bias detection strategy used to 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 malingering. 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, Zaslser, & 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 quantitative 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.

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Assessing the Veracity of Pain Complaints and Associated Disability


Nicholson, K., Psychogenic pain: Review of the construct, a novel taxonomy and neuropsychobiological model (in review).


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


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 commonly 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 idopathic 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; Chudler & 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, Umama, 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 (Cohodaravic, 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, & Lamarol, 1999; Hsieh et al., 1995; Treede, Krensha, 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, Reiman, 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, Cetolo, & 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 uniformity 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; S-Cyr, Taylor, & Nicholoson, 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 of 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 neuropsychological 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-
physiological 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 &
Punkepp, 1998). Increasing understanding of the neuro-
biology of attachment or other aspects of social or inter-
personal behavior can be expected to facilitate understand-
ing of chronic pain problems. It is suspected that
development of animal models that explore the psychobi-
ological 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 psy-
chology 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 domi-
nant 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 psycho-
logical 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 dis-
position 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 indepen-
dent of peripheral factors. Even in such cases as sympa-
thetic 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 sen-
sation that might generate some peripheral nociception.
However, especially if such pain is severe, one might
suspect there is central magnification of any actual periph-
eral 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 a very poor understanding of the interaction of psy-
chosocial factors with the pain system or other systems
(i.e., motor, immune, endocrine, autonomic, etc.). None-
theless, there has recently been increasing attention
devoted to these and related issues (Block, Kremer, &
Fernandez, 1999; Gatchel & Turk, 1999; Grzesiak & Cio-

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 pre-
disposition 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 com-
ponents 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 avoid-
ance to even little actual nociception (e.g., slight muscu-
loskeletal strain). In an individual with little disposition,
it may require much more severe injury and substantive
nociception to limit activity due to fear avoidance. Nota-
ably, 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 Dis-
order Associated with Both Psychological Factors and a
General Medical Condition versus Pain Disorder Associ-
ated with Psychological Factors. In the latter, psychologi-
cal 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 psychological factors should be considered, how these might be measured, what 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 fragments 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 these 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; Loer, Henderlite, & Conrad, 1995; Loer & 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.

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 by 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 "psy-
chological" perspective. In contrast, it has been repeatedly
suggested that explication of the interface between these
domains may be of critical importance in the understand-
ing 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 mis-
conceptions do not exist.

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